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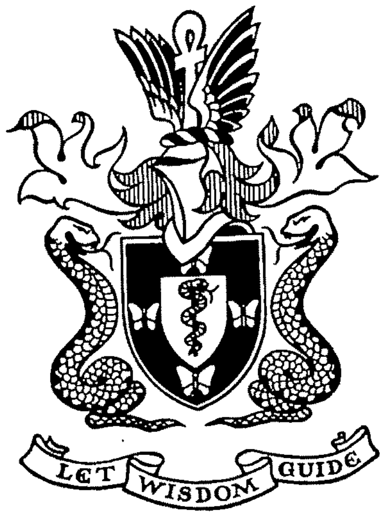

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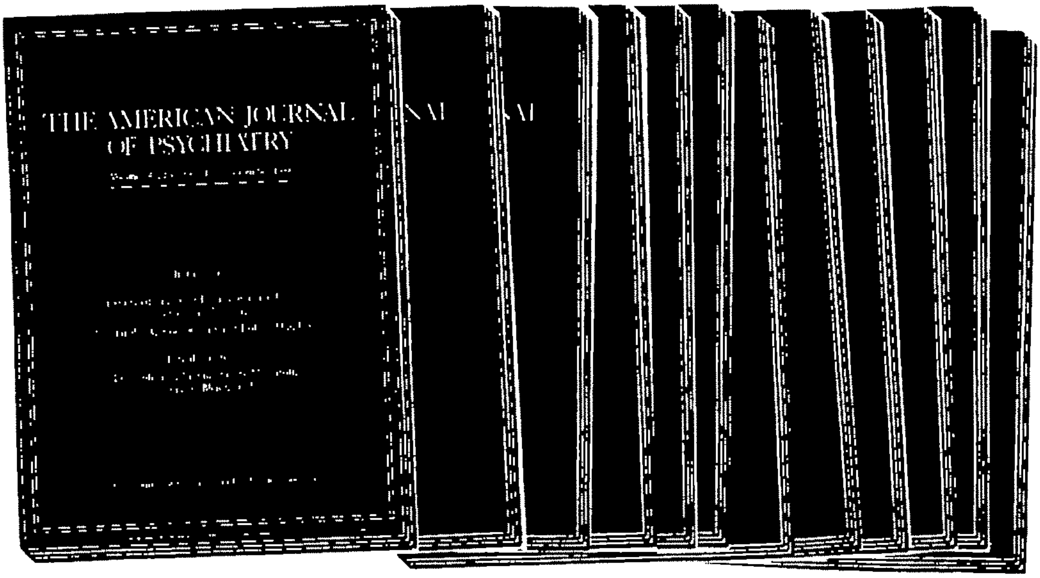
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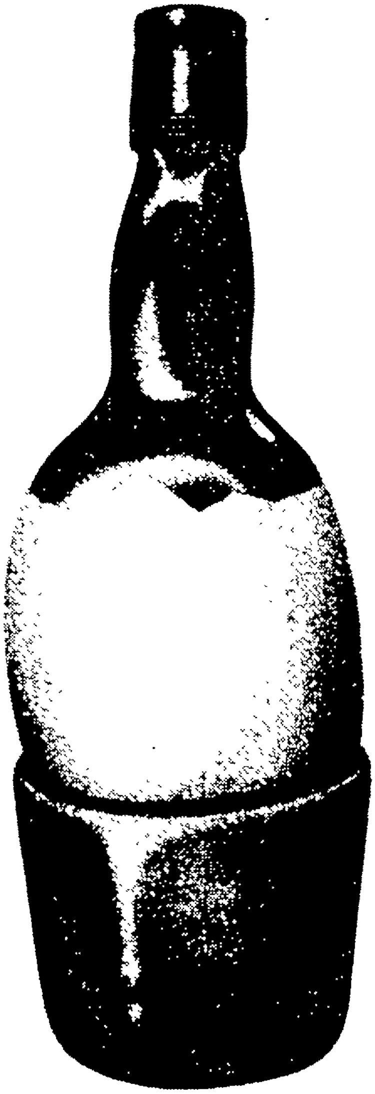

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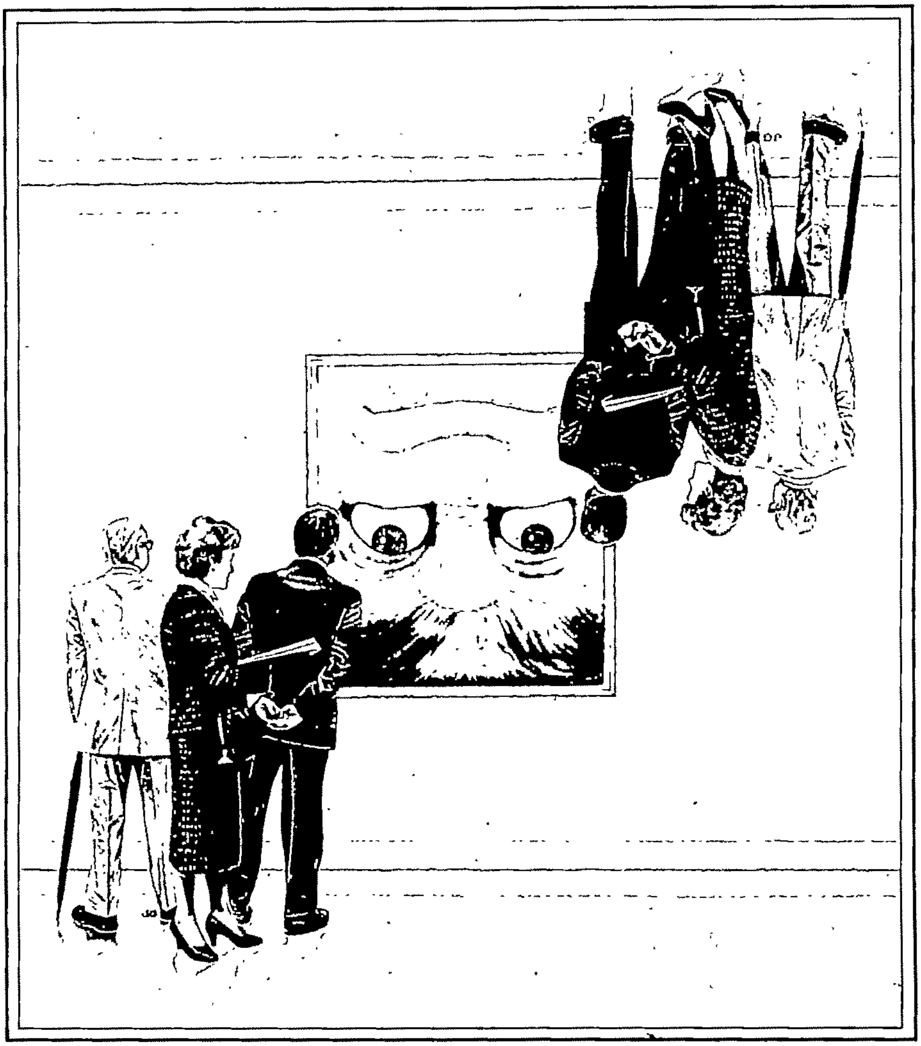

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Lecture

Changes in the Incidence of Schizophrenia?

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ERIK STRÖMGREN

In general, results of epidemiological studies on schizophrenia performed during the last century have the remarkable feature that no significant differences in expectancies and incidences have been found, neither between different populations nor over time. Recently, however, some studies have tended to show a noteworthy decrease in the incidence, as measured by statistics of first admissions. Possible sources of error are discussed, partly on the basis of recent Danish investigations. Although it is uncertain whether a true change in the incidence of schizophrenia has occurred, one feature is becoming evident: under modern treatment and care of-schizophrenics the natural history of the disease is becoming increasingly different between the two sexes. This fact, in combination with one of the hardest facts in schizophrenia research, namely the higher age of onset in females, may be useful starting points for hypotheses on aetiology, or at least necessary components of such hypotheses.

One of the most serious concerns of psychiatrists is undoubtedly our ignorance of the aetiology of schizophrenia. What we know with certainty is only that genetic factors play a role. The results of family studies, twin studies and studies on adoptees leave no doubt about that. On the other hand, twin studies have demonstrated that the genetic component is of limited significance. Monozygotic twin pairs with one schizophrenic proband show only about 40% concordance, thus leaving the responsibility for the greater part of the aetiology to environmental factors. Unfortunately, our ignorance about relevant environmental factors is nearly complete. Our knowledge that phenocopies of schizophrenia of organic origin do exist does not help us very much. We know that brain disease like Huntington's chorea, encephalitis and others can give rise to conditions which symptomatically cannot be distinguished from the other schizophrenias, and that similar conditions can be caused by drugs like amphetamine and related compounds. But such organically caused schizophrenic pictures account for less than 10% of all schizophrenic psychoses. The rest is silence.

On the other hand, the fact that schizophrenic states can be caused by well known organic factors may lead some to believe that all schizophrenias are the result of organic processes which, however, unfortunately cannot be revealed with current techniques. But that is in any case a matter of faith and should not induce us to close our eyes to the possibility of a predominantly psychological aetiology of some cases of schizophrenia.

The search for causative environmental factors is very intensive, both in the laboratories and using sociological techniques. For the latter, it would be of great importance if it could be demonstrated that the incidence of schizophrenia differs in different environments. In that case, research could be directed towards other, possibly relevant, differences between the various environments. Here, psychiatric epidemiology comes into the picture. Disappointingly, it must be said that the search for such variations of the incidence of schizophrenia in different populations, or in the same population at different points of time, has failed to give any remarkable results, except in the case of limited population groups living in isolates where arbitrary accumulations of schizophrenia, or absence of that disease, can of course easily occur. Thus, in an isolated North Swedish population, Böök (1953) found a disease expectancy of schizophrenia of close to 3%, in contrast to figures of between 0.7 and 1.0% normally found when larger populations are studied. Also, the incidence figures for schizophrenia found during a multinational WHO study (1986) in a number of countries in different parts of the world are strikingly similar.

The abundant literature concerning the frequency of schizophrenia may give a confusing impression. The widespread interest in epidemiological studies has induced many research workers, who are not sufficiently familiar with epidemiological techniques, to publish observations and conclusions which were naive and misleading, and which nevertheless occasionally had great public impact because they tallied with popular prejudice or ideologically based wishful thinking. In some cases it was obvious that even research workers, who in other contexts merited respect, were ignorant of the differences between basic epidemiological concepts like prevalence, incidence and morbid risk, carelessly comparing figures from incidence studies with figures from prevalence studies etc., in spite of the fact that these figures are of course incommensurable. It is also amazing that many comparative studies have been published in which factors like differences in age distribution and mortality have not been taken into consideration, likewise such important factors as internal migration within a population. It is comforting that many widespread ill-founded epidemiological conclusions have been corrected in recent years by competent epidemiologists, not least from Great Britain. The Maudsley Lecture by Edward Hare (1983) is a good example of this trend.

The use of the three concepts—prevalence, incidence and morbid risk, respectively—each has its indications and, under favourable research conditions, it is also possible to convert one to the other. Whereas prevalence figures are especially useful for determination of the needs for treatment and social assistance to sick people, incidence figures are those which are most readily applicable for aetiological studies. Their main weakness is that they are often derived from hospital statistics, which not only cover just a fraction of cases, but are also hampered by the fact that the extent of this fraction may differ enormously between different populations. Here nosocomial factors and threshold factors are of decisive importance, varying as they are with the social and economic conditions of the population, with the distance to the hospital, with the reputation of the hospital, with the demographic characteristics of the population, urban or rural, etc. It is therefore essential that studies of incidence and prevalence which are based on contacts with the health system, especially hospitals, are supplemented by field studies which determine true incidences and prevalences. But field studies require much manpower and time and can therefore be applied to only relatively small populations. The ideal is to combine studies based on contacts with the health system with field studies, the aim of the latter first of all being to serve as correctives to the former.

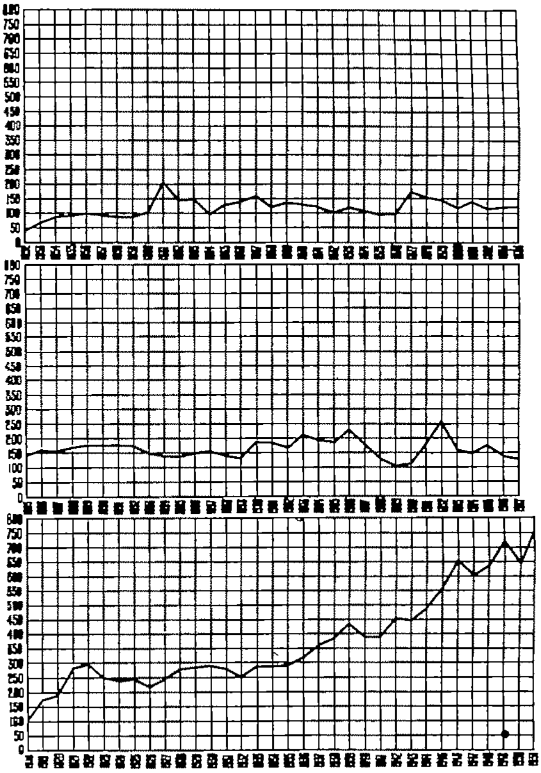

FIG 1. Annual admissions to the Aarhus Psychiatric Hospital 1852–1952.

Can we use hospital statistics at all for the determination of incidence of schizophrenia? In general, it can be said that admission statistics give only vague and unreliable estimates of the true incidence. Even in a relatively homogeneous, small population like the Danish, the probability of being admitted to a psychiatric ward varies markedly between different parts of the country. For instance, different investigations during the last decades have shown that this probability has been five to eight times greater in the city of Copenhagen than in rural areas, although the differences with regard to incidence of mental disorders could not be nearly as great. In addition, within the metropolitan area, the admission rate is much larger in the poorer parts of the city than in the more wealthy parts. However, closer analysis reveals that, with regard to schizophrenia and other severe psychoses, these differences are not nearly as large, the differences in admission rates thus being mainly due to short-lasting reactions, mostly of non-psychotic nature.

Where differences over time are concerned, it is striking that changes in admission rates are correlated primarily to availability of space in the psychiatric institutions. This is illustrated in Fig. 1, which shows admissions to the Psychiatric Hospital in Aarhus during the first 100 years of its existence, from 1852 to 1952. When the hospital was opened, it had only 130 beds. During the first years there were of course many admissions, but just until the hospital was full. The admission rate then decreased until 1861 when a large extension of the hospital took place. In 1864 there was again a short decrease; in that year Denmark was at war with Germany, and patients avoided admission to hospital, if ever possible. The stagnation during the 1870s was interrupted by the opening of a new hospital in the same area in 1877. The increase after the turn of the century was caused by transfer of chronic patients to two nursing homes. After 1910 there was also an increase caused by the introduction of supervised family care to which many of the chronic patients were transferred, and this permitted admission of more fresh cases. During World War I, there was the usual reluctance to be admitted to hospital. There was a special reason for the steep rise after the war: malaria therapy was introduced, and this very specialised treatment was concentrated in two Danish hospitals, one of them being the Aarhus hospital. This implied that patients with GPI were sent to Aarhus from a number of other hospitals and sent back again after treatment, thus accelerating the turnover in Aarhus. After the introduction of shock therapy in the 1930s, there was a new rise, with an interruption at the beginning of World War II. In conclusion, it can be said that there is no reason to suppose that any of the big changes in admission rates were caused by changes in the incidences of mental disorders; if anything, the changes rather reflected the natural history of hospital building and, more or less, the political history of Denmark.

With regard to the evaluation of admission statistics, it is generally agreed that *first admissions* are by far the most interesting. They must be related much more closely to the incidences than the total number of admissions could ever be. But closer scrutiny reveals that 'first admission' is a concept which is less useful than it might appear at first sight. The first condition is naturally that 'first admission' should mean 'first ever admission to a psychiatric institution'. In countries with national psychiatric registers, 'first admission' in this more narrow sense is a reasonably meaningful concept. But only with a number of reservations.

Serious difficulties arise, for example, when mental disorders are classified according to systems, in which the *disease course* plays a role for definition of classes. If there is a class for recurrent depressions, no first admission is possible, because a patient must have been admitted previously to fall into this class. Current classifications of bipolar illness present similar difficulties and, all in all, it cannot be recommended that definitions of classes include criteria dealing with disease course.

Schizophrenia presents special difficulties. Firstly, there is usually an interval between the onset of the disorder and the first admission, often of several years' duration. Some schizophrenics are never admitted, in some cases because they die before admission would have taken place. Secondly, many schizophrenics are not given the diagnosis of schizophrenia during their first hospital admission. At the Institute of Psychiatric Demography in Aarhus, which takes care of the Danish National Psychiatric Register, Munk-Jørgensen (1985) has recently studied a cohort of all Danish patients first admitted during 1972 who had received the diagnosis of schizophrenia either during that year or some time during the following 10 years. The study showed that only 50% of the male and 40% of the female patients were diagnosed as suffering from schizophrenia during the first admission; the remaining patients were given the diagnosis during later admissions. On the average, 1.7 years for men and 2.2 years for women elapsed between the patient's first contact with an inpatient institution and the diagnosing of schizophrenia. In retrospect, most of the patients later diagnosed as schizophrenic suffered from the same disease during their first admission and could as well have received the diagnosis at that time; reluctance to use a prognostically serious label presumably caused a delay in many cases. Therefore, although these patients were in reality schizophrenics on first being admitted, they would not be recognised as such by official statistics, and they would never appear as 'first admissions for schizophrenia'.

The ideal will be to compensate for these errors by performing field studies with the aim of finding patients who were never admitted, and to determine the age of onset for these patients as well as for those who have been admitted. But such studies are only possible under special, very fortunate circumstances. It is therefore important that Ødegaard (1952) in Norway has devised methods which make it possible to calculate, on the basis of hospital statistics and case histories, the number of schizophrenics not yet admitted under the assumption—which seemed to be valid in Norway—that sooner or later all schizophrenics who survive will be admitted. Compensation should then, if possible, also be made for the deficit caused by the fact that some schizophrenics may die at a stage when they are psychotic, but without definite schizophrenic symptoms.

In spite of all these sources of error and the uncertainty of the results of the admission statistics, it is of interest to study recent results from centres in which admission statistics can be assumed to be relatively complete and reliable. A recent report by Eagles & Whalley (1985) is highly relevant in this respect. They studied first admissions to Scottish psychiatric hospitals during the period 1969–1978. Age-standardised rates for schizophrenia fell from 19.6 per 100 000 population to 11.8, a drop of 40%. The rates for affective psychoses also decreased, but only by 28%. Other changes were not significant. These results are important because they come from an area in which registration seems to have been excellent for a long time.

In Denmark, nationwide psychiatric registration was started in 1938 and became relatively complete in 1952. Since 1970 the data have been computerised (Dupont, 1983). Thus, although some of the technical conditions are adequate, the interpretation of results to be mentioned should be made with some reserve. The presentation has a mainly methodological scope, even though some of the results may be interesting enough to stimulate further and more penetrating research.

First, mention will be made of results from some systematic census studies which have been performed since 1942, with 5-year intervals (Weeke *et al*, 1986). All patients resident in psychiatric institutions in Denmark were registered on particular census days in September in each of the census years. The data were arranged according to age, sex, institutions and, since 1957, diagnosis. As expected, there were significant changes during all quinquennia. It was interesting that, with regard to age and sex distribution, linear extrapolations from 1942 to 1957 predicted with great accuracy the status in 1970; however, after this year the figures developed in a way which had not been predicted. Most notably, predicted increases of the oldest age groups did not occur. With regard to diagnostic distribution, the most remarkable feature was the decrease of resident schizophrenics since 1953. It had been anticipated that there would be a considerable decrease, but it was a surprise to see that this decrease was much more pronounced in the females, than in the males, and particularly among middle-aged schizophrenic females. Modern combined drug and social therapy seems to work much better in females than in males. The decrease in the prevalence of resident schizophrenics which was so spectacular until 1970 has continued since then, but at a more moderate rate.

But what about the admission rates? During the last 15 years, there have been about 40 000 admissions annually to Danish psychiatric institutions. However, the percentage of first admissions has decreased slowly, from 36 to 26%. An increasing proportion of the admissions concerns day-patients.

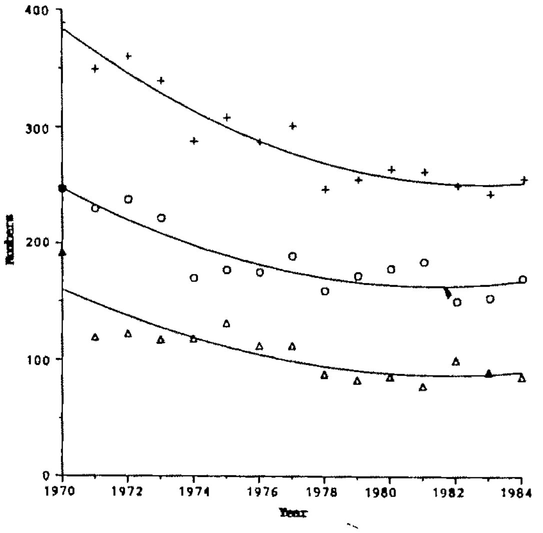

Fig. 2. First admission of schizophrenics 1970–1984 (0 male, Δ female, + male and female).

A special study has been performed concerning first admissions for schizophrenia during the period 1970–1984. Figure 2 shows the absolute numbers of first admissions for both sexes combined. The decrease is remarkable. However, it seems that the decrease is slowing down during the last few years. It might be expected that the decrease would be expressed more for females, but that is not the case. The cause for the previously mentioned difference between the sexes with respect to prevalence of resident schizophrenics is obviously *not* that schizophrenia is becoming less frequent in females than in males, but rather that female schizophrenics stay for a shorter time in hospital and have better chances to stay out of hospital after discharge.

How can we interpret the decrease of first admissions of schizophrenics? In view of the serious sources of errors, all interpretations should be made with the greatest caution. The simplest explanation for the decrease might be that psychiatrists are becoming increasingly reluctant to make the diagnosis of schizophrenia unless the evidence practically forces them to do so; an increasing number of schizophrenics would then never appear as first admissions. Others may, during the first undiagnosed admissions, be treated so successfully that they can be cared for on an outpatient basis in the future. If eventually the diagnosis of schizophrenia is made, it will not appear in the admission statistics.

Some further circumstances will have to be taken into consideration. During the observation period, have there been other notable changes in the admission patterns in general? Although the total number of admissions has not changed significantly, the rate of first admissions, as mentioned, has decreased moderately, from 36 to 26%. In contrast, for schizophrenia the relation between first and later admissions has changed drastically, from 11% in 1970 to 4% in 1984, the change being caused both by a decrease in first admissions as well as by an increase in all admissions, the latter being much more marked than the very moderate increase in all admissions for all diagnoses. These facts indicate that during this period the trend has been to substitute a number of long-lasting admissions by a greater number of short admissions; if the first admissions also tend to be shorter, the likelihood of reaching a diagnosis of schizophrenia at this point of time will decrease.

An attempt to control for all these sources of error would be very laborious. It is therefore tempting to try to attack the problem from a different angle. In this connection, it seems appropriate to mention a study which is currently being performed on the Danish island of Bornholm. I shall first present a short orientation concerning some psychiatric epidemiological investigations in this population.

During the 1930s, I was engaged in epidemiological studies on Bornholm. One of my aims was to try to ascertain all cases of psychoses that had occurred on the island since 1892, and, in addition, I made a special census of psychoses on a particular date in 1935. After finishing the study, I published a monograph (Strömgren, 1938) and concluded the book with a paragraph emphasising the importance of performing repeated surveys. I stressed how useful it would have been if somebody had carried out a similar census 50 years earlier. Then 2 years ago I was approached by four young psychiatrists who worked in the psychiatric department of the general hospital on Bornholm. They had noted the concluding paragraph in my book and pointed out that, now 50 years had nearly elapsed, it was time to repeat the survey. I agreed wholeheartedly, and during these 2 years I have been in cooperation with the Bornholm psychiatrists, first of all with Dr Søren Bøjholm.

One of the first aims has been to perform a census of schizophrenia for a particular date in 1983 and to compare this with the census from 1935. One advantage would be that the principal investigator was the same, and that the diagnostic evaluations may therefore be reasonably comparable. Another advantage is that conditions on Bornholm make epidemiological studies relatively easy. The population has not changed very much over the 50 years. All the time the population has been about 50 000, but of course the age distribution has changed. The main occupations are still fishing and farming. Industry has increased but does not dominate.

The technique for information collection differed between the two census studies. In the 1930s, I worked at the local general hospital and examined all the case notes since 1892, and in addition I used all the case notes concerning people from Bornholm who had been admitted to psychiatric hospitals in Denmark since 1859. All general practitioners on the island were visited, and in each parish there were key people who knew everybody in that parish. From these sources, information was collected about all people who were regarded as being mentally deviant. Most of them I saw personally. With regard to schizophrenia, I think I have reason to believe that very few cases may have escaped me, if any.

Since the 1930s, many things of relevance have happened, first of all a psychiatric department was established in 1961 in the general hospital in the city of Rønne. The files of this department were therefore the starting point for the new census. In addition, the general practitioners were questioned and the National Psychiatric Register was consulted.

During the recent census there were some problems which did not exist in the 1930s. First of all, complications arose when schizophrenia-like psychoses co-existed with drug abuse. In these cases, it could be very difficult to ascertain which came first, the psychosis or the abuse. Next there were some of these very patients who lived on Bornholm, but could not be regarded as belonging to the population there. They came from other parts of the country and had been drug abusers for some time when they decided, occasionally in groups, to go to Bornholm to stay in the nice countryside there, maybe in a deserted farm house. In such cases, as in other cases in which mental abnormality is the real cause for transfer to Bornholm, the person should not be regarded as belonging to the Bornholmian population. Thus, if a young schizophrenic living in Copenhagen feels persecuted and believes he will be safer in his parents' summer cottage in Bornholm and therefore moves over and stays, he does not belong to the population there. On the other hand, if people who are Bornholmians move to other parts of the country, as a consequence of their schizophrenia, they should still be regarded as belonging to the Bornholmian population. Such demographic criteria are occasionally as difficult to ascertain as the diagnosis of schizophrenia.

Difficulties were also presented by a few people who for several years have been living in complete isolation and refuse contact. It will nevertheless usually be possible to reach a probable diagnosis, and

TABLE I
Prevalence of schizophrenia on Bornholm in 1935 and in 1983

	Numbers					
	Males		*Females*		*M+F*	
Age	*1935*	*1983*	*1935*	*1983*	*1935*	*1983*
15–24	4	3	1	1	5	4
25–34	12	21	9	2	21	23
35–44	16	6	16	8	32	14
45–54	14	14	13	6	27	20
55–64	5	4	11	13	16	17
65–74	4	7	13	9	17	16
75–84	0	5	5	5	5	10
85+	0	1	0	5	0	6
Total	55	61	68	49	123	110

in any case there are so few of these cases that they cannot affect the statistics seriously.

The result of the two censuses can be seen in Table I. It is remarkable that, while the prevalence of schizophrenia in males has not changed significantly, there is a decrease with regard to female schizophrenics. The figures should be taken with considerable reserve. The study has not been completed, there are some loose ends, but it seems unlikely that the final result will deviate very much from the one seen in the table. Table II shows rates.

Here again, we seem to be confronted with a notable difference between the sexes with regard to schizophrenia. These differences might possibly provide a clue to factors of importance for the development of schizophrenia. Why is it that while maturation in general occurs earlier in women than in men, onset of schizophrenia occurs definitely later in women? A number of investigations on large representative samples of schizophrenics show this difference. Figure 3 shows the distribution of age at onset of schizophrenia in males and females based on a large amount of material from Munich to which I had access 50 years ago (Strömgren, 1935). Later studies have given curves of quite similar shape. This difference in the age of onset is much more difficult to explain than the fact that schizophrenic women are becoming much less frequent in hospital treatment than are schizophrenic males. But even the sex difference found in the census investigation in Bornholm can have a rather trivial explanation. Schizophrenia in females, arising later than in males, will relatively often lead to a mainly paranoid state with relatively good preservation of the personality which at that stage is already mature and fully developed. It may then be difficult to make the diagnosis of schizophrenia at once; but in any case these patients are treated, quite often with good results, bringing them into a state which never becomes definitely schizophrenic. If this is the explanation, it is gratifying for the patients but a disappointment for the scientist, because this explanation will not give any information concerning the aetiology of schizophrenia.

TABLE II
Prevalence of schizophrenia on Bornholm in 1935 and in 1983

	Rates					
	Males		*Females*		*M+F*	
Age	*1935*	*1983*	*1935*	*1983*	*1935*	*1983*
15–24	0.14	0.08	0.03	0.03	0.10	0.06
25–34	0.36	0.64	0.27	0.07	0.33	0.35
35–44	0.59	0.18	0.56	0.25	0.58	0.22
45–54	0.59	0.58	0.54	0.24	0.56	0.41
55–64	0.28	0.15	0.58	0.48	0.44	0.32
65–74	0.32	0.31	0.88	0.35	0.62	0.33
75–84	0.00	0.45	0.77	0.34	0.43	0.58
85+	0.00	0.36	0.00	1.23	0.00	0.88
Total	0.37	0.32	0.41	0.25	0.39	0.29

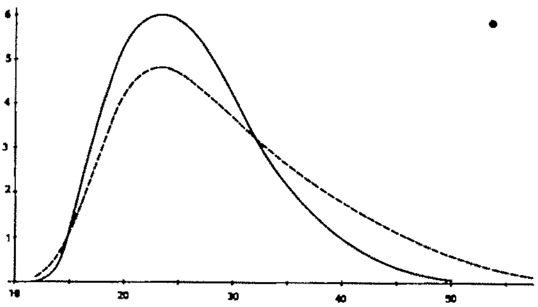

FIG. 3. Age of onset in schizophrenia (——males, - - -females).

A further step in the investigation could then be to try to identify patients who could fit this pattern, i.e. patients who have come to treatment in a stage which *may* be an early stage of schizophrenia, who have received adequate antipsychotic treatment with considerable effect and are now in a stage which cannot be diagnosed as schizophrenia. If the number of such cases should correspond to the number of schizophrenic females which seem to be 'missing' in the census, this might be taken as tentative support for the explanation given for the unexpected decrease in the number of female schizophrenics. It may seem to be quite utopian to find all such cases. In most populations, it would no doubt be unrealistic, but conditions for investigations of this kind are fortunate in the Bornholm population which is remarkably well served in a psychiatric respect, and in which identification of ill people is feasible to a degree that is unusual for a population of that size.

At this point, it may be relevant to mention another study which was carried out on Bornholm in the 1930s (Strömgren, 1937). At that time, I found the study to be of little interest, but it does touch on problems which in recent years have come into focus in schizophrenia research. At that time the Bureau of Hygiene of the League of Nations was interested in possible relationships between infectious diseases and mental disorders. Being supposed to have information on most cases of mental disorder that had occurred on Bornholm during the recent decades, I was asked to investigate whether any correlations between incidences of infectious diseases and mental disorders respectively could be ascertained. I therefore arranged the cases of major mental disorders known to me according to year of onset and compared this distribution with data collected by the medical officers of health who had continuously received information from the general practitioners about infectious diseases of public interest. No correlations whatever could be found between occurrence of epidemics of such diseases and onset of mental disorders. Many possibilities for different lengths of periods of latency between infections and occurrence of the different mental disorders were examined, but the results were entirely negative. Needless to say, these findings cannot in any way disprove the possibility that there may be correlations between particular, perhaps unreported, infectious diseases and occurrence of schizophrenia.

One of the aims of my lecture has been to illustrate ways in which large-scale statistics of a nationwide nature and more intensive studies of limited population groups may supplement each other, and also in which ways census studies and longitudinal studies can be brought into meaningful connection with each other. However, it must be admitted that even under favourable circumstances, the conclusions that can be drawn from such combined investigations are mostly uncertain and will have to be assessed with great caution. Nevertheless, it is my feeling that epidemiological studies may stand just as good a chance of providing clues to the aetiology of schizophrenia as do other scientific approaches being pursued at present. This is my excuse for discussing, in considerable detail, mainly methodological problems in psychiatric epidemiology.

References

Böök, J. A. (1953) A genetic and neuropsychiatric investigation of a North-Swedish population, with special regard to schizophrenia and mental deficiency. *Acta Genetica et Statistica Medica (Basel)* 4, 1–100, 345–414.

Dupont, A. (1983) A national psychiatric case register as a tool for mental health planning, research, and administration. The Danish model. In *Information Support to Mental Health Programs* (eds E. M. Laska, W. H. Gulbinat & D. A. Regier). New York: Human Sciences Press.

Eagles, J. M. & Whalley, L. J. (1985) Decline in the diagnosis of schizophrenia among first admissions to Scottish mental hospitals from 1969–78. *British Journal of Psychiatry*, **146**, 151–154.

Hare, E. (1983) Was insanity on the increase? *British Journal of Psychiatry*, **142**, 439–455.

Munk-Jørgensen, P. (1985) The schizophrenia diagnosis in Denmark. A register-based investigation. *Acta Psychiatrica Scandinavica*, **72**, 266–273.

Ødegaard, Ø. (1952) The incidence of mental diseases as measured by census investigations versus admission statistics. *Psychiatric Quarterly*, **26**, 212–218.

Strömgren, E. (1935) Zum Ersatz des Weinbergschen "abgekürzten Verfahrens". *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **153**, 784–797.

—— (1937) Est-il possible de démontrer une relation statistique entre la fréquence des maladies infectieuses et la fréquence des cas récents d'aliénation mentale? *Bulletin Mensuel de l'Office International d'Hygiène Publique*, **29**, 2538.

—— (1938) *Beiträge zur psychiatrischen Erblehre. Auf Grund von Untersuchungen an einer Inselbevölkerung*. Copenhagen: Munksgaard.

Weeke, A., Munk-Jørgensen, P., Strömgren, E. & Dupont, A. (1986) Changes in utilization of Danish psychiatric institutions I. An outline of the period 1957–1982. *Comprehensive Psychiatry*, **27**, 407–415.

World Health Organization (1986) Early manifestations and first-contact incidence of schizophrenia in different cultures. A preliminary report on the initial evaluation phase of the WHO collaborative study on Determinants of Outcome of Severe Mental Disorders. *Psychological Medicine*, (in press).

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Review Article

Biological and Neuropsychological Approaches to Childhood Autism

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There is growing conviction that childhood autism is a biologically based disorder. The evidence that has accrued in a variety of areas pertaining to biological abnormality in autism suggests that, with the possible exception of genetic factors, very few data are available that illuminate the autistic disorder specifically. Neurological models which might be useful in guiding further research are discussed and reasons for the slow progress in this important aspect of the study of autism are identified.

Early childhood autism, which traditionally has been considered to be the earliest form of childhood psychosis, has been the subject of a quantity of research greatly out of proportion to its incidence in the population. It is a severe disorder, beginning probably from birth, characterised by major deficits in cognitive and social functioning. In its purest form, autism occurs in less than two children in 10 000 (Lotter, 1966); in its more diluted form, i.e. when diagnostic criteria are less stringent, the incidence is still only about four in 10 000. Its interest to clinicians, researchers and lay people alike is perhaps due to the very mysteriousness of the condition, the fact that most of the children given this diagnosis tend to be attractive and appealing in appearance without outward physical signs of the very serious handicap it entails, and the fact that it is a disorder which is manifest from a very early age.

Early infantile autism was first delineated by Kanner in the early 1940s and is characterised by the following symptoms: extreme aloneness or self-isolation and withdrawal from social contact from very early in development, an obsessive need for sameness in the environment and in daily routine, a preoccupation with objects which are often used in peculiar non-functional ways, ritualistic and stereotyped behaviours such as rocking, spinning or manipulation of objects, extreme resistance to new learning, severe language disability which for more than half of cases means muteness and in most of those who do speak, bizarre non-functional language including echolalia, perseverative and stereotyped speech, idiosyncratic use of pronouns and sometimes metaphorical language.

The use of gestures to communicate, and symbolic play are notably absent in infancy (Ricks & Wing, 1976; Prior, 1979). Although Kanner claimed in his original formulations that these children had 'good cognitive potential', it has now been demonstrated repeatedly that most autistic children are moderately or severely retarded and that only a small minority (about one-fifth) show evidence of normal intellectual ability, albeit of a rather uneven and idiosyncratic kind. Autistic children with measured IQs below 40 or 50 have a very poor prognosis (DeMyer *et al*, 1973). Data on intellectual functioning levels together with results of many experimental studies suggest that the disorder is primarily cognitive rather than emotional and consequently the direction of basic research has been adapted to this conceptualisation of the disorder (Rutter, 1983).

In the most recent DSM-III classification of disorders of childhood and adolescence (American Psychiatric Association, 1980), infantile autism is now categorised as a 'pervasive developmental disorder'. This reclassification of autism involves a shift from a perception of the disorder as a psychosis to a perception of a disorder which involves failure to follow the normal developmental process in social, cognitive and psychological spheres.

Psychodynamic aetiology was stressed by Kanner and other early workers, although paradoxically at the same time it was asserted that the disorder was 'inborn' (Kanner, 1943, 1944). On the basis of his observations of the parents of autistic children, Kanner believed that they were 'refrigerator'-type parents—cold, intellectual, obsessive, highly educated people who had been unable to form the normal bonds of attachment with their child. Happily, little credence is given now to this proposition. A number of studies have supported the counter claim, that where it is found that a majority of parents of autistic children are intelligent and highly educated (and this has indeed been the case in a number of studies [e.g. Lotter, 1966; Prior *et al*, 1976]), this has most likely been the result of referral artefacts (Schopler *et al*, 1979; Wing, 1980).

The current consensus of opinion is very much towards a biological basis for childhood autism (Rutter, 1974; Schopler, 1983). However, very much more progress has been made in describing and measuring the behavioural deficits in the disorder than in solving the puzzle of its aetiology. In this paper, research aimed at revealing biologically based abnormalities in autism will be reviewed briefly as a preface to a discussion of current neuropsychological models which might be helpful in guiding future research of an aetiological type.

Biological factors

Because of the rarity of the syndrome, genetic studies are necessarily scant. However, there is accumulating evidence of some kind of genetic influence, albeit of a non-specific kind. The rate of autism and of milder cognitive and language disorders in siblings of autistic children is notably in excess of population-based rates (Lotter, 1966; Spence, 1976; Folstein & Rutter, 1977; August *et al*, 1981; Minton *et al*, 1982).

Ritvo and his colleagues (1982, 1985) have mounted a major study of genetic influences in autism and are investigating families with more than one autistic child in the USA, Canada and France. Although they report that their data are consistent with autosomal recessive inheritance of the disorder, it may not be possible to generalise the results from these very unusual families. In fact, the nature of inheritance is unknown at this stage and only a general influence can be identified. The raised risk of perinatal stress, especially in twin studies, dilutes the force of the genetic hypothesis (Folstein & Rutter, 1977). Furthermore, the well documented excess of males with the disorder has not been explained by any of the genetic theories.

A number of studies have reported significantly more perinatal complications in children who developed autism and childhood psychosis (Kolvin *et al*, 1971; Harper & Williams, 1975; Prior *et al*, 1976; Finegan & Quarrington, 1979; Links *et al*, 1980; Tsai & Stewart, 1983). However, without detailed and accurate records of pregnancy and birth histories, we are hampered by reliance on retrospective reporting by parents which may be particularly suspect when reasons for the severe abnormalities of autism are being sought. Gillberg & Gillberg (1983) have circumvented this problem in their report of a Swedish population study based on extensive and systematic official birth history data. They found high scores for 'reduced optimality' (i.e. pre-, post- and neonatal factors which are related to risk) in their sample of autistic children, thus supporting some influence of birth risk factors in at least a proportion of cases.

Major areas of research effort in the search for physiological abnormalities in autism have been neurobiological investigations and EEG, sleep, and evoked potential studies. These areas are replete with methodological difficulties. Despite this the importance of neurobiological factors in the aetiology of autism is argued strongly. Although earlier it was held that autistic children were free from overt signs of brain damage (indeed, in the past this has been one of the exclusionary criteria for the diagnosis of autism), more recent research concerned with neurobiological factors has supported biologically based aetiology. Kolvin *et al* (1971) and Lobascher *et al* (1970) concluded that at least half of their cases showed organic signs, whilst DeMyer *et al* (1972) found that only 14% of their autistic sample were within normal limits on brain dysfunction indices.

Motor milestones are often reported as slow (Kolvin *et al*, 1971; Ornitz *et al*, 1978; DeMyer *et al*, 1981), and DeMyer (1976) has claimed that gross motor abilities involving the lower limbs are at a retarded level although less handicapped than fine motor and ball-handling skill. She sees autistic children as a neurologically disabled group and has described them as 'dyspraxic' (see also Jones & Prior, 1985).

Delayed development of hand preference in autistic children is of a similar order to that reported for retarded children (Colby & Parkinson, 1977) and this has been interpreted as suggestive of neurobiological dysfunction or delay (but see McCann, 1981). Lower levels of functioning have been found in autistic children with mixed handedness compared with those with established dominance (Tsai, 1983; Fein *et al*, 1984*b*). A review of 13 studies of handedness by Fein *et al* (1984*a*) showed only two with a real increase in left-handedness, although overall they concluded that left-handedness was about twice as common as in a normal population and almost exactly the same as in retarded and epileptic populations (Satz, 1973). A proportion of neurodevelopmental findings are undoubtedly the result of inclusion of children with a variety of biologically based disorders in which autism is part of the symptoms; nevertheless there does seem good evidence to support the significance of abnormalities in this area. Damasio & Maurer (1978) have emphasised the motor deficits of autistic children and suggested that, in particular, they may implicate basal ganglia structures in the aetiology.

The incidence of reported EEG abnormalities in autistic children ranges from 10 to 83% with an average of 52% (DeMyer *et al*, 1981). However, there are no EEG abnormalities which are unique to the disorder and there is minimal evidence concerning localisation of any EEG abnormalities; most commonly they are distributed diffusely (Waldo *et al*, 1978). Tsai *et al* (1981) have reported a significantly greater incidence of epilepsy and abnormal EEGs in autistic girls as compared with autistic boys, attesting to the greater severity of the disorder in females. Unsurprisingly, cases with normal EEG are likely to have a better long-term outcome (Tsai *et al*, 1985). However, on the basis of a thorough review of psychophysiology in 'early onset psychosis', James & Barry (1980) could only conclude that "characteristic spontaneous EEG irregularities, indicating some CNS disability *may* exist". The question of interpretation of abnormalities as indicating under- or over-arousal remained unresolved according to their review, although maturational factors were seen as influential. Evidence related to cardiovascular, respiratory and electrodermal activity was similarly inconsistent and inconclusive. Sleep studies (e.g. Ornitz *et al*, 1969; Tanguay, 1977) suggest immaturity in REM sleep but no unique abnormality.

Brainstem dysfunction in autistic children has been explored using brainstem auditory evoked potentials (BAEPs) (Skoff *et al*, 1980; Fein *et al*, 1981; Gillberg *et al*, 1983a). Longer brainstem transmission time and a suggestion of greater abnormalities on the left side of the brain are reported. However, a significant proportion of cases show no abnormalities at all, and some studies do not find prolonged transmission times (Courchesne *et al*, 1984).

A number of researchers have postulated specific brainstem abnormalities as basic to autism. These include Rimland (1964), who suggested the reticular formation, Ornitz (1978), who suggested the vestibular system, MacCulloch & Williams (1971), a dorsal brainstem lesion around the nucleus of the tractus solitarius, Simon (1975), a specific lesion in the inferior colliculi, and Rosenblum *et al* (1980), "neurological dysfunction in the auditory system at the brainstem level". Empirical data to support these hypotheses have not been forthcoming. Tanguay *et al* (1982) have proposed a relationship between abnormal auditory evoked responses, implicating auditory processing deficits in some children, and the maldevelopment of neural substrates at critical early periods. This suggestion is supported by the conclusions of Niwa *et al* (1983) who investigated the P300 component of evoked response potentials in a pitch-discrimination task in autistic subjects. However, the influence of attention deficits and/or auditory processing problems on findings in this area has been investigated insufficiently to allow anything but speculation at this stage.

Biochemical research was reviewed by Piggott in 1979 and found to offer equivocal and inconsistent findings. Variation in biochemical research techniques, lack of normative and developmental data (Ritvo, 1979) and the uncertain influence of mental retardation on reported data contribute to this unsatisfactory state of affairs. There has been much focus on serotonin function areas in the central nervous system because of the theory that abnormalities of serotonin metabolism are related to psychotic disorders (e.g. Ritvo *et al*, 1970; Piggott *et al*, 1975; Yuwiler *et al*, 1975). Hyperserotoninaemia has been reported in approximately one-third of autistic children but is similarly found in many medical and neuropsychiatric disorders and in mental retardation. Its relationship to central nervous system functioning, or to any symptom or behaviour is uncertain (Kuperman *et al*, 1985). DeMyer *et al* (1981) have concluded that 'blood serotonin levels are more strongly related to intellectual status than to psychiatric diagnosis'. Neuroendoncrinological studies (Yamazaki *et al*, 1975), immunological studies (Stubbs, 1976; Stubbs *et al*, 1977) and enzyme studies (e.g. Jackson & Garrod, 1978) have also produced some evidence for non-specific abnormalities but no coherent pattern related to a diagnosis of autism has emerged. Gillberg's group reported inferential evidence for the importance of abnormalities of monoamine metabolism in at least some autistic children (Gillberg *et al*, 1983*b*).

Wing's claim (1981) that autism is not a single disorder but occurs in combination with many other developmental and organic disorders is consistent with the evidence noted above, i.e. many physiological and biochemical disturbances seen in some autistic children are also seen in children with a wide range of disturbed functioning.

Autism Association with other Disorders

It appears that one of the possible causes of autism may be a viral infection either early in life, or, as an effect later in development of a 'slow virus'. The incidence of autism in rubella children is much in excess of that in the normal population (412 in 10 000 *vs* 2–4 cases per 10 000) (Chess, 1971, 1977) suggesting a specific link between the rubella virus and the development of autism. Chess (1971) has suggested that autism may be one consequence of the invasion of the central nervous system by the rubella virus in the same way as autistic behavioural disturbance can be produced by viral encephalitis. However, any form of brain damage such as that produced by the rubella virus, may increase the risk of the development of behavioural and cognitive handicaps such as those found in autism.

Autism or autistic behaviour is also associated with some other organically based disorders involving retardation, most strongly with tuberous sclerosis, phenylketonuria and encephalitis. It has also been associated with coeliac disease, congenital syphilis, histidinaemia, toxoplasmosis, purine disorder, infantile spasms, Cornelia de Lange syndrome, congenital cytomegalovirus (Stubbs *et al*, 1984), neurofibromatosis (Gillberg & Forsell, 1984) and the fragile X syndrome (Meryash *et al*, 1982; August & Lockhart, 1984). However, the association may be between retardation and autism rather than any relationship with specific syndromes. Study of the symptoms associated with these disorders serves to illustrate the fact that autistic behaviour can be a consequence of various kinds of insults to the nervous system as well as developing without apparent organic origin.

Consideration of these kinds of data suggests a somewhat perturbing trend in biological research in autism. In the eagerness to discover biological markers, a wide variety of cases whose *primary diagnosis* is not autism but who have autistic behaviour is being investigated. Whilst this shows clearly that autistic symptoms may be correlates of numerous biologically based disorders, it does not contribute to our understanding of the basis for autism in the original Kannerian sense of the syndrome. It is important to focus research efforts on the children with no other obvious disorders if we are to contribute to knowledge of aetiological significance in autism. Thus, out of a multitude of studies, little has emerged to illuminate the puzzle of the aetiology of autism. Of the areas considered, genetics seems to offer the most promise for productive further exploration. However, the development of some theoretical underpinning to guide further research may be essential if wasteful 'fishing expeditions' are to be avoided. One approach to such an effort is to take a neuropsychological view of the handicaps of autism and to consider whether this has any advantages in guiding further research.

Neuropsychological approaches

There have been some attempts in recent speculations concerning the causes of autism to formulate some neurological models which can subsume findings in the area so far. For example, an early study by Hauser *et al* (1975) using pneumoencephalography showed enlargement in the ventricular system in a proportion of a sample of autistic children. They suggested a deficiency of substance in the left hemisphere of the brain. Unfortunately their group was a heterogeneous one precluding conclusions specific to autism.

DeLong (1978) concedes that autism probably has multiple aetiologies but suggests that at least for a subgroup a neuropsychological interpretation is relevant. He draws attention to the similarities between symptoms in autism and in the Kluver-Bucy syndrome. This disorder occurs in man as a consequence of bilateral surgical ablations of medial temporal lobes or following herpes simplex encephalitis. "Deficits in the Kluver-Bucy syndrome have been described as an incapacity for adaptive social behaviour, and a loss of recognition of the significance of persons and events. Such patients show an empty blandness, an absence of emotion or concern for family or other persons, and pursue no sustained purpose in activity" (DeLong, 1978). This description does suggest a strong parallel with the most severe and more retarded cases of autism but is less generally apt for the higher functioning group. In addition, autistic children do sometimes show extreme concern for particular (idiosyncratic) events or activities and, at least in middle childhood, for particular people, i.e. empty blandness does not characterise their behaviour in situations important to them, and this may be observed even in the most severe cases.

DeLong also comments on some similarities between autistic behaviour and some features of Korsakoff's psychosis; however, the work of Boucher & Warrington (1976) which was concerned with this issue did not provide convincing evidence for relating the amnesic syndrome to autism. Drawing on the neurological data relating to Korsakoff's psychosis, DeLong (1978) suggested that a left unilateral medial temporal lesion, which impairs verbal learning, and an intact right hemisphere might produce the pattern (which is seen in autism) of adequate learning of places, faces and nonsense patterns together with impaired social interaction and language. The problem of lack of transfer of damaged left hemisphere function to the intact right hemisphere (which would normally be expected in lesions sustained early in life) is accounted for by suggesting that such transfer only occurs when the lesion involves specific left hemisphere cortical areas. DeLong has suggested, as have Prior and her colleagues (Prior, 1979; Hoffman & Prior, 1982), that for the majority of severely retarded cases, where intellectual and social functioning is extremely impaired, damage would have to be bilateral with no right hemisphere compensation or normal development. The idea of unilateral and bilateral damage in accounting for the considerable differences between high and low functioning cases is attractive; however, the clinical picture is not sufficiently clear cut, nor are the neuropsychological data yet compelling enough to consider this idea more than an hypothesis worthy of further investigation. Furthermore, comparisons between ablated monkeys, Kluver-Bucy syndrome adults, alcohol-induced brain damage and a developmental disorder like autism must be considered as tenuous to say the least.

These hypotheses have been evaluated further by Hetzler & Griffin (1981) who have suggested bilateral neuropathology of the temporal lobes of the brain as a significant aetiological factor in autism. Their model is based not only on the Kluver–Bucy amnesic syndrome data but also incorporates evidence relating to developmental dysphasia and Geschwind's (1965) 'disconnection syndrome', as well as a variety of reported organic factors. However, these authors concede the difficulties involved in developing this proposition. Such a model would not account satisfactorily for the high functioning autistic sub-group.

Neurological models

A speculative neurological model for autism was elucidated by Damasio & Maurer in 1978. Beginning with the observation that the abnormal behaviour in autism is comparable to that seen in brain damaged adults, especially those with frontal lobe, basal ganglia or limbic system damage, they propose that "autism is consequent to dysfunction in a complex of bilateral CNS structures that include mesial frontal lobes, mesial temporal lobes, basal ganglia . . . and thalami . . ." (1978). As evidence for this theory they cite the motility disturbances, abnormalities of muscular tone, posture, gait and akinesia as signs of dysfunction of the basal ganglia, especially the neostriatum and of closely related structures of the medial aspects of the frontal lobes (see also Tsai *et al*, 1981). This also fits with the disturbances of communication, attention and perception which can occur as a result of mesial frontal lobe lesions and basal ganglia abnormalities and which are characteristic of autism. The ritualistic and compulsive behaviours commonly observed in autism are also seen in patients with damaged frontal lobes, especially those with early sustained lesions. The inability to learn by experience, to adapt to changing environments and to organise appropriate responses are common to both autistic and frontal lobe patients. Perseverance, lack of initiative, concreteness, shallow affect and lack of empathy similarly can be observed in individuals with autism and damage to the frontal lobe. Hoffmann & Prior (1986) have attempted to explore this hypothesis by administering tests purported to be sensitive to frontal lobe dysfunctioning. Autistic children did indeed show deficits in behaviour mediated by the frontal lobe, e.g. on the Milner Maze Test, where their performance was characterised by inability to form plans or strategies, considerable perseverance, inability to profit from feedback concerning errors and lack of affective response to either correct or incorrect choices. Their performance on the Wisconsin Card Sorting Test suggested impaired conceptualisation abilities, and again perseverance reponses were common.

Whilst these hypotheses deserve further investigation, the leap from behavioural observation to physiological abnormality is large and in view of the lack of evidence for lesions in autistic patients the proposed connection between frontal lobe damage and autistic behaviour remains tenuous. It would be encouraging if there were some evidence of structural abnormalities consistent with neuropsychological hypotheses. However, recent computerised axial tomography studies (Hier *et al*, 1979; Damasio *et al*, 1980; Campbell *et al*, 1982; Gillberg & Svendsen, 1983; Prior *et al*, 1984) have offered little support. Although abnormalities are sometimes reported in a proportion of samples, they seem to be characteristic of those children with severe retardation and other organic signs and thus say little specifically about autism. No abnormalities have been observed in high functioning cases (Prior *et al*, 1984). Of course the problem may be biochemical rather than structural, but again there is no evidence to support any hypothesised abnormality.

The speculative nature of these neurological models is well illustrated by the fact that, whilst using the same kind of behavioural evidence, Damasio & Maurer (1978) implicate older cortical structures, whilst Tanguay (1977) suggested the neocortex as the locus of abnormality. Tanguay's suggestion is that autism may be the result of dysfunction in the "frontolimbic system" and that autistic behaviour may be a later reaction to a major early insult. He emphasised the importance of timing in the effects of any neural insult—a theoretically critical issue which can only be investigated properly with comprehensive developmental research (Tanguay & Edwards, 1982). Geschwind & Galaburda (1985) have speculated recently on cerebral lateralisation, biological mechanisms and pathology of various kinds, including developmental disorders such as autism. Whilst their arguments are too complex to be summarised here, the nub of their thesis in relation to autism concerns links between anomalies of handedness, differential rates of development of the right and left cerebral hemispheres and aspects of the immune system. An interesting suggestion is that the worsening of conditions at or near puberty (as, for example, the increase in fits seen in autism) argues for a major role for the sex hormones. They also suggest that the higher incidence of developmental learning disorders (of which autism might be an extreme form) in males may be related to testosterone possibly interacting with genetic influences.

A limbic system model proposed by Lamondella in 1977 has received surprisingly little attention in the autism literature. Lamondella suggests that the limbic system may be the site of dysfunction in autism. This suggestion had in fact been made in 1960 by Schain & Yannet and a related hypothesis had been proposed again in modified form by Deslauriers & Carlson in 1969. Since the limbic system is responsible for arousal, social interaction, emotion, sensory thresholds, attention to novel stimuli, as well as basic communication functions, Lamondella (1977) suggests "intrinsically or extrinsically triggered limbic malfunctions as the cause of early infantile autism". Limbic epilepsy may also be proposed as the cause of the increase in fits in adolescent and young adult autistic cases. Derryberry & Rothbart (1983) have noted that the limbic system seems to be significant in controlling information processing within the cortex via the reticular system and that "direct cortical projections from the limbic systems may serve to alert central processing mechanisms to the presence of a motivationally-relevant stimulus, and to direct attention toward that stimulus".

Lamondella offers a model which divides the limbic system into subsystems that correspond to levels of forebrain organisation all of which are relevant to communicative functions. Level 1 involves primary sign behaviour, Level 2 visceral sign complexes, Level 3 somatic sign schemata and somatovisceral sign learning, Level 4 signal communication, and Level 5 propositional communication. It is hypothesised that in autism there is a breakdown around Level 4 (thalamic level) or in some cases even lower in the system. The thalamus is said to be influential in neocortical speech systems and thus this is relevant to the fact that a universal symptom in autism is language abnormality (Prior *et al*, 1976). Such a model also fits well with Hermelin's (1982) conceptualisation of autism as a failure to develop adequate communication in either thought *or* feeling as a consequence of developmental deficits in genetically preprogrammed capacities for non-verbal as well as verbal communication. The interesting question of critical periods for the development of limbic functions is also pertinent since it brings in the notion of the ability to compensate for damaged limbic components. This factor is unknown in human subjects but data from monkeys suggest that compensation is only possible up to the age of 1 or 2 years. After that time limbic damage has permanent effects on behaviour. Given the belief that autism is supposed to have its onset before the age of 2 or 3 years, it is of interest to speculate on the relationship between age of onset and plasticity in the limbic system. It is claimed that other structures may take over limbic system functions in congenital damage cases.

Although Lamondella's model is difficult to test and lacks support from any extant physiological or neurological studies, it appears worthy of further exploration since it fits well with behavioural observations of autistic children, it incorporates the notions of critical periods and plasticity, it covers the most critical and basic aspects of autism, the social, motivational and communication deficits, and it is not weakened by the findings so far of no particular structural defects in the brains of autistic individuals. It also allows for some heterogeneity in symptoms and could conceivably be elaborated to include some explanation of the fact that a proportion of autistic children do show normal intelligence, can develop language and can make a reasonable adjustment in later life. However, imperfect understanding of the structures and the functions of the limbic system, its relationship with other brain functions and the connexion between structure and function in the individual almost certainly means that we may wait some time before being able to explore this hypothesis productively.

At a somewhat different level of brain organisation are the proposals of Prior and Hoffmann (Prior, 1979, 1984; Hoffmann & Prior, 1982). Drawing together behavioural, psychometric, neurophysiological and experimental evidence, they have suggested that it appears that much of the behaviour of higher functioning autistic children, at least, reflects mediation or control of function by the right hemisphere of the brain. Hoffmann & Prior (1982) have presented evidence which shows that higher functioning autistic children perform according to their chronological age level on tests purporting to assess right brain functions but are grossly handicapped, in fact perform more poorly than mental age-matched (and therefore younger) controls, on tests purporting to assess left brain functions. In addition, there is some evidence from dichotic listening studies (Prior & Bradshaw, 1975) and from evoked potential data suggesting abnormal lateralisation of function (Dawson *et al*, 1982, 1986). It is not yet clear whether this pattern of performance in autistic children reflects immaturity (with right hemisphere and less mature strategies being used in all cognitively demanding situations) or whether there is in fact selective damage to the left hemisphere in a subgroup of autistic children who may have been able to compensate for such damage by greater development of the right hemisphere permitting the attainment of quasi-normal intelligence except on tests of high language content.

There are a number of theoretical problems with this model, especially since, if autism has its onset early in life, there ought to be more effective compensation for lateralised damage in the infant brain. Bilateral damage would have to be proposed for the low functioning majority of autistic children who share many of the behavioural symptoms while lacking the intellectual and language attainments of the high functioning group. If it were considered that the two groups represent different disorders, of course this problem would be resolved in part at least. Reference to Kanner's early discussions of the disorder suggest that early cases of infantile autism did not show the degree of global retardation which characterises so many of the cases now called autistic. Despite these caveats, there is some further support for the proposals in a study of lower functioning autistic children using the Halstead–Reitan Neuropsychological Test Battery for Children (Dawson, 1983), where results essentially similar to those of Hoffmann & Prior (1982) were found with significantly greater left than right hemisphere dysfunction. However, Dawson was careful to note that only five autistic children showed left hemisphere impairment alone, while the remaining five showed involvement of both hemispheres. Failure to develop the usual symbolic functions of the left hemisphere, in particular language and gesture, is hardly surprising since autistic children appear to be detached from normal social, cognitive and linguistic experiences during the early developmental period. Thus, atypical hemisphere functioning is perhaps to be expected.

Lamondella has suggested that the two hemispheres may process limbic input differentially. This allows some connecting threads to be drawn between his model and the hemispheric dysfunction proposals. Lamondella notes support for the hypothesis of differential interaction of right and left hemispheres with limbic functions and suggests that integration of the two functional areas may occur via systems which could in turn be part of the limbic system. Further exploration of such a hypothesis may well be very profitable. Fein *et al* (1984*a*) have been critical of the hypothesis of left hemisphere dysfunction on the grounds that it lacks corroborative anatomical and physiological data, and are sceptical regarding its applicability to low functioning cases and the perceived overemphasis of the language disorder in autism with de-emphasis of the social impairments. They also note the marked individual differences across cases and the likelihood of similar variation in the biological/neural substrates of the disorder. However, this does not mean that investigations permitted by more sophisticated techniques of measurement of brain function at various levels will not prove illuminating on this proposal.

It is also helpful to consider Luria's (1973) model of brain function, which is based on the idea of three zones—primary, secondary and tertiary—each zone being dependent on adequate development of the preceding structures and functions. Abnormality in the primary functional area leading to inadequacy in processing of sensory information will in turn affect higher cortical processes. The development and interaction of these zones in young children is seen as a 'bottom-upwards' process, with intact lower zones necessary for adequately functioning higher zones—the secondary and tertiary areas. Thus multiple impairments in a number of areas can occur as a consequence of insult or deficits in a single primary area. This principle of hierarchial development of the brain could then explain the varying consequences of early damage to the limbic system (see also Tanguay & Edwards, 1982).

Conclusions

Consideration of the research concerned with biological factors in childhood autism shows that we appear to have made little progress in the search for specific aetiologies. It seems that we may not be very much closer to finding causes than we were 40 years ago when the search began. There are some obvious reasons for this lack of progress despite considerable quantities of research. In summary, they are:

(a) the use of heterogeneous samples of autistic children who, whilst they all exhibit autistic behaviour, do so for many different and sometimes obvious reasons;
(b) methodological and measurement problems in biological research which makes findings difficult to replicate and to interpret;
(c) lack of normative developmental data in most of the areas studied making comparisons between autistic and other groups difficult to interpret;
(d) failure to take account of retardation factors in sample selection and measurement;
(e) the primitive nature of our knowledge of brain physiology and of the functional relationships between brain and behaviour.

It certainly appears at this stage that a search for a unique cause for autism may be fruitless. A variety of neuropathological conditions may produce broadly defined behaviour symptoms. Wing (1981) and others argue strongly, and many researchers who work with autistic children imply, that autism may not be a specific disorder but can occur in many very different types of children with many different underlying problems. If the search for aetiological factors in childhood autism is to be more productive in future, it will be necessary to focus on 'pure' cases (as far as this is possible), who are preferably high functioning, thus minimising the influence of retardation. Equally important is hypothesis-testing on the basis of theoretical models that could account for the core features of the disorder and that arise from, and are consistent with, current neuropsychological data.

References

August, G. & Lockhart, L. (1984) Familial autism and the fragile-x chromosome. *Journal of Autism and Developmental Disorders*, **14**, 197–204.

——, Stewart, M. & Tsai, L. (1981) The incidence of cognitive disabilities in the siblings of autistic children. *British Journal of Psychiatry*, **138**, 416–422.

Boucher, J. & Warrington, E. K. (1976) Memory deficits in early infantile autism: some similarities to the Amnesic Syndrome. *British Journal of Psychology*, **67**, 73–87.

Campbell, M., Rosenbloom, S., Perry, R., George, A., Kricheff, I., Anderson, L., Small, A. & Jennings, S. (1982) Computerized axial tomography in young autistic children. *American Journal of Psychiatry*, **139**, 510–512.

Chess, S. (1971) Autism in children with congenital rubella. *Journal of Autism and Childhood Schizophrenia*, **1**, 33–47.

—— (1977) Follow-up report on autism in congenital rubella. *Journal of Autism and Childhood Schizophrenia*, **7**, 68–81.

Colby, K. M. & Parkinson, C. (1977) Handedness in autistic children. *Journal of Autism and Childhood Schizophrenia*, **7**, 3–9.

Courchesne, E., Kilman, B., Galambos, R. & Lincoln, A. (1984) Autism: processing of novel information assessed by event-related brain potentials. *Electroencephalography and Clinical Neurophysiology*, **59**, 238–248.

Damasio, A. R. & Maurer, R. G. (1978) A neurological model for childhood autism. *Archives of Neurology*, **35**, 777–786.

Damasio, H., Maurer, R. G., Damasio, A. R. & Chui, H. C. (1980) Computerized tomographic scan findings in patients with autistic behaviour. *Archives of Neurology*, **37**, 504–510.

Dawson, G. (1983) Lateralized brain dysfunction in autism: Evidence from the Halstead-Reitan Neuropsychological Battery. *Journal of Autism and Developmental Disorders*, **13**, 269–286.

——, Warrenburg, S. & Fuller, P. (1982) Cerebral lateralization in individuals diagnosed as autistic in early childhood. *Brain and Language*, **15**, 353–368.

——, Finley, C., Phillips, S. & Galpert, L. (1986) Hemispheric specialization and the language abilities of autistic children. (In press).

DeLong, G. R. (1978) A neuropsychologic interpretation of infantile autism. In *Autism: A Reappraisal of Concepts and Treatment* (eds M. Rutter and E. Schopler). New York: Plenum.

DeMyer, M. (1976) Motor, perceptual-motor and intellectual disabilities of autistic children. In *Early Childhood Autism* (ed. L. Wing). 2nd ed., Oxford: Pergamon Press.

——, Barton, S. & Norton, J. A. (1972) A comparison of adaptive, verbal and motor profiles of psychotic and non-psychotic subnormal children. *Journal of Autism and Childhood Schizophrenia*, **2**, 359–377.

——, ——, DeMyer, W., Norton, J., Allen, J. & Steele, R. (1973) Prognosis in autism: a follow-up study. *Journal of Autism and Childhood Schizophrenia*, **3**, 199–246.

——, Hingtgen, J. N. & Jackson, R. K. (1981) Infantile autism reviewed: a decade of research. *Schizophrenia Bulletin*, **7**, 388–451.

Derryberry, D. & Rothbart, M. (1983) Emotion, attention and temperament. In *Emotion, Cognition and Behavior* (eds C. Izard, J. Kagan and R. Zajonc) New York: Cambridge University Press.

Deslauriers, A. & Carlson, C. (1969) *Your Child is Asleep*. Illinois: Dorsey.

Fein, D., Skoff, B. & Mirsky, A. (1981) Clinical correlates of brainstem dysfunction in autistic children. *Journal of Autism and Developmental Disorders*, **11**, 303–315.

——, Humes, M., Kaplan, E., Lucci, D. & Waterhouse, L. (1984*a*) The question of left hemisphere dysfunction in infantile autism. *Psychological Bulletin*, **95**, 258–281.

——, Waterhouse, L., Lucci, D., Snyder, D. & Humes, M. (1984*b*) Cognitive functions in left and right-handed autistic children. Paper presented at the meeting of the International Neuropsychological Society, Houston, Texas.

Finegan, J. & Quarrington, B. (1979) Pre-, peri-, and neonatal factors and infantile autism. *Journal of Child Psychology and Psychiatry*, **20**, 119–128.

Folstein, S. & Rutter, M. (1977) Infantile autism: a genetic study of 21 twin pairs. *Journal of Child Psychology and Psychiatry*, **18**, 297–322.

Geschwind, N. (1965) Disconnection syndromes in animals and man. *Brain*, **88**, 237–294.

Geschwind, N. & Galaburda, A. (1985) Cerebral lateralization. Biological mechanisms, associations, and pathology: I A hypothesis and a program for research. *Archives of Neurology*, **42**, 428–459.

Gillberg, C. & Gillberg, J. (1983) Infantile autism: a total population study of reduced optimality in the pre-, peri-, and neonatal period. *Journal of Autism and Developmental Disorders*, **13**, 153–166.

—— & Svendsen, P. (1983) Childhood psychosis and computed tomographic brain scan findings. *Journal of Autism and Developmental Disorders*, **13**, 19–32.

—— & Forsell, C. (1984) Childhood psychosis and neurofibromatosis — more than a coincidence. *Journal of Autism and Developmental Disorders*, **14**, 1–8.

——, Rosenhall, U. & Johansson, E. (1983*a*) Auditory brainstem responses in childhood psychoses. *Journal of Autism and Developmental Disorders*, **13**, 181–195.

——, Svennerholm, L. & Hamilton-Hellberg, C. (1983*b*) Childhood psychosis and monoamine metabolites in spinal fluid. *Journal of Autism and Developmental Disorders*, **13**, 383–396.

Harper, J. & Williams, S. (1975) Age and type of onset as critical variables in early infantile autism. *Journal of Autism and Child Schizophrenia*, **5**, 25–36.

Hauser, S. L., DeLong, G. R. & Rosman, N. P. (1975) Pneumographic findings in the infantile autistic syndrome. *Brain*, **98**, 667–688.

Hermelin, B. (1982) Thoughts and feelings. *Australian Autism Review*, **1**, 10–19.

Hetzler, B. & Griffin, J. (1981) Infantile autism and the temporal lobe of the brain. *Journal of Autism and Developmental Disorders*, **11**, 317–330.

Hier, D. B., LeMay, M. & Rosenberger, P. B. (1979) Autism and unfavorable left-right asymmetries of the brain. *Journal of Autism and Developmental Disorders*, **19**, 153–159.

Hoffmann, W. & Prior, M. (1982) Neuropsychological dimensions of autism in children: a test of the hemispheric dysfunction hypothesis. *Journal of Clinical Neuropsychology*, **4**, 27–42.

—— & —— (1986) Frontal lobe deficits in autistic children. In preparation.

Jackson, M. J. & Garrod, P. J. (1978) Plasma, zinc, copper and amino acid levels in blood of autistic children. *Journal of Autism and Childhood Schizophrenia*, **8**, 203–208.

James, A. L. & Barry, R. J. (1980) A review of psychophysiology in early onset psychosis. *Schizophrenia Bulletin*, **6**, 506–525.

Jones, V. & Prior, M. (1985) Motor imitation abilities and neurological signs in autistic children. *Journal of Autism and Developmental Disorders*, **15**, 37–46.

Kanner, L. (1943) Autistic disturbances of affective contact. *Nervous Children*, **2**, 217–250.

—— (1944) Early infantile autism. *Journal of Pediatrics*, **25**, 211–217.

Kolvin, I., Ounsted, C., Humphrey, N. & McNay, A. (1971) Six studies in the Childhood Psychoses. II—Phenomenology of Childhood Psychoses. *British Journal of Psychiatry*, **118**, 385–395.

Kuperman, S., Beeghly, J., Burns, T. & Tsai, L. (1985) Serotonin relationships of autistic probands and their first-degree relatives. *Journal of the American Academy of Child Psychiatry*, **24**, 186–190.

Lamondella, J. (1977) The limbic system in human communication. In *Studies in Neurolinguistics* (eds H. Whitaker and H. A. Whitaker). New York: Academic Press.

Links, P. S., Stockwell, M., Abichandani, F. & Simeon, J. (1980) Minor physical anomalies in childhood autism. 1: Their relationship to prenatal and perinatal complications. *Journal of Autism and Developmental Disorders*, **10**, 273–285.

Lobascher, M., Kingerlee, P. & Gubbay, S. (1970) Childhood autism: aetiological factors in 25 cases. *British Journal of Psychiatry*, **117**, 525–529.

Lotter, V. (1966) Epidemiology of autistic conditions in young children. I. Prevalence. *Social Psychiatry*, **1**, 124–137.

Luria A. R. (1973) *The Working Brain*. London: Penguin.

MacCulloch, M. & Williams, C. (1971) On the nature of infantile autism. *Acta Psychiatrica Scandinavia*, **47**, 295–314.

McCann, B. S. (1981) Hemispheric asymmetries and early childhood autism. *Journal of Autism and Developmental Disorders*, **11**, 401–411.

Meryash, D., Szymanski, L. & Park, S. (1982) Infantile autism associated with the Fragile-X syndrome. *Journal of Autism and Developmental Disorders*, **12** (3), 295–301.

Minton, J., Campbell, M., Green, W., Jennings, S. & Samit, C. (1982) Cognitive assessment of siblings of autistic children. *Journal of the American Academy of Child Psychiatry*, **21**, 256–261.

Niwa, S., Ohta, M. & Yamazaki, K. (1983) P300 and stimulus evaluation process in autistic subjects. *Journal of Autism and Developmental Disorders*, **13**, 33–42.

Ornitz, E. (1978) Neurophysiologic studies. In *Autism: A Reappraisal of Concepts and Treatment* (eds M. Rutter & E. Schopler). New York: Plenum Press.

——, Ritvo, E., Brown, M., La Franchi, S., Parmelee, T. & Walter, R. (1969) The EEG and rapid eye movements during REM sleep in autistic and normal children. *Electroencephalography and Clinical Neurophysiology*, **26**, 167–175.

——, Guthrie, D. & Farley, A. (1978) The early symptoms of childhood autism. In *Cognitive Deficits in the Development of Mental Illness* (ed. G. Serban). New York: Brunner, Mazel.

Piggott, L. R. (1979) Overview of selected basic research in autism. *Journal of Autism and Developmental Disorder*, **9**, 199–217.

——, Frohman, C., Ward, V. & Gottlieb, J. (1975) The effect of plasma from psychotic children on tryptophan uptake in chicken erythrocytes. *Neuropsychobiology*, **1**, 284–295.

Prior, M. (1979) Cognitive abilities and disabilities in infantile autism: A review. *Journal of Abnormal Child Psychology*, **7**, 357–380.

—— (1984) Developing concepts of childhood autism: The influence of experimental cognitive research. *Journal of Consulting and Clinical Psychology*, **52**, 4–16.

—— & Bradshaw, J. L. (1975) Hemispheric functioning in autistic children. *Cortex*, **15**, 73–81.

——, Gajzago, C. & Knox, D. (1976) An epidemiological study of autistic and psychotic children in the four eastern states of Australia. *Australia and New Zealand Journal of Psychiatry*, **10**, 173–184.

——, Tress, B., Hoffmann, W. & Boldt, D. (1984) Computed tomographic study of children with classic autism. *Archives of Neurology*, **41**, 482–484.

Ricks, D. M. & Wing, L. (1976) Language, communication and the use of symbols. In *Early Childhood Autism* (ed. L. Wing). New York: Pergamon Press.

Rimland, B. (1964) *Infantile Autism*. New York: Appleton-Century-Crofts.

Ritvo, E. (1979) *Autism: Diagnosis, Current Research and Management*. New York: Spectrum.

Ritvo, E., Yuwiler, A., Geller, E., Ornitz, E., Saeger, K. & Plotkin, S. (1970) Increased blood serotonin and platelets in early infantile autism. *Archives of General Psychiatry*, **23**, 566–572.

——, Ritvo, E. C. & Brothers, A. M. (1982) Genetic and immunohematological factors in autism. *Journal of Autism and Developmental Disorders*, **12**, 109–114.

——, Spence, A., Freeman, B., Mason-Brothers, A., Mo, A. & Marazita, M. (1985) Evidence for autosomal recessive inheritance in 46 families with multiple incidences of autism. *American Journal of Psychiatry*, **142**, 187–192.

Rosenblum, S., Arick, J., Krug, D., Stubbs, E., Young, N. & Pelson, R. (1980) Auditory brainstem evoked responses in autistic children. *Journal of Autism and Developmental Disorders*, **10**, 215–226.

Rutter, M. (1974) The development of infantile autism. *Psychological Medicine*, **4**, 147–163.

—— (1983) Cognitive deficits in the pathogenesis of autism. *Journal of Child Psychology and Psychiatry*, **24**, 513–532.

Satz, P. (1973) Left-handedness and early brain insult: An explanation. *Neuropsychology*, **11**, 115–117.

Schain, R. J. & Yannet, H. (1960) Infantile autism: An analysis of 30 cases and a consideration of certain neurophysiological concepts. *Journal of Pediatrics*, **57**, 560–567.

Schopler, E. (1983) New Developments in the definition and diagnosis of autism. In *Advances in Clinical Child Psychology* (ed. A. Kazdin) Vol. 6. New York: Plenum.

——, Andrews, C. & Strupp, K. (1979) Do autistic children come from upper-middle-class parents? *Journal of Autism and Developmental Disorders*, **9**, 139–152.

Simon, N. (1975) Echolalic speech in childhood autism: Consideration of possible underlying loci of brain damage. *Archives of General Psychiatry*, **32**, 1439–1446.

Skoff, B., Mirsky, A. & Turner, D. (1980) Prolonged brainstem transmission time in autism. *Psychiatry Research*, **2**, 157–166.

Spence, M. A. (1976) Genetic studies. In *Autism: Diagnosis, Current Research and Management* (ed. E. Ritvo). New York: Halstead/Wiley.

Stubbs, E. (1976) Autistic children exhibit undetectable hemagglutination-inhibition antibody titers despite previous rubella vaccination. *Journal of Autism and Childhood Schizophrenia*, **6**, 269–274.

——, Crawford, M., Burger, D. & Vanderbart, A. (1977) Depressed lymphocyte responsiveness in autistic children. *Journal of Autism and Childhood Schizophrenia*, **7**, 49–55.

——, Ash, E. & Williams, C. (1984) Autism and Congenital Cytomegalovirus. *Journal of Autism and Developmental Disorders*, **14**, 183–190.

Tanguay, P. (1977) Clinical and electrophysiological research. In *Autism: Diagnosis, Current Research and Management* (ed. E. Ritvo). New York: Spectrum.

—— & Edwards, R. M. (1982) Electrophysiological studies of autism: The whisper of the bang. *Journal of Autism and Developmental Disorders*, **12**, 177–184.

——, Edwards, R., Buchwald, J., Schwafel, J. & Allen, V. (1982) Auditory brainstem evoked responses in autistic children. *Archives of General Psychiatry*, **39**, 174–180.

TSAI, L. Y. (1983) The relationship of handedness to the cognitive, language and visuo-spatial skills of autistic patients. *British Journal of Psychiatry*, **142**, 156–162.

—— & STEWART, M. A. (1983) Etiological implication of maternal age and birth-order in infantile-autism. *Journal of Autism and Developmental Disorders*, **13**, 57–65.

——, STEWART, M. & AUGUST, G. (1981) Implication of sex differences in the familial transmission of infantile autism. *Journal of Autism and Developmental Disorders*, **11**, 165–173.

——, TSAI, M. & AUGUST, G. (1985) Brief report: Implication of EEG diagnosis in the subclassification of Infantile Autism. *Journal of Autism and Developmental Disorders*, **15**, 339–344.

WALDO, M., COHEN, D., CAPARULO, E., YOUNG, J., PRICHARD, J. & SHAYWITZ, B. (1978) EEG profiles of neuropsychiatrically disturbed children. *Journal of the American Academy of Child Psychiatry*, **17**, 656–670.

WING, L. (1980) Childhood autism and social class: A question of selection? *British Journal of Psychiatry*, **137**, 410–417.

—— (1981) Sex ratios in early childhood autism and related conditions. *Psychiatry Research*, **5**, 129–137.

YAMAZAKI, K., SAITO, Y., OKADA, F., FUJIEDA, T. & YAMASHITA, I. (1975) An application of neuroendocrinological studies in autistic children and Heller's syndrome. *Journal of Autism and Childhood Schizophrenia*, **5**, 323–332.

YUWILER, A., RITVO, E. R., GELLER, E., GLOUSMAN, R., SCHNEIDERMAN, G. & MATSUNO, D. (1975) Uptake and efflux of serotonin from platelets of autistic and nonautistic children. *Journal of Autism and Childhood Schizophrenia*, **5**, 83–98.

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A Self-rating Scale for Bulimia
The 'BITE'

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A new brief questionnaire, the Bulimic Investigatory Test, Edinburgh (BITE), for the detection and description of binge-eating is described. Data from two separate populations demonstrate satisfactory reliability and validity. The scale has measures of both symptoms and severity. All items in the DSM-III definition of bulimia and Russell's definition of bulimia nervosa are covered but the questionnaire is more than just an operationalised checklist of these diagnostic criteria.

Over the past few years, there has been increasing interest in the prevalence of binge-eating (bulimia nervosa). One study (Halmi *et al*, 1981) found a prevalence rate of 13% in a population of American college students. In order to identify binge-eaters the authors used a short questionnaire, which included items on all the DSM-III (American Psychiatric Association, 1980) criteria for bulimia and for bulimia nervosa as described by Russell (1979).

A more recent study by Pyle *et al* (1983) used a slightly more sophisticated questionnaire to look at the incidence of binge-eating among college freshmen. This study found a prevalence of 4.5% in women, which is substantially lower than the figures found by Halmi *et al* (1981). This questionnaire also used DSM-III criteria, with the inclusion of questions about behaviour in the past as well as at the time of completion. In addition, Pyle *et al* defined what they meant by the term 'binge-eating', something that Halmi *et al* did not.

Hawkins & Clements (1980) have produced a questionnaire designed to provide descriptive and quantifiable information about behaviour and attitudes in binge-eating. This is a small 19-item questionnaire, of which only nine items are used in the calculation of the score. Although it could be used as a method of quantifying binge-eating behaviour, it is not comprehensive enough for epidemiological work. Nor would it be sensitive enough to enable identification of those with less severe pathology.

Much descriptive work has been completed on bulimia nervosa (e.g. Russell, 1979; Palmer, 1979; Wardle & Beinhart, 1981; Fairburn, 1983). It would seem more appropriate, when designing a questionnaire concerned with a particular disorder, to incorporate all the currently acknowledged symptoms and behaviour associated with that disorder. However, all the previous studies have relied on the American DSM-III diagnostic criteria and have used these as a basis for the wording of the individual items. Often no attempt is made to define what is meant by such terms as 'binge-eat'. Such questionnaires were all designed for the individual studies of which these were part and were often not tested for reliability or validity on separate populations before use. In a comprehensive review of the available questionnaires for use with binge-eaters, Gandour (1984) concludes that those currently available have been little used and they lack data on their reliability and validity. However, she indicates that a reliable self-report instrument would have a number of important potential uses.

These methods of questionnaire design are likely to lead to an overestimate of the incidence rate through misinterpretation of the questions, and/or a loss of information about those suffering from a less severely disordered eating pattern. The present authors felt that there was a need for a more sensitive questionnaire that would allow such people to be identified. In addition, there was a need for a tried and tested tool that explored fully the symptoms and behaviour associated with bulimia nervosa.

A questionnaire of this type has been developed for anorexia nervosa by Garner & Garfinkel (1979), the Eating Attitudes Test (EAT). The EAT has been used successfully as a screening tool to identify both clinical (Garner & Garfinkel, 1979) and subclinical cases of anorexia nervosa (Button & Whitehouse, 1981). Although the EAT is a proven, reliable questionnaire, it was not designed for use with binge-eaters: the questions are specifically concerned with the behaviour and feelings associated with anorexia nervosa. A more

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References for this paper have been combined with those for the following paper and can be found on page 31.5. Similarly, all authors' affiliations are given at the end of the following paper, on page 29.

comprehensive measure of eating behaviour is the Eating Disorders Inventory (EDI) Garner *et al*, 1983*a*). This is a questionnaire designed to assess a broad range of attitudes and behavioural aspects of anorexia nervosa and bulimia. It consists of eight subscales measuring dimensions such as drive for thinness, body dissatisfaction, bulimia and maturity fears. This instrument is a major advance in the comprehensive assessment of anorexia nervosa. However, it was not designed for use with binge-eaters. The population on which it was validated consisted of 129 anorexic subjects and did not include a group of binge-eaters that met DSM-III criteria. Furthermore, it is too cumbersome for use as a quick screening instrument.

Although binge-eaters share some common features with anorexics, they differ in many ways (Russell, 1979). Binge-eating is a very secret behaviour, and it is helpful to subjects completing a questionnaire if they can see that the questions are relevant to their problem.

In summary, the aim of the present study was to design and validate a questionnaire which would identify binge-eaters, provide clinical information on cognitive and behavioural aspects of the disorder and have properties similar to those of the EAT. The questionnaire was to include the most pertinent signs and symptoms associated with binge-eating described currently in the literature. This could then be used both as an epidemiological tool for identifying cases and subclinical cases in a given population and as a screening device for use in outpatient clinics. In addition, it would serve as a useful tool in a treatment setting, allowing severity to be quantified as well as being a measure of the response to treatment.

Method

Questionnaire design

An initial list of all the symptoms and behaviour associated with binge-eating was compiled from the current literature. In particular, those symptoms described by Palmer (1979), Russell (1979) and Bruch (1975) were included, as well as those listed in DSM-III. From these symptoms a preliminary list of questions was devised, which was given to a group of subjects, both normal controls ($n=10$) and binge-eaters ($n=7$). The binge-eaters were all currently receiving treatment from one of the authors.

After discussion with the subjects, all those items that were either misinterpreted or found to be ambiguous were deleted or reworded. A final list of 40 questions was then drawn up, the Bulimic Investigatory Test, Edinburgh (BITE). This list contained seven items concerned with dieting behaviour, 27 concerned with symptoms and behaviour associated with binge-eating and six concerned with more specific information on the frequency of the most significant behaviour.

For the pilot studies, a further section of questions intended to elicit information about eating patterns and demographic factors, e.g. height, was included in the BITE.

Pilot studies

Two independent groups of binge-eaters and normal controls were used to test the validity of the BITE. An important test of the cross-validity of a questionnaire is its ability to predict a case when it is applied to a new group not involved in its original preparation. Accordingly, two separate studies were undertaken. The first study was designed to test the face validity of the questionnaire and its acceptability to subjects. Following statistical analysis, the results from this study were used to decide which items should be included in the final form. The second study was designed to test the validity of the BITE as a sensitive and reliable measure of binge-eating, and its ability to identify cases accurately. Scoring was designed to be carried out by computer, using the SPSS system (Nie *et al*, 1975). However, scoring by hand can also be done quickly and easily.

First study subjects

For the first study the subjects were 15 female binge-eaters (Binge 1) and 40 normal controls (Control 1) (13 males, 27 females). The binge-eaters were all at various stages of treatment by one of the authors. The control group included various members of the hospital staff and a group of third-year medical students.

First study results

The BITE was given to both groups of subjects and then scored. On the basis of the scores on the 27 questions on symptoms, the subjects were divided into two groups, high scorers (total score >20) and low scorers (total score <20). Sixteen subjects were high scorers (mean score $=33.73$, s.d. $=8.14$) and 39 low scorers (mean score $=5.9$, s.d. $=5.37$).

The high-scoring group contained 14 subjects from the Binge 1 group and two from the Control 1 group. In the low-scoring group there were 38 from the Control 1 group and one from the Binge 1 group. It seemed clear that the questionnaire had succeeded in identifying the bulimic cases. Statistical analysis confirmed that this result was highly significant ($\chi^2=95.69$, $P<0.00001$, 1 d.f.). The one bulimic patient who scored below 20 was at the end of treatment.

The individual items in the BITE were then analysed to determine whether they were significant determinants of group membership, again using the chi-squared test. All but three of the items concerning symptoms were significant at the level $P<0.05$ (1 d.f.), while 19 were significant at a level of $P<0.0001$. On the basis of this analysis, it was decided to include three items from the dieting questions in the calculation of the total score. The three non-significant items were retained as it was felt they provided important clinical information.

Following the results of the first study, we subdivided the questionnaire into two subscales. The first subscale is

TABLE I
Comparison of subjects and controls for the first and second studies

Group	*n*	*Age (years, mean ± s.d.)*	*Height (cm, mean ± s.d.)*	*Weight (kg, mean ± s.d.)*
Binge 1	15	24.3±5.7	163.3±6.2	59.5±11.7
Control 1	40	24.2±5.5	168.2±9.9	61.3±9.8
Binge 2	32	25.3±6.0	163.5±3.4	62.1±10.1
Control 2	32	24.8±5.0	165.2±7.3	57.4±5.8

made up of 30 items relating to symptoms, behaviour and dieting, and is called the Symptom Subscale. The second subscale contains the six items measuring the severity of behaviour as defined by its frequency, and is called the Severity Scale.

The Symptom Subscale has a maximum possible value of 30, with a score of 20 as the criterion score. The Severity Subscale has a maximum score of 39 and a score of five or above is considered to be clinically significant. A total combined score of 25 or above is considered to be indicative of the presence of a severely disordered eating pattern.

Second study subjects

The subjects were 32 female binge-eaters (Binge 2), each of whom fulfilled DSM-III criteria for bulimia. These women took part in the study before commencing treatment. The control group (Control 2) comprised 32 normal females, including members of the hospital staff and psychology students. Demographic data on the subjects from both studies are presented in Table I.

Second study results

The BITE was completed by the second group of binge-eaters and normal controls. None of the normal control group produced a Symptom Score (mean = 2.94, s.d. = 2.94) or a Severity Score (mean = 0.44, s.d. = 0.29) above the cut-off points (20 and 5 respectively). All the binge-eaters scored above the cut-off on the Symptom Subscale (mean = 26.03, s.d. = 2.25), (see Fig. 1). All the binge-eaters scored above the cut-off score of 25 for the total score (mean = 36.19, s.d. = 4.47). Two subjects had a Severity Score of 4, the rest scored 5 or above (mean = 10.16, s.d. = 3.63). A total of 20 (62.5%) of the control subjects scored a Severity Score of 0. There is a highly significant difference between the total scores of the two groups ($t = 31.68$, $P < 0.001$, 62 d.f.) and also between both the Symptom Subscale and the Severity Subscale scores ($t = 35.29$, $P < 0.05$, 62 d.f. and $t = 14.65$, $P < 0.05$, 62 d.f. respectively).

Reliability

The inter-item reliability coefficient for all items on each subscale was calculated using the α reliability coefficient. Using both the subjects and the controls from the second study, α was 0.96157 for the Symptom Subscale and 0.62342 for the Severity Subscale. Thus, the items in both subscales demonstrate a high degree of internal consistency.

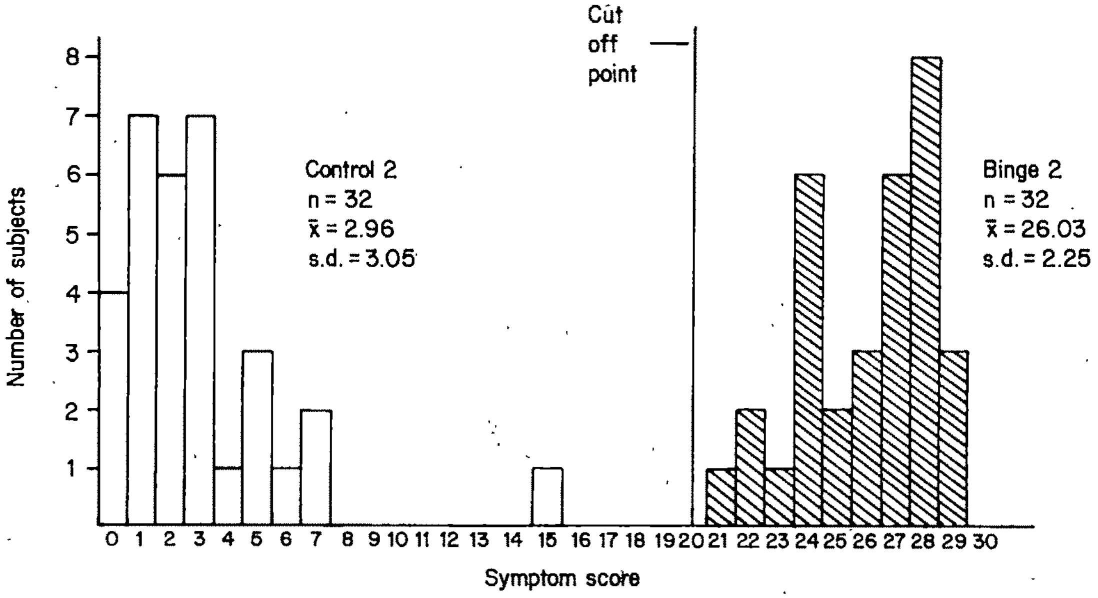

FIG. 1 This figure shows the scores of the Control 2 and Binge 2 subjects on the Symptom Subscale.

TABLE II
Correlation between BITE and EDI, EAT scores

Measure correlated with BITE		*Pearson's correlation coefficient* *R*	*No. of subjects* *N*	*Significance* *P<*
EDI	Drive to thinness	0.589	57	0.001
	Binge-eating	0.678	57	0.001
	Body dissatisfaction	0.352	57	0.01
	Ineffectiveness	0.506	57	0.001
	Perfectionism	0.143	57	NS
	Interpersonal distrust	0.227	57	NS
	Interoceptive awareness	0.519	57	0.001
	Maturity fears	0.438	57	0.001
EAT	Total	0.697	72	0.0001
	Diet subscale	0.621	72	0.0001
	Binge subscale	0.681	72	0.0001
	Oral control	0.232	72	NS

Relationship between the BITE and other measures

Scores on the BITE from the second group of binge-eating subjects (Binge 2) were compared with their scores on the EAT and the EDI (Garner & Garfinkel, 1982). The mean scores for the Binge 2 subjects on the EAT was 43.5 and the mean score on the bulimia subscale of the EDI was 10.5. The correlations are shown in Table II.

Third study subjects

Twenty-seven subjects in a treatment study completed the BITE before treatment, 8 weeks into treatment and at the end of 15 weeks treatment, to assess whether the questionnaire was sensitive to change. These subjects were all female, all met DSM-III criteria for bulimia and were bingeing at the rate of at least four times per month at the start of the study. The mean rate of bingeing was 7.2 times per week, at the start of the treatment study. The age-range of these subjects was 16–42 years, with a mean age of 22 years. The study involved a comparison of behaviour therapy, cognitive therapy and group psychotherapy. The preliminary results have been reported elsewhere (Freeman *et al*, 1985).

Third study results

Table III shows the scores on the BITE for these 27 subjects before starting treatment for bulimia, after 8 weeks and at the end of therapy. Significant reduction in scores occurred between 0–8 weeks and 8–15 weeks, showing that the BITE is highly sensitive to change.

TABLE III
Change of BITE scores with treatment

	n	*Mean*	*s.d.*	*t value*	*2-tail P*
Pre-treatment	27	33.85	5.97		
				8.24	0.000
Mid-treatment	27	15.03	8.60		
				3.78	0.001
End-treatment	27	8.29	6.02		

Fourth study subjects (test–retest reliability)

To assess test–retest reliability, 30 non-case female controls completed the BITE on two occasions separated by at least 1 week. These subjects were members of hospital paramedical staff and mothers whose children attended a university day nursery. In addition, ten women who were DSM-III bulimia cases completed the BITE on two occasions separated by 15 weeks. These were subjects who were part of a waiting-list control group for another study. All ten subjects met DSM-III criteria at the end of the waiting-list period as well as at the beginning.

Fourth study results (test–re-test reliability)

Mean scores for the 30 non-case women who completed the BITE on two occasions were as follows: first occasion 3.33 (s.d. 2.57, range 0–9), second occasion 3.22 (s.d. 2.27, range 0–9). A Pearson coefficient of $R = 0.86$ ($P < 0.0001$) shows a high positive correlation between the two sets of scores. Mean scores for the ten bulimic women who completed the BITE on two occasions were as follows: first occasion 34.4 (s.d. 3.13, range 29–38), second occasion 31.8 (s.d. 5.39, range 21–42). A Pearson coefficient of $R = 0.6825$ ($P < 0.05$) shows a high positive correlation.

Discussion

It is important that a questionnaire of this type should be easy to administer, acceptable to subjects and simple to score. The BITE meets all these requirements. It is self-explanatory and can easily

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be given to large numbers of subjects for completion. Those subjects who have significant scores can be identified easily. The average time for completion of the questionnaire is less than 10 min. Those subjects who took part in the pilot studies reported that the BITE was very acceptable and many commented on the pertinence of the questions.

The sensitivity of any new screening instrument is very important. The results clearly indicate that the modified BITE produces neither false positives nor false negatives. It is able to distinguish binge-eaters clearly from normal subjects while at the same time picking up less severe levels of pathology.

As can be seen in Fig. 1, one subject from the Control 2 group had a Symptom Score of 15. This score would suggest that the subject had a disturbed, but not clinically significant eating pattern. This subject was interviewed and it became clear that she had unusual eating habits, but not of sufficient severity to be classified as a binge-eater. Similarly, two of the Control 1 subjects were found to have disordered eating patterns, again not clinically significant. The presence of subjects with eating problems in groups of randomly selected controls illustrates the problem of finding 'normal' control subjects. Binge-eating is not a discrete clinical state; there are many intermediate subclinical stages between normality and a DSM-III diagnosis of bulimia nervosa. A shorter questionnaire relying on a few key questions is unlikely to have been sensitive enough to identify and classify correctly such subclinical cases.

Furthermore, the BITE would appear to be sensitive to changes in both symptoms and behaviour during and after treatment. In this capacity it should serve an invaluable role as a measure of response to treatment and allow comparison of the effectiveness of different treatment methods.

Unlike other questionnaires used to identify binge-eaters, the BITE does not differentiate subjects in an all-or-none fashion. There is a large range of possible scores, and subjects suffering from binge-eating of various degrees of severity can be detected. Those scoring less than the criterion score of 25, but more than a proposed lower cut-off of 10, are likely to reflect a subclinical group of subjects who have a disordered eating pattern.

The use of frequency of behaviour as a measure of severity is open to criticism. However, the authors feel that it will serve as an alerting sign to those using the questionnaire. A high score on this scale (10 or above) indicates that the subject is binge-eating and fasting or engaged in purging behaviours more than once a week. It is acknowledged that frequency of behaviour alone does not automatically imply a case. A possible diagnosis of binge-eating should only be made when the subject scores above 25 on the two scales combined. There are groups of patients, e.g. psychogenic vomiters, as described by Fairburn (1979), who may well score highly on the question concerning self-induced vomiting, but who would not meet the criteria for a diagnosis of binge-eating.

The BITE is put forward as a tested, valid questionnaire, designed specifically for use by those interested in binge-eating. There is a need for such an instrument for use in the field of eating disorders, which will allow workers to look at binge-eating in a systematic way. The EAT has been used widely. However, it is of limited use when looking at the symptoms of binge-eating. It is hoped that the introduction of the BITE will provide as useful a measure of binge-eating tendencies as does the EAT of anorexia nervosa.

The significant positive correlation between the EAT and BITE scores for the Binge 2 subjects does not mean that the two questionnaires are measuring the same clinical dimension. Both instruments are measures of abnormal eating patterns and attitudes to eating, but the behaviour with which each is concerned differs significantly. It would not be possible to assign a DSM-III diagnosis of bulimia to a subject on the basis of their score from the EAT. The BITE provides such information as well as detailed clinical information concerning a subject's individual difficulties. In addition, it allows the clinician a method of quantifying the severity of the problem as defined by frequency of behaviour and the presence or absence of different purging behaviours.

Until now, all those concerned with eating disorders have had to design their own questionnaires. These are often not tested for reliability and validity on a separate population before use. The BITE is put forward as a tried and tested alternative that can be used easily for a range of different purposes. It can be used in epidemiological studies concerned with incidence rates in various populations; it can also be used as a measure of severity of symptoms and response to treatment by both psychiatrists and GP's in outpatient work. Its ease of administration and scoring makes it ideally suited to these tasks.

There is still a great deal of ignorance concerning both the incidence and prevalence rates of binge-eating. Fairburn (1983) states that bulimia nervosa may well be an important undetected source of psychiatric morbidity. The BITE should go some way to help us gain a better understanding of the true prevalance rate of binge-eating.

Appendix 1

BITE instructions for administration and scoring

Uses

The BITE is a 33-item self-report measure, designed to identify subjects with symptoms of bulimia or binge-eating. It can be used to identify binge-eaters in a given population or as a screening instrument for use in a clinical setting. In addition, it serves as a useful measure of severity and response to treatment. The BITE consists of two subscales: the Symptom Scale, which measures the degree of symptoms present, and the Severity Scale, which provides an index of the severity of bingeing and purging behaviour as defined by their frequency. Scores on the Symptom Scale can be subdivided into three groups: high, medium and low scores. Those subjects achieving a high score have a high probability of meeting the DSM-III criteria for bulimia and Russell's (1979) criteria for bulimia nervosa. An additional front data sheet accompanies the BITE, which provides useful demographic data relevant to the study and treatment of binge-eating. Use of this data sheet is optional; it does not contribute to the subject's final score.

Administration

When the BITE is used as a screening instrument or in survey work, the subjects should be asked to complete the questionnaire based on their feelings and behaviour over the past 3 months. Where the BITE is to be used as a measure of response to treatment, only the past month should be considered.

Scoring

Symptom Scale

All the questions, with the exception of the three starred (6, 7 and 27), make up the Symptom Scale. The underlined questions (1, 13, 21, 23, and 31) score one point for a 'No' response. The remaining 25 items score one point for a 'Yes' response. The maximum possible score is 30.

Severity Scale

The three starred items (6, 7 and 27) comprise the Severity Scale. The total score is the sum of the numbers corresponding to the circled responses.

Interpretation of results

Symptom Scale

In general the scorers on this scale can be subdivided into three main groups; high scorers with a score of 20 or more, medium scorers with a score of 10–19 and low scorers with a score below 10.

A symptom score of 20 or more indicates a highly disordered eating pattern and the presence of binge-eating. There is a high probability that a subject who achieves such a score will fulfil DSM-III criteria for a diagnosis of bulimia.

A symptom score in the medium range (10–19) suggests an unusual eating pattern, but not to the extent that a subject in this range would meet all the criteria for a diagnosis of bulimia. An example of this might be a compulsive eater who eats excessively but does not binge-eat. A score in the 15–19 range should certainly be followed up by an interview. Subjects in this category may well reflect a subclinical group of binge-eaters, either in the initial stages of the disorder or recovered bulimics.

A symptom score in the low range (0–10) falls within normal limits. Such a score indicates the absence of both compulsive eating and binge-eating.

Severity Scale

The severity scale measures the severity of bingeing and purging behaviour, as defined by its frequency. A score of 5 or more on this scale is considered clinically significant. A score of 10 or more indicates a high degree of severity. A significant score on this scale should ideally be followed up by interview, regardless of the symptom score.

A high score on this scale alone may identify the presence of psychogenic vomiting, or laxative abuse, in the absence of binge-eating.

Any score on the severity scale should be checked against the relevant question in order to check for this type of behaviour.

Appendix 2

Bulimic Investigatory Test, Edinburgh (BITE)

Optional front data sheet

1. What is your sex? MALE 1 FEMALE 2 (*please circle number*)

2. Are you:
MARRIED 1 SINGLE 2 DIVORCED 3 SEPARATED 4 WIDOWED 5

3. What is your occupation? ______________

4. If married, what is your spouse's occupation? ______

5. What is your age? ________years

6. What is your height? ______feet ______inches, or ______cm

7. What is your weight? ______stone ______pounds, or ______kg

8. What is the most that you have ever weighed? ______stone ______pounds, or ______kg

9. What is the least that you have weighed at your present height? ______stone ______pounds, or ______kg

10. What would your ideal weight be if you could choose it? ______stone ______pounds, or ______kg

11. Do you feel yourself to be VERY OVERWEIGHT 5
OVERWEIGHT 4 (*please*
AVERAGE 3 *circle*
UNDERWEIGHT 2 *number)*
VERY UNDERWEIGHT 1

12. Do you have regular periods? (if applicable) YES 1 NO 2

13. How often, on average, do you eat the following meals?

	EVERY-DAY	5/7 DAYS	3/7 DAYS	1/7 DAYS	NEVER	
BREAKFAST	1	2	3	4	5	
LUNCH	1	2	3	4	5	(*circle*
DINNER	1	2	3	4	5	*number*)
BETWEEN MEAL SNACKS	1	2	3	4	5	

14. Have you ever consulted someone in a professional capacity for advice on dieting/eating? YES 1 NO 2

15. Have you ever been a member of a slimming club? YES 1 NO 1

16. Have you ever suffered from any type of eating disorder? YES 1 NO 2

. . . . . if yes, please give details over:

Bulimic Investigatory Test, Edinburgh

1. Do you have a regular daily eating pattern? YES NO

2. Are you a strict dieter? YES NO

3. Do you feel a failure if you break your diet once? YES NO

4. Do you count the calories of everything you eat, even when not on a diet? YES NO

5. Do you ever fast for a whole day? YES NO

*6. . . . If yes, how often is this?
EVERY SECOND DAY 5 2–3 TIMES A WEEK 4
ONCE A WEEK 3 NOW AND THEN 2 HAVE ONCE 1

*7. Do you do any of the following to help you lose weight? (*circle number*)

	Never	Occasionally	Once a Week	2–3 Times Week	Daily	2–3 Times a Day	5+ Times a Day
TAKE DIET PILLS	0	2	3	4	5	6	7
TAKE DIURETICS	0	2	3	4	5	6	7
TAKE LAXATIVES	0	2	3	4	5	6	7
MAKE YOURSELF VOMIT	0	2	3	4	5	6	7

8. Does your pattern of eating severely disrupt your life? YES NO

9. Would you say that food dominated your life? YES NO

10. Do you ever eat and eat until you are stopped by physical discomfort? YES NO

11. Are there times when all you can think about is food? YES NO

12. Do you eat sensibly in front of others and make up in private? YES NO

13. Can you always stop eating when you want to? YES NO

14. Do you ever experience *overpowering* urges to eat and eat and eat? YES NO

15. When you are feeling anxious do you tend to eat a lot? YES NO

16. Does the thought of becoming fat *terrify* you? YES NO

17. Do you ever eat large amounts of food rapidly (not a meal)? YES NO

18. Are you ashamed of your eating habits? YES NO

19. Do you worry that you have no control over how much you eat? YES NO

20. Do you turn to food for comfort? YES NO

21. Are you able to leave food on the plate at the end of a meal? YES NO

22. Do you deceive other people about how much you eat? YES NO

23. Does how hungry you feel determine how much you eat? YES NO

24. Do you ever binge on large amounts of food? YES NO

25. . . . If yes, do such binges leave you feeling miserable? YES NO

26. If you do binge, is this only when you are alone? YES NO

*27. If you do binge, how often is this?
HARDLY EVER 1 ONCE A MONTH 2
ONCE A WEEK 3 2–3 TIMES A WEEK 4
DAILY 5 2–3 TIMES A DAY 6

28. Would you go to great lengths to satisfy an urge to binge? YES NO

29. If you overeat do you feel *very* guilty? YES NO

30. Do you ever eat in secret? YES NO

31. Are your eating habits what you would consider to be normal? YES NO

32. Would you consider yourself to be a compulsive eater? YES NO

33. Does your weight fluctuate by more than 5 pounds in a week? YES NO

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Physical and Psychological Characteristics of Five Male Bulimics

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The case histories of five men who met DSM-III criteria for bulimia and details of their physical characteristics are given. Various eating disorder questionnaires were administered and the results indicated that most of these instruments would not have identified the men as suffering from an eating disorder. The necessity of caution in asserting the prevalence of bulimia using these measures is emphasised.

Bulimia as defined by DSM-III (American Psychiatric Association, 1980) is an eating disorder characterised by recurrent episodes of binge-eating which leave the patient feeling depressed and guilty. There is an awareness that the eating pattern is not normal and a fear of being unable to control the amount ingested. To avoid putting on weight, the patient will resort to inducing vomiting, abusing laxatives or diuretics, severely restrictive diets, or a combination of these methods.

The prevalence of bulimia in males has been estimated at between 0.4 and 5% of the US college population (Halmi *et al*, 1981; Pyle *et al*, 1983). In the UK, most estimates of bulimia in males are extremely small; Freeman & Henderson (1986) found one male bulimic in a survey of 625 male students; Clarke & Palmer (1983) found none in a postal survey of 120 male students. The prevalence of bulimia in women is much greater. Estimates of the incidence in US populations vary from 2.1 to 13% (Halmi *et al*, 1981; Pyle *et al*, 1983). Surveys in the UK have found the prevalence of women meeting DSM-III criteria for bulimia in certain populations to be between 1.9 and 4.5% (Cooper & Fairburn, 1983; Freeman & Henderson, 1986). As Fairburn (1984) has pointed out, the majority of these estimates are derived from the evidence of self-report questionnaires given to highly selected populations, usually of students. There is no reason to suppose that students are particularly disposed to eating disorders; in fact the most recent survey, of consecutive patients in a family practice, found that 23% of the women and 10% of the men had abnormal eating habits and concerns, as assessed by their replies to questions about binge-eating, vomiting etc. At least 10.9% met the criteria for bulimia (Zinkand *et al*, 1984). In addition, such self-report questionnaires have been derived from information on and validated for groups of women; there is no reason to suppose bulimic men would necessarily produce high scores.

Published reports of bulimia in men are rare, although anecdotal evidence abounds. Some physical and sexual data on 14 male bulimics has been provided by Herzog *et al* (1984), and Gwirtsman *et al* (1984) reported some clinical data and neuroendocrine findings for three male bulimics.

Button & Whitehouse (1981) used the EAT scale derived by Garner & Garfinkel (1979) to try to identify individuals with abnormal concerns with eating and weight. Using a cut-off point of 30 on this scale they found no men with problems. None of the 120 male students surveyed by Clarke & Palmer (1983) scored above 30 on the EAT scale and were therefore excluded from further study.

In this paper we report the case histories of five men who met DSM-III criteria for bulimia. Their scores on various questionnaires, including the EAT, are compared with available norms.

Subjects

Four of the men were referred by their GPs to a treatment study of bulimia. The first case was identified during the student survey conducted by Freeman & Henderson (1986).

Case 1

This 20-year old first-year student of divinity had been bingeing two or three times per week for at least 2 years. Periodically, he fasted for up to 6 days at a time and used enemas to control his weight. He also drank great quantities of diuretic tea. He exercised excessively, often spending 3 h/day lifting weights, walking and doing yoga. Unfortunately this subject did not consider himself to be in need of any help and would not cooperate with the assessment programme; however, questionnaire results are available. He had a psychiatric history in his home country (the USA) of psychoanalysis for unspecified problems including drug abuse. He did not complain of any physical effects of his disordered eating. He had no family history of affective disorder. Although this subject knew his eating was abnormal, as required by DSM-III criteria, he felt he had his problems with eating under control and therefore was not afraid of not being able to stop voluntarily. He did not meet Russell's

(1979) criteria for bulimia nervosa, as he neither induced vomiting nor took laxatives to control his weight. He did not have a morbid fear of becoming fat.

Case 2

An unemployed married man, aged 28 years and the father of two children, this subject had chronic kidney failure. He had been receiving dialysis treatment for 6 years. His binge-eating began when he lost his job 2 years before seeking help. When seen, he was bingeing several times per week and occasionally inducing vomiting. He did not abuse laxatives. He was much concerned at the potential harm he was doing himself by bingeing and vomiting, particularly since he invariably binged on foods not included in his therapeutic diet, e.g. bread and jam, chocolate and cakes. Both of his parents were extremely overweight, and this subject himself had been obese as a teenager. His mother then put him on a diet, which consisted in eating only meat, and he lost two stones in weight. He had been a very heavy drinker from the ages of 17 to 19, and at that time also had *grand mal* fits of unknown cause. He liked to eat in the street as soon as he had purchased the sweets, cakes etc. on which he preferred to binge. He said he dreamed of Mars Bars. When angry with his children—aged 1 and 2 years—he never hit them; he threw food at them. He had previously been diagnosed as suffering from a severe personality disorder with obsessional schizoid features. It was not clear how much his physical disorders contributed to his bulimia, but given his past history it was felt he still met DSM-III.

Case 3

A single, second-year chemistry student aged 20, this young man would control his eating for weeks at a time, and then binge and vomit continuously for as long as 5 days. His problems had started when he went to university. Having been very active at school in sports, he found the sedentary student life led to him putting on weight, since he continued to eat the same amount of food as before. This upset him and he began to diet. One morning he found himself to be over-eating at breakfast time and decided to keep on eating and then he induced vomiting. By the time he sought help, he felt his eating during the binge phases was completely out of control. Because he could not attend lectures or go out with his friends at such times, he felt his whole life was being affected, which made him feel very depressed. He would often eat the same meal again and again, e.g. he would have fish, chips and beans in the student refectory, followed by rice pudding. He would then induce vomiting. Having checked that there was no one around who had seen him eat the first meal, he would then eat fish, chips, beans and rice pudding again and induce vomiting. He felt he was more likely to binge when depressed. He had no previous psychiatric history, nor had anyone in his immediate family. He denied any feelings of physical ill-health as a consequence of his bingeing behaviour.

Case 4

A 27-year old community worker, presently employed as a care assistant in a home for mentally handicapped adults, this man had a 3-year history of inexplicable vomiting and regurgitation. During this period he had lost about 15 kg in weight, and was referred to the gastrointestinal unit, which reported no significant findings. About 1 year after the vomiting began, he started to binge—as a consequence, he said, of being so hungry after vomiting. At the time of presentation he was bingeing daily and vomiting about eight times a day. He bent over in order to regurgitate and, although he denied that he vomited on purpose, he admitted that he could not vomit unless he bent over, and sometimes not unless he also pressed his abdomen. He felt guilty about over-eating, as he saw it, and stole food from the kitchens where he worked. He ate secretly in the street to disguise the amount of food he consumed. He admitted that he was very pleased when people commented on how much more attractive he looked since he had lost so much weight.

The patient's problems seem to have stemmed from the period immediately after he and the rest of the family found out that his father, an alcoholic, was also homosexual. Their family was Catholic and deeply religious, and they rallied round to support the father, but the patient was very ashamed. The father's increasingly outrageous behaviour over the next few years, such as bringing his lovers home, etc., had finally caused a separation between the patient's parents. As a consequence, the mother was being treated for depression.

The patient suffered many physical symptoms resultant on his disordered eating; he complained of constant listlessness, cramps and pins and needles in his legs, rashes on his legs and a large number of colds and coughs. He had lost several teeth as a consequence of his frequent vomiting.

He did not meet Russell's (1979) criteria for bulimia nervosa, because he did not have a morbid fear of becoming fat.

Case 5

A 25-year old married man employed as a commis chef, this subject presented with a 2-year history of bingeing, self-induced vomiting, laxative and non-prescribed amphetamine abuse. His eating problems had begun when he was on a diet. He had dieted until he was 70% of normal body weight for a man of his height and age. At this time he found himself eating large quantities of sweets and cakes. He induced vomiting immediately and realised he had found a way to eat what he liked without putting on weight. Latterly he only induced vomiting occasionally and relied on amphetamines to control his hunger and laxative abuse to control his weight. Physical symptoms he experienced included peripheral paraesthesiae, frequent palpitations, severe swelling of the parotid glands, skin rashes, dehydration and occasional severe chest pain. He also complained of loss of energy and spasms in his calf muscles. He sometimes noticed his vomitus to be blood-stained. Although he had no previous psychiatric history or family history of psychiatric disorder, he was found to be clinically depressed, but failed to respond to two different antidepressants.

Procedure

All the subjects were assessed over a period of 2 or 3 weeks, to check that they met DSM-III criteria for bulimia, and to see whether or not they also met Russell's (1979) criteria for bulimia nervosa. They were weighed and their heights noted. A full personal history and history of weight and eating patterns were taken. The BITE (Henderson & Freeman, p. 18, this journal 1987), EAT 40 and EAT 26 (Garner & Garfunkel, 1979; Garner *et al*, 1982) and EDI (Garner *et al*, 1983*a*,*b*) were administered. The BITE is a self-rating 33-item questionnaire designed to detect subjects with bulimia or binge-eating. It has two scales, a Symptom Scale and a Severity Scale, which allow for the measurement of the degree of symptoms present and an index of the severity of bingeing and purging behaviours. The EDI, EAT 40 and EAT 26 are standard instruments, which assess the attitudes and objective features of eating disorders.

Subjects were asked to keep a diary of everything they ate and drank, and of how often they binged, vomited and abused laxatives. They were asked to note the occasions and quantities of other drugs taken, and of how often they engaged in exercise designed to counter the effects of bingeing.

TABLE I
Heights of the subjects and their weights as a percentage of MPMW[1]

		Weight as % MPMW[1]			
Case	*Height (m)*	*Present*	*Highest*	*Lowest*	*Ideal*[2]
1	1.83	109	115	93	96
2	1.76	89	124	79	90
3	1.76	93	101	91	92
4	1.90	89	118	89	100
5	1.85	103	103	79	82
	$\bar{x}=1.82$	$\bar{x}=97$	$\bar{x}=112$	$\bar{x}=84$	$\bar{x}=92$

1. MPMW—Matched Population Mean Weight—the mean weight of individuals of the same age, sex and height, as derived from the tables of the Society of Actuaries found in *Scientific Tables* (Diem & Lentner, 1970).
2. Ideal weight—the subjects' preferred weight

TABLE II
The ages, chronicity and frequency of bulimic behaviour

			Frequency per week of			
Case	*Age of onset (years)*	*Duration of illness (years)*	*Binge*	*Vomit*	*Laxative abuse*	*Other*
1	20	3	3	0	0	7[1]
2	26	2	7	2	0	0
3	19	1	5	5	0	0
4	24	3	7	56	0	0
5	23	2	2	0	7	7[2]

1. Enemas, diuretic tea, excessive exercise.
2. Amphetamine abuse.

Results

Tables I–III show the results obtained by the subjects on the various measures and questionnaires.

Discussion

Physical characteristics

Zinkand *et al* (1984) noted a tendency for bulimic men to be taller and heavier than men without eating disorders, but Herzog *et al* (1984) found them to be lighter. The bulimic men described here were taller and lighter than average (Table I). The thinner subjects' ideal weights were close to their present weights. The two heavier subjects, while of perfectly normal weight, wanted to be much thinner. Herzog *et al* (1984) found a mean age of onset in bulimic men of 18.6 years, while in this sample mean age of onset was higher (22.4 years).

Psychological characteristics

The scores the subjects obtained on the EDI and EAT would not have identified these men as having eating problems. Subjects' scores on the subscales of the EDI were more similar to those of normal than bulimic women. The cut-off scores on the EAT 40 and EAT 26 are 30 and 20 respectively; the subjects' scores were below these. Inspection seemed to indicate that the men's scores on the EAT and subscales of the EAT were between those of normal and bulimic women. It is interesting that these subjects did have scores for bulimia which were well elevated above those of normal men and women on both the EDI and EAT scales. It was on the less objective scales and more on those indicating attitude that they failed to show large differences from normals. One can speculate that this may be in part a result of a response bias operating; the subjects seemed reluctant to mark any response as 'always' or 'never'. Another reason why the subjects' scores were so low may have been that they were upset by the very obvious female bias to the questions in the EDI and EAT scales. One subject simply did not make any responses to the questions in the EDI asking how much one likes the shape of one's buttocks, hips and thighs etc.; another subject had clearly answered these questions at random. One item on the EAT 40 deals with menstruation and this was clearly inapplicable.

It is clear that the subjects' scores would have identified them as being bulimic using the BITE; the cut-off is 25 for the total score. The subjects' scores were much more similar to those of bulimic than to those of normal women.

TABLE III

The subjects' mean scores on the standard eating disorder questionnaires, the EDI (Garner et al, 1983a,b) and EAT (Garner et al, 1979, 1982), compared with norms where available. It includes subjects' scores on the BITE scale developed by Henderson & Freeman (1987)

	Bulimic men	*Bulimic women*[1]	*Normal men*[1]	*Normal women*[1]
EDI				
Desire for thinness	5.4	15.2	1.6	5.0
Bulimia	8.0	11.2	1.0	1.8
Bodily dissatisfaction	7.2	17.4	3.9	9.8
Ineffectiveness	6.8	13.9	1.6	2.1
Perfectionism	3.8	9.9	6.2	5.6
Interpersonal distrust	4.6	7.4	3.1	2.3
Interoceptive awareness	7.6	12.3	1.4	2.3
Maturity fears	1.6	5.9	2.7	2.3

	Bulimic men	*Bulimic women*[2]	*Normal women*[3]
EAT 40	28.5	55.5	15.4
EAT 26	17.8	38.4	9.9
Factor 1: Dieting	8.5	20.8	7.1
Factor 2: Bulimia	6.5	10.6	1.0
Factor 3: Oral control	2.8	7.0	1.9

						BITE scores Mean		
Subjects	*1*	*2*	*3*	*4*	*5*	*Bulimic men*	*Bulimic*[4] *women*	*Normal*[4] *women*
Symptom	19	25	21	20	24	21.8	26.03	2.94
Severity	8	7	6	7	19	9.4	10.16	0.44
Total	27	32	27	27	43	31.2	36.19	3.38

1. Garner *et al*, 1983*b*.
2. Garner & Garfinkel, 1979.
3. Garner *et al*, 1982.
4. Henderson & Freeman, 1987.

Although Herzog *et al* (1984) found evidence that 26% of the men with anorexia and bulimia in their study were homosexual, none of the five men here described admitted to any homosexual interests; all claimed to be exclusively heterosexual.

Cases 1 and 3 refused any treatment. Case 4 is still in treatment. After receiving a kidney transplant and undergoing intensive psychotherapy, Case 2 was still bingeing and vomiting, as was Case 5 after successful treatment with antidepressants.

Conclusion

Bulimia may be more common in men than has previously been supposed. Button & Whitehouse (1981) and Clarke & Palmer (1983) found no evidence of eating disorder in the groups of men they surveyed using the EAT. In view of the results described in this paper, it would seem that caution must be used in the administration of test instruments derived from and devised for women, as the pattern for men does not seem to be as clear-cut. Most of the subjects described here would have been missed if the most widely used instruments (i.e. the EDI and EAT) had been used for screening, yet all subjects had clinically significant and distressing pathology.

Appendix 1

Diagnostic criteria for bulimia nervosa

DSM-III (American Psychiatric Association, 1980)

A. Recurrent episodes of binge-eating (rapid consumption of a large amount of food in a discrete period of time, usually less than 2 h).

B. At least three of the following:

(a) Consumption of high-calorie, easily ingested food during a binge.

(b) Inconspicuous eating during a binge.

(c) Termination of such eating episodes by abdominal pain, sleep, social interruption or self-induced vomiting.
(d) Repeated attempts to lose weight by severely restrictive diets, self-induced vomiting or use of cathartics and/or diuretics.
(e) Frequent weight fluctuations greater than 10 pounds (4.5 kg) due to alternate binges and fasting.

C. Awareness that the eating pattern is abnormal and fear of not being able to stop voluntarily.
D. Depressed mood and self-depreciating thoughts following eating binges.
E. Bulimic episodes are not due to anorexia nervosa or any other known physical disorder.

Russell (1979)

A. Powerful intractable urges to over-eat.
B. They seek to avoid the fattening effects of food by inducing vomiting or abusing purgatives or both.
C. A morbid fear of becoming fat.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* 3rd ed. (DSM-III). Washington, DC: Division of Public Affairs, APA.

Bruch, H. (1975) *Eating Disorders*. New York: Basic Books.

Button, E. J. & Whitehouse, A. (1981) Subclinical anorexia nervosa. *Psychological Medicine*, **11**, 509–516.

Clarke, M. G. & Palmer, R. L. (1983) Eating attitudes and neurotic symptoms in university students. *British Journal of Psychiatry*, **142**, 299–304.

Cooper, P. J. & Fairburn, C. G. (1983) Binge-eating and self-induced vomiting in the community: a preliminary study. *British Journal of Psychiatry*, **142**, 139–144.

Diem, K. & Lentner, C. (eds) (1970) *Scientific Tables*. Basle: Geigy.

Fairburn, C. G. (1979) Self induced vomiting. *Journal of Psychosomatic Research*, **24**, 193–197.

—— (1983) Bulimia nervosa. *British Journal of Hospital Medicine*, 537–542.

—— (1983) Bulimia: its epidemiology and management. In *Eating and its Disorders* (eds A. J. Stunkard & E. Stellar). New York: Raven Press.

Freeman, C. P. L. & Henderson, M. (1986) A survey of Scottish students' eating patterns. (In preparation.)

——, Sinclair, F., Turnbull, J. & Annandale, A. (1985) Psychotherapy for bulimia: a controlled study. *Journal of Psychiatric Research*, **19**, 473–478.

Gandour, M. J. (1984) Bulimia: clinical description assessment, etiology and treatment. *International Journal of Eating Disorders*, **3**, 3–83.

Garner, D. M. & Garfinkel, P. E. (1979) The eating attitudes test: an index of the symptoms of anorexia nervosa. *Psychological Medicine*, **9**, 273–279.

——, Olmstead, M. P., Bohr, V. & Garfinkel, E. (1982) The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine*, **12**, 871–878.

——, Olmstead, M. P. & Polivy, J. (1983*a*) The eating disorder inventory: a measure of cognitive–behavioural dimensions of anorexia nervosa and bulimia. In *Anorexia Nervosa: Recent Developments in Research*. New York: Alan R. Liss.

——, Olmstead, M. P. & Polivy, J. (1983*b*) Development and validation of a multi-dimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, **2**, 15–34.

Gwirtsman, H. E., Roy-Burn, P., Lerner, L. & Yager, J. (1984) Bulimia in men: report of three cases with neuroendocrine findings. *Journal of Clinical Psychiatry*, **45**, 78–81.

Halmi, K. A., Falk, J. R. & Schwarz, E. (1981) Binge eating and vomiting: a survey of a college population. *Psychological Medicine*, **11**, 697–706.

Hawkins, R. C. & Clements, P. F. (1980) Development and construct validation of a self-report of binge-eating tendencies. *Addictive Behaviours*, **5**, 219–226.

Herzog, D. B., Norman, D. K., Gordon, C. & Pepose, M. (1984) Sexual conflict and eating disorders in 27 males. *American Journal of Psychiatry*, **141**, 989–990.

Nie, N. H., Hull, H. C., Jenkins, J. G., Steinbrenner, K. & Bent, D. H. (1975) *S.P.S.S., Statistical Package for the Social Sciences* 2nd ed. New York: McGraw-Hill.

Palmer, R. L. (1979) The dietary chaos syndrome: a useful new term? *British Journal of Medical Psychology*, **52**, 187–190.

Pyle, R. L., Mitchell, J. E., Eckert, D. E., Halverston, P. A., Neuman, P.A. & Goff, G. M. (1983) The incidence of bulimia in freshman college students. *International Journal of Eating Disorders*, **2**, 75–85.

Russell, G. (1979) Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychological Medicine*, **9**, 429–448.

Wardle, J. & Beinhart, H. (1981) Binge-eating: a theoretical review. *British Journal of Clinical Psychology*, **20**, 97–109.

Zinkand, H., Cadoret, R. J. & Widmer, R. B. (1984) Incidence and detection of bulimia in a family practice population. *Journal of Family Practice*, **18**, 555–560.

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Life Events, Vulnerability and Onset of Depression:
Some Refinements

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A prospective study of 400 largely working-class women with children living at home has once again demonstrated the major importance of long-term severe threatening life events in provoking caseness of depression. However, it again shows that only about one out of five women experiencing such an event go on to develop depression at a case level. This paper demonstrates that more sensitive characterisation of severe events can greatly increase the size of this association. This is done both by improving the description of the event itself and by taking into account various ways in which the event can 'match' characteristics of the women present at the first interview, well before the occurrence of the event or any onset of depression.

The Life Event and Difficulty Schedule (LEDS), the full version of which was produced in 1969, has consistently shown large associations between life events and the development of clinical depression in the general population (see Brown & Harris, 1978; 1986). The approach is investigator-based and attempts to deal with the meaning of events for particular individuals. Since bias may arise if a person who has developed depression is asked about the meaning of events, it is based on a *contextual* method of rating events. A judgement is made by the investigator about the likely meaning of the event, given the particular biography and current circumstances of the person, ignoring anything the person says about his or her reaction. Therefore contextual ratings deal with specific meaning in the sense of taking into account the circumstances surrounding a particular event or difficulty. Other commonly used life-event instruments have dealt with general meaning, in the sense that the meaning of a whole class of events (such as a birth) is used irrespective of the context in which the event occurs (Brown, 1981, 1983).

The LEDS is based on a semi-structured interview which is used to gather as full and coherent an account as possible of any incident which may be relevant to the research enquiry. The interviewer establishes the presence or otherwise of 40 types of possible event and also the occurrence of various kinds of ongoing difficulty. A set of previously developed rules is used to decide whether or not an event is to be included. Therefore, there is no question of including the death of a distant relative if this is not already covered by rules about what can and cannot be defined as an 'event'. Once the occurrence of an event or a difficulty has been established, a second level of interviewing allows the investigator to specify various aspects of the meaning of the event or difficulty for the individual. In assessing, say, the threat of having a third child in an overcrowded flat, raters make a judgement of what most persons in such circumstances would be likely to feel, by taking into account what is known of the person's plans and purposes, as these are reflected in his or her biographical circumstances. By ignoring self-reports about meaning, various potential sources of bias stemming from the respondent can be excluded. However, some of what is gained in terms of controlling bias may be lost in terms of precision of measurement. Much potentially valuable information must be ignored; strict rules do not permit feelings of, say, attachment or commitment to be taken into account when the degree of stress is assessed contextually. Since the presence of depression or the recent experience of a loss may inflate the reported amount of previous attachment to the lost object, such information cannot be used safely. This style of measurement is particularly concerned with controlling respondent biases often overlooked by other methods, but it also guards equally against various investigator biases. Manuals which give strict definitions of which incidents can be counted as events, and directories giving thousands of examples of precedents of contextual meaning, rated on the various scale-points (such as threat or loss), are designed to prevent interviewers from allowing knowledge of the respondent's symptoms or reported emotional responses to influence their ratings. Consensus meetings of other interviewers in the research team, who are blind to the subject's symptoms and reactions, provide a further check on investigator bias. The approach to difficulties is basically the same (Brown & Harris, 1978).

'Severity' of threat in the LEDS is assessed in terms of the immediate or more long-term impact of events and the ongoing impact of difficulties, but the first stage of the instrument that establishes their presence can be used as a basis for the measurement of a variety of other qualities, such as loss, danger and chances of resolution. Only one type of event has proved so far to be of central aetiological importance for depression—events which are *severe* on long-term contextual threat. They are in fact defined by two rating scales. They must be rated '1—marked' or '2—moderate' on a four-point scale of long-term threat, judged in contextual terms about 7-10 days after the event's occurrence, and must also be focused on the subject—either alone or jointly with someone else (Brown & Harris, 1978). Also only a restricted class of difficulties—termed *major* difficulties—appears to be of critical importance for depression. These must be at least marked (1-3) on a six-point scale of unpleasantness, involve non-health problems and have lasted 2 years (Brown & Harris, 1978). However, physical health difficulties are included occasionally as a major difficulty if they restrict a subject's plans or activities in some critical way: for example, causing her to give up a job, move house or postpone marriage, either in the case of her own physical health because it presents so serious a difficulty, or in the case of other people's health because she is involved in continuous responsibility for their care and supervision. These *provoking agents* form only a small minority of the events and difficulties recorded by the LEDS instrument.

There has been a good deal of uncertainty about the importance of the aetiological link between life events and the onset of depressive disorders. Results of research have been presented for the most part in terms of the apparently straightforward notion of 'variance explained' (r^2). The amount explained in these terms is invariably low even when an investigator-based instrument such as the LEDS is used. The percentage is usually no more than about 10% (for checklist respondent-based instruments it is considerably lower [Brown & Harris, 1986]). In the light of this, some have concluded that "life events are unlikely to have clinical or preventive importance" (Andrews & Tennant, 1978). However, the same conclusion would have to hold for aetiological agents of accepted importance such as heavy smoking and lung cancer. The paradox is simply the result of the fact that such an index takes into account that most heavy smokers do not develop lung cancer (and that most with a provoking agent do not develop depression). However, most instances of caseness of depression, when strictly defined, do have a severe event or major difficulty occurring before onset, just as most instances of lung cancer are associated with heavy smoking. An index frequently used in epidemiological research, Levin's attributable risk percentage, reflects this since it gives the proportion developing a condition that is associated with the putative causal risk factor, allowing for the fact that in some instances it will be associated by chance (Cole & MacMahon, 1971; Markush, 1977; Brown & Harris, 1986).

A review of onset of caseness of depression in 10 population studies of women in the general population, using the LEDS and similar high thresholds for defining a case of depression, shows that all agree that the majority of women experienced a provoking agent before onset. The actual proportion having a provoking agent ranges from 62 to 94% with an average of 83% (Cooper & Sylph, 1973; Brown & Harris, 1978; Brown & Prudo, 1981; Finlay-Jones & Brown, 1981; Costello, 1982; Campbell *et al*, 1983; Bebbington *et al*, 1984; Brown *et al*, 1985; Parry & Shapiro, 1986). The average attributable risk percentage is 73%. While this should strictly allow for the differences in length of the study period, it serves as a reasonable approximation of the order of effects that have been found (Brown, 1986).

However, the use of 'variance explained' does remind us of the need to understand why only about one out of five women experiencing a provoking agent goes on to develop depression at a caseness level. In recent years, most of the effort to explain this differential response has been channelled into exploring the role of vulnerability factors such as low self-esteem or lack of social support. In the way that we have used the concept of vulnerability, such factors are only associated with an increased risk when a provoking agent is also present. In this paper we explore a complementary approach—one that seeks to improve the sensitivity of the current LEDS contextual measures of threat. The paper uses material from a prospective enquiry. First, we deal with information concerning events and difficulties collected at the time of the follow-up interview—that is with additions to the current LEDS schedule. Later, material collected at the first interview is used to characterise these same events; this latter exercise can therefore only be carried out in a prospective enquiry.

Design of the enquiry

Working-class women with a child at home and living in an inner-city area (Islington, North London) were selected since research had suggested that they are particularly likely to develop depressive disorders (Brown & Harris, 1978). The cooperation of general practitioners was enlisted and they sent an introductory letter together with a screening questionnaire to women aged between 18 and 50 years, explaining the nature of the research. Women were only rejected if they did not meet the criterion of being from European or Caribbean backgrounds, having at least one child under 18 years of age living at home and a spouse or cohabiting partner who was in a manual occupation (Goldthorpe & Hope, 1974). In addition, all single mothers were included regardless of occupational or class criteria, as there was evidence that such women have a high rate of depressive disorder (e.g. Moss & Plewis, 1977; Surtees *et al*, 1983). For separated, divorced and widowed women who were living as 'single parents', the husband's occupation was taken to define class status if he had lived with the woman in the previous five years. For all other women not living with a man, their own current occupation was taken (for details, see Brown *et al*, 1986*a*).

The screening process identified 435 suitable women, of whom 91% (395/435) completed the first stage of the survey. The main investigation was conducted in two phases, approximately 1 year apart. In the first phase, measures of the quality of personal ties, support received and self-esteem were collected, together with an account of psychiatric disorder both at the time of interview and in the preceding 12 months. Two interviews, lasting in all between 3 and 4 hours, were usually necessary (O'Connor & Brown, 1984). The second phase comprised measures of life events and difficulties occurring during the follow-up year, together with actual social support received during any significant crises and the woman's response to it and details of any psychiatric disorder over the follow-up year. Severe life events and marked difficulties in the year before the first interview were also covered. Previous work suggests that these can be collected reliably over such a period of time (Neilson, 1984). A full interview was completed for 89% of the women.

A shortened version of the Present State Examination (PSE) instrument was used to collect material about basic symptoms (Wing *et al*, 1974; Cooper *et al*, 1977). Although the PSE was initially designed to cover the month immediately preceding interview, it was possible to modify the approach to examine the past 12 months, probing for episodes of distress and then collecting details about symptoms for any episode of affective disorder, even if this had cleared up by the time of interview. Once symptoms had been collected, indices of severity and diagnosis were derived by presenting the PSE symptoms to a panel of experienced raters who were blind to possible aetiological factors. Subjects considered to have had definite psychiatric disorders are classified as *cases* for the periods involved. For caseness of depression, at least four core symptoms are required in addition to depressed mood (Finlay-Jones *et al*, 1980). In all instances, many other symptoms were also rated and, in Islington, women judged to be cases of depression had an average of 19.1 (Brown *et al*, 1985). Experience with the PSE over the last 15 years does not leave much doubt that such cases are broadly at the level of severity of conditions treated by psychiatrists in outpatient practice. This threshold for caseness has recently been compared with two other well known thresholds, the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1978), and the ID threshold of the PSE (Wing & Sturt, 1978) in an extensive population study in Edinburgh. The Bedford College criterion for caseness was found to be higher than those of the ID/CATEGO system of other PSE users and the 'major depressions' of the RDC (Dean *et al*, 1983).

New measures

For cross-sectional analysis

Refinement of the severity threat rating

Only one addition to the rating of threat was introduced for the Islington survey. This was a straightforward extension of the present rating. A simple 'upper' and 'lower' division of severe events was developed with anchoring examples. An example of 'lower' is a woman working as a secretary, whose husband is warned of likely redundancy and where jobs in his specialised occupation are difficult to get. The event was rated low because the woman had a full-time job and the redundancy had not yet occurred. An 'upper' severity rating would be given to an event where redundancy had actually occurred and was already giving rise to considerable financial problems affecting the family.

Loss and danger

Two four-point ratings of loss and danger were developed in a previous study, although minor amendments were made to the loss rating for the current project (Finlay-Jones & Brown, 1981). Six classes of experiences among those with a severe event were considered to involve loss: (a) death, (b) separation, (c) loss of employment of subject or 'chief wage-earner', (d) loss of material possessions, (e) loss of physical health by subject and (f) loss of a cherished idea. The last included either disruption of a woman's expectation of a key person's commitment, faithfulness or trustworthiness, an event which might lead the subject to question these qualities in herself (e.g. learning her daughter has been taking heroin) or incidents that may be an even more fundamental challenge to identity (e.g. discovering that her husband is bisexual). Also included among losses are disappointments defined as severe events leading to a reversion of a previously unpleasant situation following a period when the problem had been dormant or had improved, and where it had apparently been reasonable for the subject to assume there might have been some more or less permanent change for the better. Events were not included where there had been no such revision and where the event underlined, as it were, the loss of a particular 'cherished idea' that had occurred more than 1 year before. Only a handful of 'disappointment' events were added to the total loss events. A second and independent rating was made of 'danger' which was defined as the degree of unpleasantness of a specific future crisis which might occur as a result of the event.

Chances of resolution

Each difficulty was rated on a nine-point scale in terms of the chances of its resolution. The ratings were considered in contextual terms, taking into account possible intervention by the subject or someone else. The rating also considered whether any resolution would be likely to involve other serious problems in the process. The scale has been used here as a simple dichotomy, contrasting difficulties considered to have at least some chance of resolution (or where it was unclear) with those which had little or no chance without creating major additional problems—that is 'low' chances of resolution.

'Matching'—difficulties

One major innovation is an attempt to explore the fruitfulness of the notion that the risk of depression is increased after a severe event when there is a *match* with certain characteristics of the woman and her life-context at the time of the first interview. At heart, the notion deals with the congruence of the meaning of the event, judged in contextual terms, with some earlier state. Three areas were considered as a basis for matching. One concerned marked difficulties present at the time of the first interview (as well as at the point when the event occurred) and is therefore referred to here under the heading of measures that can be used in cross-sectional analysis. The remaining two concerned commitment and conflict and required a prospective design.

For use in prospective studies

Commitment

At the first interview, each woman discussed at considerable length five areas of her life—children, marriage (or living with someone), housework, employment and other activities outside the home. For each area she was rated on a four-point scale in terms of her *commitment*. This concerned the idea of the role or activity rather than particular personal relationships or actual current activities. For example, like other women, single parents were rated in terms of their commitment and enthusiasm about the idea of marriage (or living with someone). But if a woman chose to express her commitment (or lack of it) in terms of her current situation we took this into account. For example, when a woman was married, the nature of her relationship with her husband often dominated her answers to our questions. We took such comments into account but also kept in mind that it was possible for a woman to be highly dissatisfied with her actual experiences in a given area and yet be committed to the idea of that area as worthwhile in general. For the present exercise, a woman was considered committed to a particular area if rated '1—marked. In addition, we asked women in detail about any relationship (other than with husband or child) they named as 'very close'. All but one-fifth of the women named someone and the average number was two (O'Connor & Brown, 1984; Brown *et al*, 1986*a*). Since we did not develop a rating of commitment for such ties, we counted a woman as committed if she rated '1—marked' on a four-point scale of dependency, made for every such close relationship. Although the notion of commitment is broader than that of dependency, the latter provides a reasonable approximation for the present exercise.

Role conflict

Role conflict was rated at first interview, particularly any arising from diverging obligations, such as that between domestic and external spheres. Conflict had to be reflected directly in the woman's actual social circumstances (e.g. full-time work *vs* time spent with children), although its rating in each instance was based on the feelings she expressed about it (Brown & Bifulco, 1985). Thus, a high role conflict rating ('1—marked' or '2—moderate' on a four-point scale) was given where a woman reported feeling strain or psychological tension between two such domains in her life, both of which made demands on her. Role conflict was also rated where strain was reported by the woman in the way she related to others: for instance, if she experienced conflict over having high hostility as well as high dependency needs in her key relationships, or if her desire to get close to people conflicted with her mistrust of others.

Response to severe events and difficulties in terms of hopelessness

A four-point rating of feeling of hopelessness in relation to a particular event or difficulty (or complex of related events and difficulties) was made. One rating involved the predominant pattern during the year and the other the woman's outlook towards the end of the follow-up period. This enabled us to characterise the response at the beginning of any crisis arising in the year and to take account of any change that might occur in the response to a subsequent life event that might precede an onset of depression. The ratings take into account the woman's reported response to her life as a whole in the context of the crisis. Therefore, hope could be high in spite of a devastating loss or an irresolvable difficulty, in the sense that feelings of hopelessness were not generalised to life as a whole. However, the rating gave special weight to the women's response to the particular crisis. Figure 1 gives a schematic outline of the basic ratings in terms of the two interviews—we use the terms *D-event*, *C-event* and *R-event* for matching difficulty, commitment and role conflict events respectively.

Reliability of measures

The inter-rater reliability of the well established measures such as the PSE has been shown to be high (Cooper *et al*, 1977; Wing *et al*, 1977). Similarly the occurrence, severity and dating of life events and difficulties have shown high inter-rater agreement, as well as high inter-respondent agreement, when a patient and a close relative have been interviewed separately by different interviewers (Brown & Harris, 1978, 1982; Tennant *et al*, 1979; Parry *et al*, 1981). High inter-rater reliability of loss and danger ratings has also been demonstrated (Finlay-Jones & Brown, 1981).

For measures new to the current study, the inter-rater agreement has also been high. When 20 interviews were rated independently by different raters, agreement on the 'upper' and 'lower' threshold of events reached 0.70 agreement (on kappa) and the scale measuring resolution of difficulties showed complete agreement. In terms of the

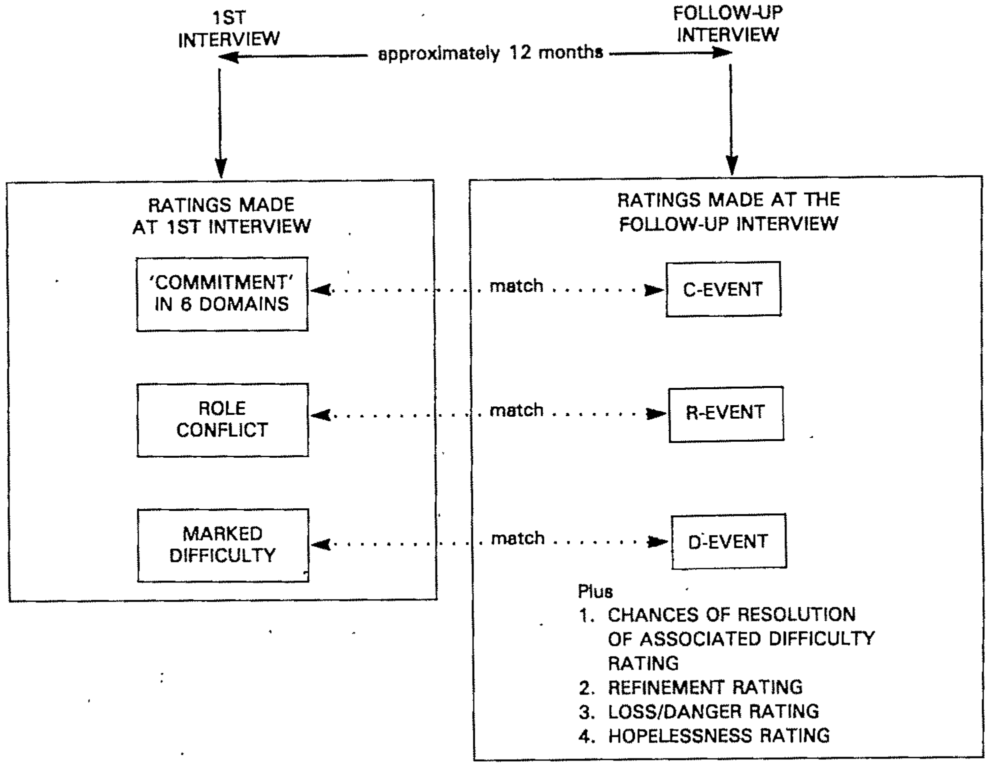

FIG. 1. Schematic outline of the ratings for key severe events.

matching scales, agreement on commitment also reached a satisfactorily high level (e.g. commitment to marriage reached a kappa of 0.89 and motherhood 0.88) as was role conflict (0.79).

A full inter-rater reliability of the hopelessness measure has not yet been undertaken, but inter-rater agreement on ratings based on transcribed descriptions was 0.80.

Results

In order to establish the causal importance of provoking agents, it is necessary to consider all 303 women followed up who were not suffering from depression at first interview. However, most of the questions tackled in this paper are concerned with whether this present measure of severe event can be improved and here it is sufficient to consider only the 130 women with a severe event in the follow-up year (or in some instances the 150 with either a severe event or major difficulty). The 26 weeks before onset has been taken for onset cases; but in order to maximise the number with a severe event, thereby increasing the sensitivity of the analysis, the whole follow-up period has been taken for those without an onset. (However, for the calculation of attributable risk percentage this would be misleading and a 6-month period must also be taken for those without onset.)

Retrospective measures

Refining the rating of severe event in the light of material collected at follow-up

Provoking agents in Islington were highly related to onset of depression. Of the 32 women with such an onset in the follow-up year, 29 had a severe event in the 6 months prior to onset; a number of studies have now concluded that severe events of aetiological importance almost always occur within 6 months of onset (Brown & Harris, 1986). One further woman had a major difficulty as her only provoking agent. We will concentrate first on the nature of the severe events occurring in the sample as a whole (see Table I). One simple descriptive rating of the LEDS divides events into nine categories involving either an interpersonal crisis with (1) husband or lover, (2) close relative or friend, (3) child, (4) the death of someone close, (5) a pregnancy or birth to the subject, (6) health of subject, (7) health of close relative or friend, and two broad-based categories, (8) involving work, housing or material deprivation and (9) a small number of miscellaneous crises such as burglaries. The scheme is crude and sometimes ambiguous in the sense that some events straddle more than one category—for example, a pregnancy rated severe because of the woman's poor housing and economic deprivation was rated here under 'pregnancy'—but it serves to give a general

TABLE I

Severe events[1] in terms of broad descriptive categories.

	Women without onset (101 women & 130 events) % events		*Women with onset (29 women & events) % events*			*Onsets in event category % onset*
Relationships						
1 Husband/lover	23	59	38	79	23	27 (11/41)
2 Close relative/friend	12		7			12 (2/17)
3 Child	18		17			17 (5/29)
4 Death	6		17			38 (5/13)
5 Pregnancy/birth		6		10		27 (3/11)
Health						
6 Subject	7	9	3	3		8 (1/13)
7 Other	2		0			
Other						
8 Job, housing & material deprivation	22	25	7	7		6 (2/35)
9 Miscellaneous	4		0			
	100		100			

1. All events other than 'linked' events are included, except for onset cases, where only the event preceding the onset is considered.

introduction to the range of incidents covered by severe events. All severe events occurring in the follow-up year have been taken for women without an onset of depression. However, if events were 'linked' to each other, only one was taken (e.g. a death of a father counted and not first learning of his cancer), or if there was more than one event in any one of the nine categories (e.g. a daughter found taking drugs and a son sent to court—both in category 2—'child'), this was again counted only once. Of the 130 severe events occurring to the 271 women without an onset, 59% involved interpersonal crises, 6% a pregnancy or birth, 9% health of the subject or a close relative, and 25% other events largely concerning work, housing or material deprivation (Table I).

For women with onset of depression, the severe event occurring nearest to onset was classified. Table I shows that the 29 depressed women with a severe event experienced more interpersonal crises than other women (79 *vs* 59%; $P<0.05$). Such interpersonal events, together with those involving a pregnancy or birth, were more likely to be associated with an onset of depression (23% [23/101] *vs* 8% [3/48]; $P<0.02$: Table I).

It has been clear from this kind of evidence that severe events are sometimes of a kind that are unlikely to provoke depression and this is why we developed the simple refinement of severe events into 'upper' and 'lower' categories. However, once we turn to characterising women in terms of events, there is a problem because they could experience more than one. Therefore, further *ad hoc* rules had to be developed to characterise women (rather than events). For instance, in dealing with the refinement of the severity rating, we characterised a *woman* as 'upper' if she had a severe event rated 'upper' at any time in the 6 months before onset of depression or in the 12 months before interview for other women. Those with a severe event in the upper division had a four-fold greater risk of depression (28% [27/98] *vs* 6% [2/32]; $P<0.01$).

However, this refinement of long-term threat rating does not take us very far, and we next turn to the rating of loss. There is widespread agreement that it is particularly the experience of loss that is involved in the onset of depression and a recent study using the LEDS to compare depression and anxiety conditions confirmed this (Finlay-Jones & Brown, 1981). In this study in a general practice setting in North London, depressive conditions tended to be preceded by severe events involving loss, and anxiety states by those involving danger. Similar results were found in a study conducted in Edinburgh, where severe life situations involving personal loss were highly predictive of depressive symptoms and those involving threat predictive of anxiety symptoms (Martin & Ingham, 1985). In the current study in Islington, we characterised a woman as having a loss if such an event occurred in the 6 months before onset or the year before interview for other women. Severe events involving loss defined in this way were more likely to be associated with depression (27% [24/88] of loss *vs* 12% [5/42] of non-loss women; $P<0.05$). Nevertheless, it should be noted that women in the non-loss group still had a higher risk of depression than those without a severe event (i.e. 12% [5/42] *vs* 2% [3/173]; $P<0.01$). When loss and the 'upper' *vs* 'lower' severity rating are both taken into account, there is a five-fold difference in rate of onset

TABLE II
Onset among 303 women in terms of presence of a provoking agent and some of its characteristics

	% onset	
No provoking agent	1 (2/153)	
Provoking agent:		
Major difficulty only	5 (1/20)	20 (30/150)
'Lower' severe event	6 (2/32)	
'Upper' severe event: non-loss	16 (4/25)	
'Upper' severe event: loss	32 (23/73)	
$P<0.001$	11 (32/303)	

among those with a severe event (Table II). 'Upper' events involving loss have a rate of 32%; 'lower' severe events with a risk of depression of 6% do not differ from major difficulties without severe events which have a risk of 5%. However, the latter two groups still had a four-fold greater risk of depression than those women with no provoking agent at all.

'Matching' of severe events with a difficulty present at the first interview

After the follow-up interview had been rated, a decision was made for every event, whatever its threat rating, about whether or not it was 'linked' to an ongoing difficulty and these were used later as the basis for the matching exercise. These 'link' ratings were made by a worker specially employed for the task and not involved in the original data collection and who was therefore blind to the information about symptoms and subjective responses. Difficulties are a source of events, since they tend to throw up events throughout their course. However, the criteria for deciding whether an event was linked with a prior difficulty did not require a *causal* relationship between the two, though this might well be present. For example, an event would be considered 'linked' if it had only been rated severe because of the presence of the difficulty. Thus a pregnancy rated severely threatening because of poor housing would be considered to be linked to the prior housing difficulty.

For the matching exercise itself, severe events and marked difficulties present at the first interview were considered. A period of 6 months and not 2 years was taken for the length of difficulty, as we did not wish to prejudge the relevant length of time, and on grounds of common sense this appeared to be long enough to differentiate a difficulty from events arising 'on their own'. In order to check whether the 'link' records were adequate to embody the notion of events 'matching' difficulties, we reconsidered the written accounts of the women with a severe event so that we could decide if any severe event not rated as 'linked' could be held to 'match' difficulties. At this stage, three further links were identified on the basis that the *meaning* of both the event and the difficulty for the subject and the implications for her life plans were critical for deciding on matching. In the majority of instances, difficulty and event were related in a straightforward sense (e.g. a husband leaving home in the background of a history of marital disputes over his heavy drinking and violence). Of the 130 women with a severe event, 35 had at least one matched to a prior marked difficulty (i.e. rated 1–3 on contextual severity and not involving health) that had lasted at least 6 months at the point of the first interview. (Under the LEDS, any health difficulty that creates major interference with any of the roles of the subject is considered to have non-health aspects and has been included in this exercise.)

Our experience suggests that because ratings of difficulties are 'harder' than those of commitment, or role conflict, D-events can be rated accurately enough in retrospect (Brown & Harris, 1982; Neilson, 1984) and thus do not demand a prospective design. In fact, for reasons of pressure on time, the ratings of difficulties were only made at the follow-up interview. However, in no case was a marked difficulty at the time of the first interview rated later without there being indications in the first interview material that it was already present at the earlier date. Thus there is unlikely to be any bias in the dating of these difficulties as a result of the respondent's subsequent experience of a linked severe event and/or depression in the follow-up year.

TABLE III
Onset among the 130 women with a severe event in terms of matching

	% onset
Severe event matching prior difficulty (D-event)	
Yes	46 (16/35)
No	14 (13/95)
	22 (29/130) $P<0.001$
Severe event matching prior commitment (C-event)	
Yes	40 (16/40)
No	14 (13/90)
	22 (29/130) $P<0.01$
Either C-event or D-event	
Yes	37 (24/65)
No	8 (5/65)
	22 (29/130) $P<0.001$

Women with such a *D-event* had a three-fold greater chance of developing depression (Table III). However, severe events occurring at the same time as a marked difficulty to which they did not match showed no increase whatsoever in the risk of depression. Of a total of 35 D-events, 37% concerned a husband or boy-friend, 26% a child, 31% finance or housing and 6% the subject's health. (The original computer-based exercise gives an almost identical result.)

Prospective analyses using earlier ratings

'Matching' of severe events with commitment at the first interview

A second form of matching concerned commitment at the time of first interview. All but 18% (54/303) of the 303 women followed-up had at least one marked commitment in one of the six areas with an average for the whole sample of 1.52. A total of 31% (40/130) of those with at least one severe event occurring in the follow-up year had one that *matched* an area in which they had earlier showed marked commitment and where it was reasonable to assume that the event would threaten any sense of worth, security or sense of hope the woman derived from the commitment or activity associated with it. We read all the written notes of the interview concerning both the commitment rating and the event before making a decision. However, in the majority of instances the decision could have been made by a simple comparison of the area of commitment and the 13-point shortened classification of the event and whether they logically belonged together (e.g. commitment to motherhood—child event). (In the case of someone named as very close, the event had to concern the person for whom the subject had been rated markedly dependent.) But there were some situations where the matching could not be done without taking account of more information. For example, loss of a job by a husband when the firm was also issuing redundancies to other workers was not considered to match high commitment to marriage, because we did not judge that in itself the event would threaten any sense of worth arising from the marriage, but we did include losses of jobs which in some way could be related to a husband's unreliability.

Women with a severe event that *matched* an area of marked commitment (a *C-event*) were almost three times more likely to develop depression (Table III, $P<0.01$). As a check on possible bias, since the decision concerning matching did at times involve an element of interpretation, we carried out a supplementary analysis based only on the simple matching of labels, employing a 38-point basic classification, a condensed version of which appears in Table I (Brown & Harris, 1978). In two instances, however, it was necessary to look up what or who was involved (one where the event was rated as 'goal disappointment' and one where it involved a 'key person from the past'): otherwise the exercise could have been carried out by computer with identical results. This much cruder matching exercise still resulted in a large difference in risk of depression (35% [14/40] *vs* 17% [15/90]; $P<0.02$) indicating the basic result was not biased in favour of our hypothesis by the interpretative nature of the matching exercise.

The two types of matching severe events dealt with so far, C-events and D-events, were uncorrelated (kappa = 0.02; NS). The overlap between them due to chance covered 31% of D-events and 17% of C-events. A woman with either type had a four-fold greater risk of depression than women with a non-matching severe event (Table III). Furthermore, as many as 80% (24/30) of those developing depression after a provoking agent had either a C-event or a D-event.

TABLE IV

Onset of depression among 150 women with a provoking agent in terms of the presence of a D-event (i.e. severe event matching a marked difficulty) and a major difficulty

	Type provoking agent (i.e. severe event or major difficulty)		
Major difficulty	*D-event % onset*	*Non-D-event % onset*	*Major difficulty without event % onset*
Yes	46 (13/28)	7 (1/14)	5 (1/20)
No	43 (3/7)	15 (12/81)[1]	—

1. Six women had a marked difficulty lasting 6–24 months and one had an onset.

One final point should be made about the role of difficulties. Most of the D-events were linked to difficulties that had gone on for at least 2 years rather than only 6 months (i.e. were major difficulties and therefore 'provoking agents') (Table IV). The presence of a major difficulty when an accompanying severe event did not match it did not raise the risk above that for women with a severe event alone—7% (1/14) and 15% (12/81) respectively (Table IV).

All four amendments to the basic severe event rating discussed so far make an independent contribution to onset (Table V)[1]. The risk is particularly high among those with both a C-event *and* D-event (73% [8/11]) and there is a 20-fold difference between them and those with the least risk (i.e. with a 'lower' severe event or only a major difficulty).

TABLE V

Onset among 303 women by characteristics of provoking agent

	C-event (event matching prior commitment)		
Type of provoking agent	*Yes % onset*	*No % onset*	*Total % onset*
1. D-event (severe event matching prior difficulty)	73 (8/11)	33 (8/24)	46 (16/35)
2. Both 'upper' *and* loss event (& not 1)	44 (7/16)	14 (5/35)	24 (12/51)
3. Remaining provoking agents	8 (1/13)	2 (1/51)	3 (2/64)
4. No provoking agents	—	—	1 (2/153)

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[1] This result and that following for Table VI are confirmed by logistic regression. Copies of all such analyses can be obtained from the authors.

Matching events with role conflict at the first interview

In an earlier analysis, we dealt with role conflict at the first interview and the raised rate of depression following a matching severe event in the subsequent year — an R-event (Brown & Bifulco, 1985). Such role conflict arising from diverging obligations, particularly between domestic and external spheres, was related to risk of depression. Among those with a provoking agent, one-third of those with role conflict developed depression compared with half this rate for those with no such conflict (Brown & Bifulco, 1985). Further analysis indicated that this increased risk was explained by the 'match' between the life event preceding onset and the conflict present at the first interview (43% [9/21] of women with a matching R-event and 18% [20/109] of those without developed depression; $P < 0.02$). For instance, a single parent whose conflict arose from the clash between the obligation to work and the obligation to be a good mother developed depression when she found out that her school-age daughter was pregnant, thus underlining her failure in one of her obligations. R-events and C-events were quite unrelated. However, R-events were highly related to D-events and, since only two instances of depression occurred for women with an R-event and without a D-event (and only one with a C- and a D-event), we have not included R-events in our current model. A total of 37% (13/35) of D-events and 8% (8/95) of non-D-events were R-events ($P < 0.001$); and 18% (7/40) of C-events and 16% (14/90) of non-C-events were R-events (NS).

Vulnerability: self-evaluation

As predicted on the basis of earlier research, it has also been established that a measure of low self-esteem made at first interview (negative evaluation of self [NES]) acts as a vulnerability factor in the sense that risk is increased only when there is a provoking agent (Brown & Bifulco, 1985; Brown *et al*, 1986*a*). However, Table VI makes clear that, except where both a C-event and a D-event are present, negative evaluation of self does increase risk for women in the categories defined in terms of event characteristics given in Table V (see footnote 1).

TABLE VI

Onset among 150 women with provoking agent by its characteristics and prior negative evaluation of self (NES)

	C-event (severe event matching prior commitment)			
	Yes		*No*	
	NES		*NES*	
Type of provoking agent	*Yes* *% onset*	*No* *% onset*	*Yes* *% onset*	*No* *% onset*
1. D-event (severe event matching prior difficulty)	63 (5/8)	100 (3/3)	45 (5/12)	25 (3/12)
2. Both 'upper' *and* loss event (& not 1)	67 (4/6)	30 (3/10)	27 (3/11)	8 (2/24)
3. Other provoking agent	0 (0/4)	11 (1/9)	8 (1/13)	0 (0/38)
	50 (9/18)	32 (7/22)	25 (9/36)	7 (5/74)

Anterior year results

Material was collected at the follow-up interview about events and difficulties in the 12 months *before* first interview. Therefore it was possible to repeat the analyses concerning characteristics of severe events for this anterior year. It was not possible to do this for findings concerning commitment or role conflict, since such ratings should be made at a time prior to the events concerned. However, for the cross-sectional measures, results were closely comparable. Of the 33 women with an onset in the anterior year, 25 had a severe event in the 6 months before, and for 95% (24/25) of these, at least one was 'upper' on severity; 72% (18/25) had one concerning a loss and 48% (12/25) one arising from a marked difficulty, a D-event. The same results already given for the follow-up year were 93, 83 and 55% respectively. A total of 72% of the events nearest to onset (18/25) involved an interpersonal crisis compared with 79% in the follow-up series.

Some examples

In order to illustrate the various ratings we have outlined so far, we will give three examples of women coping with a severe event. They have been selected to involve the same category of event — 'child' (see Table I). The examples illustrate the two extreme risk categories in Table VI and an intermediate one.

Marked risk (63% onset cases [5/8] — Table VI)

Mrs Steven had been widowed 3 years and had three children in their teens living at home. She had had a very difficult childhood, involving loss of parents and violence. When seen for the first time, she had many problems. For example, her electricity and gas had been cut off in the year as part of her chronic and major financial difficulties and her 16-year old son had been put on probation for violent and destructive behaviour and the resulting difficulty, which had lasted for 6 months by the time of our first interview, had been rated 'marked' with 'no or little chance of resolving'. She was rated as having negative evaluation of self. (She felt no good as a mother ["too soft"] and not much good as a housewife ["hopeless at budgeting"] and blamed herself over her son's delinquency). She had a few depressive symptoms and these were enough for her to be rated a borderline case of depression. She also had marked anxiety symptoms. She was rated as marked in her commitment to motherhood (e.g. "the first thing is the kids"; "I would have felt terrible if I couldn't have had the kids"; "I wouldn't have minded staying single if I could have had them"). At follow-up she had had an onset of caseness of depression just after appearing in court for not keeping up her contributions to paying for her son's £400 fine. The court told her, she said, that the penalty was 60 days in prison and this stood if she got behind again. It was a C-event given her commitment to motherhood and 'upper' in severity. Since her son had been a marked difficulty for a number of years with little or no chance of resolution, it was *also* a D-event. She had not expressed role conflict about her children and therefore it was not an R-event. The event itself was not considered a loss. Her son's behaviour had already previously led to her 'losing' important good ideas about him and there had not been a recent improvement that would justify a rating of loss in the sense of a 'disappointment'. However, it was rated '2—moderate' on 'danger' because of its threatening future implications. She was rated as having no hope: she reported she saw no way out as she simply did not have the money. Although she felt very angry at the court's behaviour, she still blamed herself and said she felt ashamed. The event appeared to reinforce prior feelings of failure.

Moderate risk (25% onset [3/12] — upper row, Table VI)

Mrs Rice was divorced with three children in their teens, but showed marked commitment to her work and to nothing else. She felt she had been a good mother and that her children were a credit to her. She was not rated as showing negative self-evaluation although she made some negative comments (e.g. "I'm too soft" and "not really efficient"). However, in the year before interview, her son of 17 had left home after a row which upset her greatly. This had been followed by a depressive episode at caseness level, but by the time of the first interview she showed only symptoms of anxiety. The difficulty at this time was rated 'marked' and 'unclear about chances of resolving'.

During the follow-up year her son was arrested for shoplifting. When she saw him for the first time in 6 months after this there was "shouting and swearing" and onset developed soon after the event. She was considered to have a D-event since the difficulty in her relationship with her son had lasted 6 months at first interview. The event was 'upper' on severity and a 'loss' since he had not been in trouble with the law before. Her initial reaction to her son leaving home had been to feel "bleak but not hopeless", but in response to his arrest in the follow-up year she was rated as having no hope ("there was nothing I could do to put things right"; "prison here he comes"). She did not feel "100% responsible", but it was "one more failure totted up". She had also expressed conflict concerning children at first interview—"it has been a battle between the children and studying". "I've been inclined to put them first, but they've been difficult to cope with." "In an ideal world I wouldn't have had any". Thus, this was also an R-event.

Low risk (0% onset cases [0/38] — Table VI)

Mrs Gordon was a 38-year old married woman with three teenage children at home and a full-time job. When seen at first interview, her self-evaluation was rated as positive ("I'm very confident." "I'm extrovert and cheerful." "I'm natural—nothing special—just me") with only a few minor negative comments ("I'm not a very decisive person"). She reported no role conflict, and the only commitment rated 'marked' was to her work ("I'm very involved with my work, I don't think I could ever give up work. I think it's important for a woman to have a job"). Her commitment to both marriage and motherhood were low. Her marriage was rather poor with low confiding and frequent arguments and now that her children were older, she felt they could look after themselves and required less of her attention.

In the follow-up year, Mrs Gordon's 18-year old son was involved in a fight while he was out for the day at the coast. The police were involved and he was charged with assault and ordered to pay £100 fine. However, Mrs Gordon heard nothing of this until a few weeks later, by which time her son had made arrangements to pay the fine out of his wages and this was being deducted by his employer every week. Mrs Gordon only heard about the affair when the employer rang her for certain details he required about her son.

This event was rated as 'lower' on threat largely because, by the time Mrs Gordon had heard about it, the penalty for the offence had already been determined and her son had clearly already coped well with the situation. She reported being quite optimistic about the future. However, it was rated high on loss since it was the son's first offence and therefore involved the loss of a 'cherished' idea in our terms—of a son as a responsible and reliable young adult. Because Mrs Gordon had no role conflict centred on her children, only low commitment and no prior difficulty, this did not constitute a matching event. Her psychiatric state remained 'normal' throughout.

Further analysis

The interpretation of the results concerning severity and loss appears to be straightforward and requires little further comment. In earlier research it has been only the small minority of events involving severe long-term threat that have raised the risk of depression, and the 'upper' and 'lower' threshold merely adds a refinement to the definition of a severe event. The result concerning 'loss' replicates that of the earlier study of depression and anxiety (Finlay-Jones & Brown, 1981) and also confirms a widespread belief in the critical importance of loss for the development of depression. We can only add at this point that half of the loss events (44/88) involved failure if this is used in the sense of a rejection, a major row or of involving an incident such as an examination failure or divorce.

The interpretation of C-events involving a prior marked commitment also appears to be straightforward. The idea underlying the contextual ratings of events is that the degree of threat reflects the likely impact on the respondent's plans and purposes and that threat will be greater to the degree that the person's sense of self is caught up in these. The importance of the biographical material underlying the rating is that it can be used to make an indirect assessment of such prior plans. A *direct* assessment of prior attachments and commitments is ruled out by the fact that reports of these some months later can never be guaranteed to be free from inaccuracies of memory. Particularly when other circumstances have changed in the intervening period, a respondent may distort the degree to which she was previously committed. Since the commitment ratings were made at the first interview before onset of the depressive condition, they are a *direct* and unbiased

TABLE VII

Marked commitment in at least one area at the first interview by presence of a provoking agent, negative evaluation of self (NES) and onset for those with a provoking agent

Marked commitment present	*Provoking agent (n=303)* %	*NES (n=303)* %	*Onset (n=150)* %
Yes	50 (126/250)	22 (56/250)	21 (27/126)
No	45 (24/53) NS	47 (25/53) $P<0.001$	13 (3/24) NS

assessment of relevant plans. The resulting increase in the strength of the link between the provoking event and onset can thus be seen as empirical support for the theoretical basis on which the rules of the contextual threat ratings were devised. We were surprised, however, that further analysis did not reveal any patterning in terms of number or type of commitment and other key variables. The only association to emerge was for those without any instance of marked commitment, 18% (53/303) of the sample, who had double the rate of negative evaluation of self (Table VII). However, such a lack of commitment did not translate into a higher risk of depression; indeed, there was a tendency for those with no marked commitment to have, if anything, a lower risk of depression (Table VII). In fact, this result sums up our conclusions about C-events. They appear to reflect the fact that marked commitment increases the saliency of a loss and hence risk of depression. The fact that a C-event, by definition, cannot occur among those with no such commitments, and that these latter women, if anything, have a lower risk of depression in spite of having much more negative evaluation of self, supports this straightforward interpretation.

The interpretation of D-events is more complex. Although, as already noted, we have not included R-events stemming from role conflict in our current model, the association between them and matching D-events does appear to contribute to the understanding of the mechanism underlying D-events. However, this contribution becomes clearer if response in terms of hopelessness to the D-event is considered at the same time.

Hopelessness

In our general theory of depression, we have speculated about the importance of generalised hopelessness in response to any crisis, and that the presence of a vulnerability factor such as lack of a close confiding relationship serves to increase the probability of such a response (Brown & Harris, 1978). For the 110 women questioned in detail about their response to a particular severe event, only 18% reported complete lack of hope. Since the measure was based on material collected after any onset of depression, it is open to various kinds of inaccuracy and bias. However, it is of note that, among the 35 women with a D-event, marked hopelessness was highly related to a quite independent judgement made by the interviewer about the possibility of the difficulty being resolved—64% (9/14) among those with a matching difficulty with low chances of resolution compared with 20% (4/20) of the rest ($P<0.01$) (one not known). Such hopelessness was also low among those with a severe event but without a D-event (9% [7/77]). A provisional argument can therefore be made that women with marked difficulties are more likely to respond with hopelessness once any severe event arises linked to the difficulty. Although tentative, this interpretation is made more plausible by the fact that the difference in hopelessness between those with and without a D-event also holds for those *not* developing caseness of depression (Table VIII). (This is confirmed by logistic regression.)

TABLE VIII

Marked hopelessness in response to a provoking agent in terms of whether the woman was an onset case and had a D-event

D-events	*Onset case*	*Not onset case*	*Total*
	% marked hopelessness		
Yes	56 (9/16)	27 (4/17)	42 (13/33)
No	23 (3/13)	6 (4/64)	9 (7/77)
Total	41 (12/29)	10 (8/81)	18(20/110)[1]

[1] Two D-events and 18 non-D-events not known for 'hopelessness'.

The presence of either marked hopelessness or an R-event goes a good way to explaining the much higher rate of onset among those with a D-event: those with either hopelessness (5/6), matching role conflict (5/9) or both (2/4) had a higher risk of onset than the remaining women with a D-event (63% [12/19] *vs* 25% [4/16]; $P<0.05$. However, those with a D-event without marked hopelessness or matching role conflict still had a relatively high rate of onset, and for a satisfactory explanation it is probably necessary to take account of vulnerability as represented by negative evaluation of self. As might be expected, since all D-events were by definition linked to a long-term marked difficulty in key areas of a woman's life, they were also associated with negative evaluation of self (57% [20/35] of those with a D-event *vs* 32% [30/95] of the rest of the women with a severe event; $P<0.05$. Those with a C-event but no D-event had a rate of 34% [10/29]). (In fact this association reflects an underlying association between the presence of an ongoing marked difficulty of 6 months or more at first interview and negative evaluation of self—47% [35/75] *vs* 20% [46/228]; $P<0.001$.) When negative evaluation is taken into account together with marked hopelessness and matching role conflict, the particularly high rate of onset among those with a D-event disappears (59% [15/26] onset among those with one or the other *vs* 11% [1/9] of the remaining women with a D-event; $P<0.02$).

Discussion

This analysis has indicated that considerably more can be done, even in a cross-sectional survey, to increase the level of association between a severe event and onset of depression. Even if C-events involving prior commitment and R-events involving prior role conflict are not considered because they concern material collected at the first interview, differentiation in terms of risk of depression is still considerable. If we take into account the refinement of the severity rating, whether the severe event involves a loss and whether it is linked to an ongoing marked difficulty, there is a 15-fold difference between the two extreme risk categories (i.e. using the last column of Table V). Once matching commitment and negative evaluation of self are taken into account, the added precision is impressive (i.e. Table VI). It does appear possible to define a pattern of experience where the majority will quickly develop clinical depression. If such a group is formed by taking the presence of a severe event matching a prior difficulty (D-event) *or* an 'upper' loss event in the context of both negative evaluation *and* matching commitment (C-event), 63% (20/32) of all onsets of depression fall into this high-risk group, and as many as 51% (20/39) of the risk group develop depression (whole of the top row and first column of the second row in Table VI). If a somewhat less strict definition of high risk is taken—a D-event, or a severe event with 'upper' loss with either negative evaluation of self *or* matching commitment—then these proportions are 81% (26/32) and 43% (26/60) respectively (whole of the top row and the first three columns of the second row in Table VI).

Of course, negative evaluation is a vulnerability factor and cannot be seen strictly as part of the event. However, this analysis begins to threaten any tidy conceptual distinction between provoking agent and vulnerability factor and we will consider this in a later publication. However, we believe nonetheless that they can and should be measured separately, and ideally, as in this enquiry, at different points in time. Severe events linked to an ongoing difficulty (D-events) are particularly important here. They are quite highly associated with negative evaluation of self, as a result of the underlying association between marked long-term difficulties and negative evaluation of self. It seems reasonable to assume for the present that much of this association reflects the influence of the ongoing difficulty on self-esteem and we have presented evidence for this in another paper (Brown *et al*, 1986*b*). (In Table IV it was seen that the great majority of these difficulties had lasted over 2 years: the average length was 4.1 years.) If this is accepted, then a long-term difficulty will serve to reduce self-esteem and in time usher in the life event leading to onset. However, we have also seen that the association of D-events with negative evaluation of self provides only part of the explanation of their particularly high link with depression, and it is probably necessary to take into account other cognitive processes. Here we discussed the overlap of D-events with matching role conflict events (R-events) and also the tendency of women to respond to D-events with marked hopelessness. In his extraordinary story, *Albert Nobbs*, George Moore tells of the experiences of a woman who spent her life disguised as a waiter in a Dublin hotel. A crisis occurs in which she tells her story of deprivation and hardship to someone who discovers her secret: "I thought nobody would ever hear it, and I thought I should never cry again . . . It is much sadder than I thought it was, and if I had known how sad it was I shouldn't have been able to live through it". It is a common experience that great deprivation, hardship and disappointment can be shifted more or less permanently to some peripheral part of consciousness; and one role of life events can be to tell us something central about our lives that we have known in one sense all along. The role of matching difficulties and commitments indicates the importance of the loss of something upon which one has heavily staked a part of oneself and, furthermore, that one can lose again something essentially already lost.

The present study is an exploratory one and needs replication. However, all the main findings were the result of hypotheses formulated before the study began and all those involving measures made at follow-up were confirmed by an additional analysis of the anterior year data. As a whole, the findings of course underline the importance of social environment and cognitive factors in the aetiology of depression. They also offer some support for the conclusion of the earlier research in Camberwell that it is the meaning of *particular* events that tends to be crucial for depression and that, in so far as generalised hopelessness plays a role in the aetiology, it often stems from a highly particular set of circumstances.

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References

Andrews, G. & Tennant, C. (1978) Being upset and becoming ill: an appraisal of the relation between life events and physical illness. *Medical Journal of Australia*, **1**, 324–327.

Bebbington, P., Sturt, E., Tennant, C. & Hurry, J. (1984) Misfortune and resilience: a replication of the work of Brown & Harris. *Psychological Medicine*, **14**, 347–363.

Brown, G. W. (1981) Life events, psychiatric disorder and physical illness. *Journal of Psychosomatic Research*, **25**, 461–473.

—— (1983) Accounts, meaning and causality. In *Accounts and Action* (eds G. N. Gilbert & P. Abell. London: Gower.

—— (1986) Depression. In *Life Events and Illness* (eds G. W. Brown and T. O. Harris). New York: Guildford Press (in press).

—— & Harris, T. O. (1978) *Social Origins of Depression: A Study of Psychiatric Disorder in Women*. London: Tavistock Publications; New York: Free Press.

—— & Prudo, R. (1981) Psychiatric disorder in a rural and an urban population. 1. Aetiology of depression. *Psychological Medicine*, **11**, 581–599.

—— & Harris, T. O. (1982) Fall-off in the reporting of life events. *Social Psychiatry*, **17**, 23–28.

—— & Bifulco, A. (1985) Social support, life events and depression. In *Social Support: Theory, Research and Applications* (ed. I. Sarason). Dordrecht: Martinus Nijhoff.

—— & Harris, T. O. (1986) Establishing causal links: the Bedford College studies of depression. In *Life Events and Psychiatric Disorders* (ed. H. Katschinig). London: Cambridge University Press.

——, Craig, T. K. J. & Harris, T. O. (1985) Depression: disease or distress? Some epidemiological considerations. *British Journal of Psychiatry*, **147**, 612–622.

——, Andrews, B., Harris, T., Adler, Z. & Bridge, L. (1986*a*) Social support, self-esteem and depression. *Psychological Medicine* (in press).

——, Bifulco, A., Harris, T. O. & Bridge, L. (1986*b*) Life stress, chronic subclinical symptoms and vulnerability to clinical depression. *Journal of Affective Disorders*, **11**, 1–19.

Campbell, E., Cope, S. & Teasdale, J. (1983) Social factors and affective disorder: an investigation of Brown and Harris's model. *British Journal of Psychiatry*, **143**, 548–553.

Cole, P. & MacMahon, B. (1971) Attributable risk percent in case-control studies. *British Journal of Preventive and Social Medicine*, **25**, 242–244.

Cooper, B. & Sylph, J. (1973) Life events and the onset of neurotic illness: an investigation in general practice. *Psychological Medicine*, **3**, 421–435.

Cooper, J. E., Copeland, J. R. M., Brown, G. W., Harris, T. O. & Gourley, A. J. (1977) Further studies on interviewer training and inter-rater reliability of the present state examination (P.S.E.). *Psychological Medicine*, **7**, 517–523.

Costello, C. G. (1982) Social factors associated with depression: a retrospective community study. *Psychological Medicine*, **12**, 329–339.

Dean, C., Surtees, P. G. & Sashidharan, S. P. (1983) Comparison of research diagnostic systems in an Edinburgh community sample. *British Journal of Psychiatry*, **142**, 247–256.

Finlay-Jones, R. A. & Brown, G. W. (1981) Types of stressful life event and the onset of anxiety and depressive disorders. *Psychological Medicine*, **11**, 803–815.

——, ——, Duncan-Jones, P., Harris, T. O., Murphy, E. & Prudo, R. (1980) Depression and anxiety in the community: replicating the diagnosis of a case. *Psychological Medicine*, **10**, 445–454.

Goldthorpe, J. H. & Hope, K. (1974) *The Social Grading of Occupations: A New Approach and Scale*. London: Oxford University Press.

Markush, R. E. (1977) Levin's attributable risk statistic for analytic studies and vital statistics. *American Journal of Epidemiology*, **105**, 401–407.

Martin, P. Mc. & Ingham, J. G. (1985) Dimensions of experience and symptomatology. *Journal of Psychosomatic Research*, **29**, 475–488.

Moss, P. & Plewis, I. (1977) Mental distress in mothers of pre-school children in Inner London. *Psychological Medicine*, **7**, 641–652.

Neilson, E. (1984) Psycho-social factors in physical disorders: A preliminary report to the ESRC.

O'Connor, O. & Brown, G. W. (1984) Supportive relationships: fact or fancy? *Journal of Social and Personal Relationships*, **1**, 159–195.

Parry, G. & Shapiro, D. A. (1986) Social support and life events in working-class women: stress buffering or independent effects? *Archives of General Psychiatry*, **148**, 373–385.

——, —— & Davies, L. (1981) Reliability of life-event ratings: an independent replication. *British Journal of Clinical Psychology*, **20**, 133–134.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research diagnostic criteria; rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.

Surtees, P. G., Dean, C., Ingham, J. G., Kreitman, N. B., Miller, P. McC. & Sashidharan, S. P. (1983) Psychiatric disorder in women in an Edinburgh community: associations with demographic factors. *British Journal of Psychiatry*, **142**, 238–246.

Tennant, C., Smith, A., Bebbington, P. & Hurry, J. (1979) The contextual threat of life events: the concept and its reliability. *Psychological Medicine*, **9**, 525–528.

Wing, J. K. & Sturt, E. (1978) The PSE-ID-CATEGO System — a supplementary manual. London: Institute of Psychiatry (mimeo).

——, Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

——, Nixon, J. M., Mann, S. A. & Leff, J. P. (1977) Reliability of the PSE (ninth edition) used in a population study. *Psychological Medicine*, **7**, 505–516.

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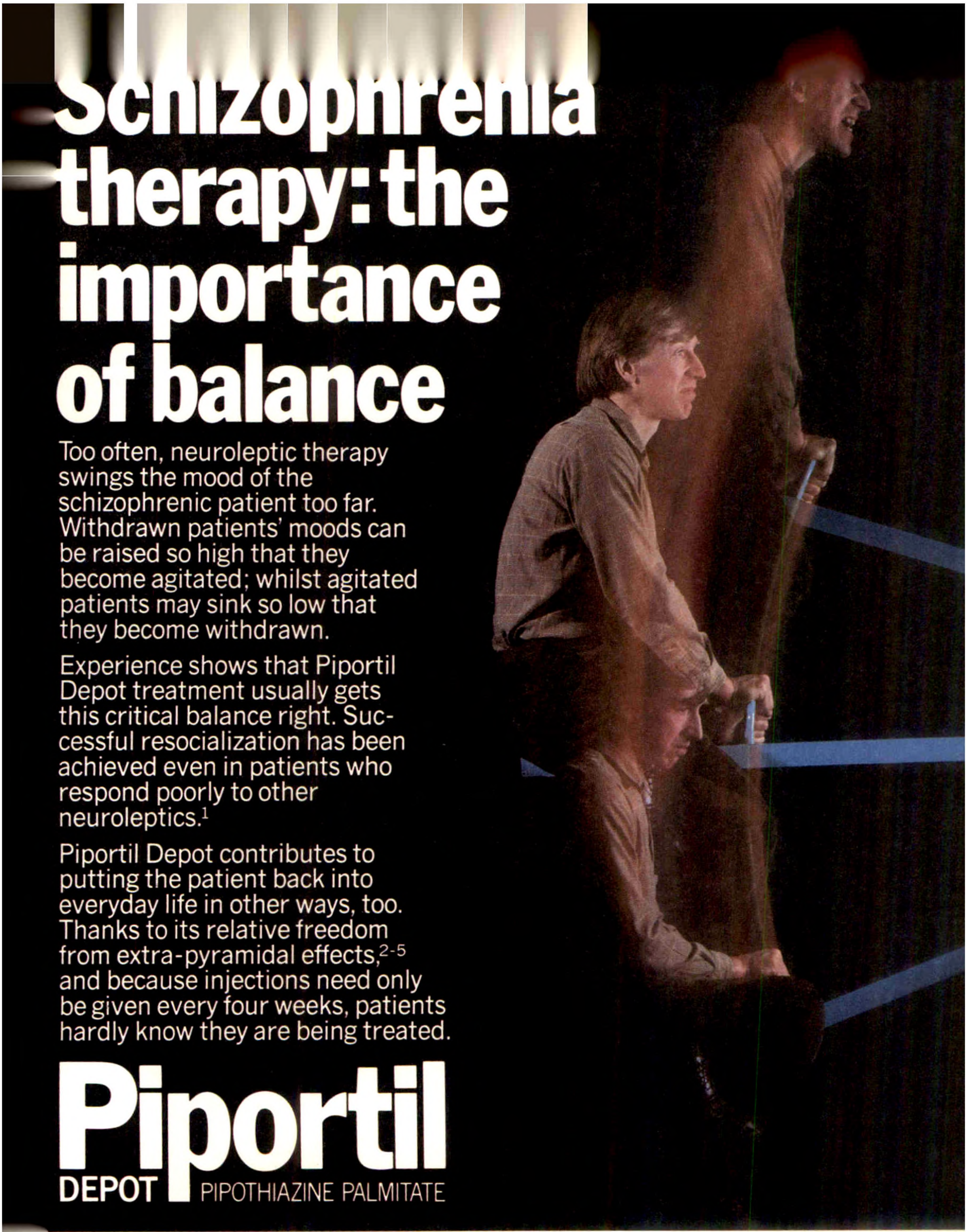

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A SCHIZOPHRENIC.

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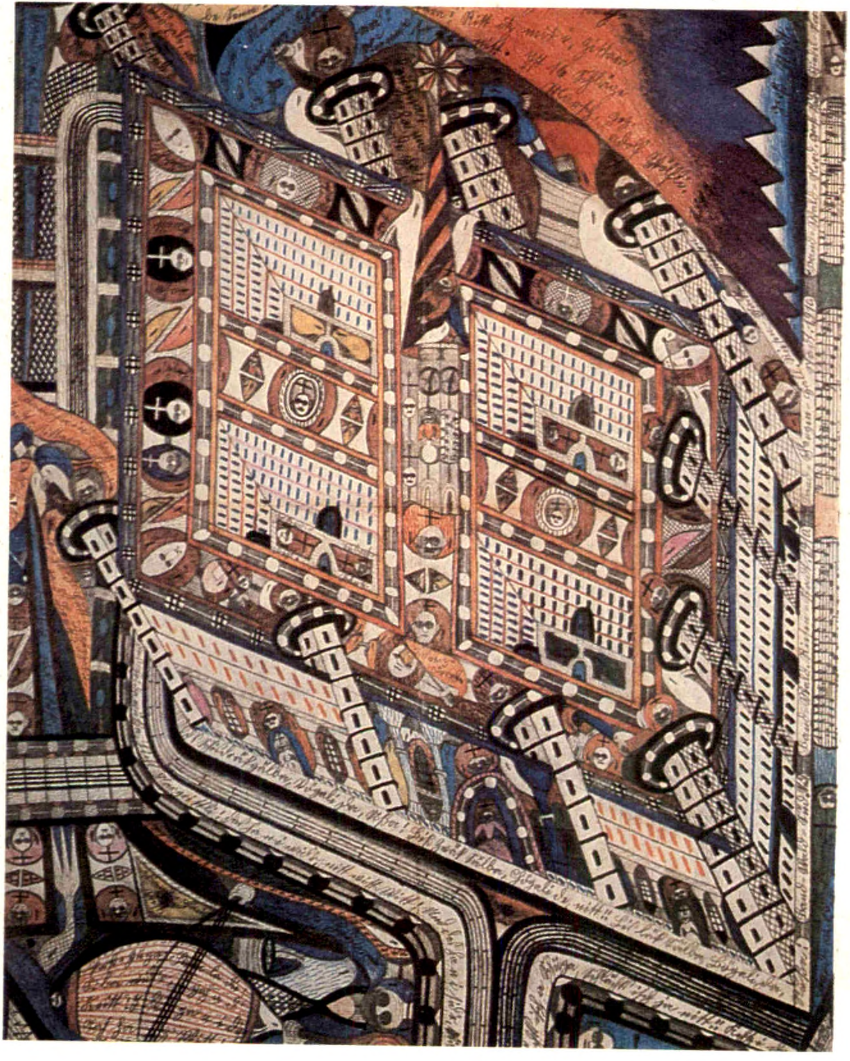
The picture below is one of a number that, we believe, give an insight into the schizophrenic mind and is reproduced with the kind permission of the Museum of Fine Art, Berne.



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British Journal of Psychiatry (1986), **150**, 43–48

Prediction of Outcome in Depressive Illness by the Newcastle Diagnosis Scale

Its Relationship with the Unipolar/Bipolar and DSM-III Systems

M. W. P. CARNEY, E. H. REYNOLDS and B. F. SHEFFIELD

The Newcastle scores of a group of 64 and subsample of 52 severely depressed inpatients were not normally distributed. Evidence for discontinuity in these distributions was adduced from the contrast in outcome between the endogenous and neurotic patients thus defined, the endogenous consistently doing better than the neurotic group. The unipolar/bipolar system failed to predict different results for endogenous and neurotic patients unless unipolar was subdivided into endogenous and neurotic subgroups. The DSM-III criteria for major depression and melancholia failed to identify subgroups of differing prognoses.

The controversy between the exponents of the monist and dualist theories of the classification of depressive disorders was reignited by the studies of Kiloh & Garside (1963), Carney *et al* (1965) and Sandifer *et al* (1966) favouring the two-type thesis of depression. Their findings, however, were contested by Kendell (1968) and Post (1972). The argument continued, mainly in the correspondence columns of the *British Journal of Psychiatry* (e.g. Garside, 1973), until the mid-1970s when interest in it faded. The rating scale devised by Carney *et al* (1965)—the Newcastle Index—continues to be used, however, in making the distinction between endogenous and neurotic depressions (e.g. Checkley, 1978; Bech *et al*, 1980; Coppen *et al*, 1983; Carney, 1986). Much was made of the signficance of finding a unimodal or bimodal distribution of Newcastle scores for the monists' and dualists' cases respectively: Garside & Roth (1978) pointed out that a unimodal distribution does not exclude the possibility of at least two depressive populations and Kendell (1982) has recently emphasised that evidence of discontinuity in some independent correlate such as clinical outcome or the distribution of a biological marker like the dexamethasone suppression test (DST) would provide such evidence, even in the presence of a unimodal distribution of scores.

The findings of fresh studies, using the Newcastle Index (Davison *et al*, 1984; Zimmerman *et al* 1986a: Zimmerman *et al*, 1986b ('Iowa Group')) have revived interest in the issue. Though in each study the Newcastle distribution did not depart significantly from normal, and therefore failed to replicate the original Newcastle distributions (Carney *et al*, 1965; Carney & Sheffield, 1972), other evidence suggested that these two kinds of depression were not accounted for by differences in severity of illness alone.

The Iowa group applied the Newcastle Index to 159 depressed inpatients and found that endogenous depressives so-defined had a significantly higher rate of the DST non-suppression than Newcastle Index neurotic depressives (Zimmerman *et al*, 1986a). In 152 of these patients they showed that the morbid risk for alcoholism in endogenous depression was lower than that for neurotic depression (Zimmerman *et al*, 1986b). In both studies the relationship between the Newcastle scores and these variables was non-linear. Davidson *et al* (1984) studied the Index in 36 depressed patients: endogenous and non-endogenous patients thus defined responded differently to isocarboxazid.

This paper describes relationships between Newcastle scores, Hamilton scores and other measures of outcome found incidentally in the course of a series of trials of S-adenosyl methionine (SAM), a normal brain metabolite with antidepressant properties (Carney *et al*, 1983; Reynolds & Stramentinoli, 1983; Reynolds *et al*, 1984). This paper deals with patients admitted to a pilot trial and three admitted to a controlled comparison with amitriptyline (all treated with SAM) together with the first 46 entering the placebo-controlled trial of SAM.

TABLE I
Demographic data from the first 58 patients completing the SAM trials

		Age (years)	*Sex*		*Treatment*	*Group*
	n	*Mean ± s.d.*	*M*	*F*	*SAM*	*Placebo*
Endogenous	31	55.3 ± 18.6	14	17	18	13
Neurotic	27	53.6 ± 12.6	7	20	16	11

Method

The Newcastle scales were applied to depressed patients admitted successively on clinical grounds to Northwick Park and Kings College Hospitals. These patients were judged to be primary depressives, free of non-depressive disorders (epilepsy, drug and alcohol abuse, schizophrenia and organic brain states) who had scored at least 20 on the Hamilton Rating Scale (HRS). They all gave informed consent and underwent a 7-day drug-free 'wash-out' period before being admitted to one of the above trials. S-Adenosyl methionine or placebo were given daily intravenously to a total of 14 injections, each under identical conditions. Patients were assessed on admission to the trial on several rating scales, including the HRS; and on the 7th and 14th days on the HRS. A total of 64 patients (including 52 Northwick Park patients) were assessed for these trials. An arbitrary decision for logistical reasons was made to study further the first 46 patients to enter the double-blind trial, together with the 15 patients entering the other trials—61 in all. Of the 61 patients, 58 completed the 2-week trial period and the Hamilton data are complete and available in 52 of these. Patients scoring more than 5 on the Newcastle scale were classified as 'endogenous'; and 5 or less 'neurotic'. There were no significant differences between these two categories on age or sex distribution or the allocation of SAM or placebo (Table I). Neither did the pilot and double-blind trial patients differ on age and sex distributions.

Results

The distributions of the Newcastle scores of all 64 patients and the 52 Northwick Park patients assessed for the projects are shown in Figs 1 and 2 respectively (there were too few King's patients to be dealt with separately in this analysis). While the distribution for the whole group departs significantly from normality ($\chi^2 = 18.47$, d.f. $= 6$, $P < 0.01$), that of the Northwick Park subsample is more clearly bimodal ($\chi^2 = 28.7$; d.f. $= 6$: $P < 0.001$). Though these results, to a large extent, echo the original Newcastle findings, rigorous conclusions should not be based on them for reasons given below. However, when the mean falls in HRS for various values of Newcastle scores are compared (Table II) the series is clearly non-linear with an abrupt increase from 3–5 to 6–8, indicating discontinuity in the data. Moreover, though there are no significant differences between the initial mean HRS scores for 'endogenous' and 'neurotic' patients in both the total sample and the Northwick Park subsample, when the mean falls in HRS over the trial period in both groups are compared, there are significant differences, the 'endogenous' patients achieving the greater fall in each case (Table III). For the total sample this was 9,97 (endogenous) and 3.06 (neurotic) and for the Northwick Park patients, 10.59 and 2.13 respectively.

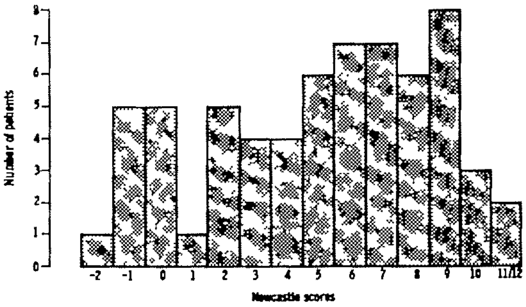

FIG. 1. Newcastle scores of 64 NPH and Kings patients

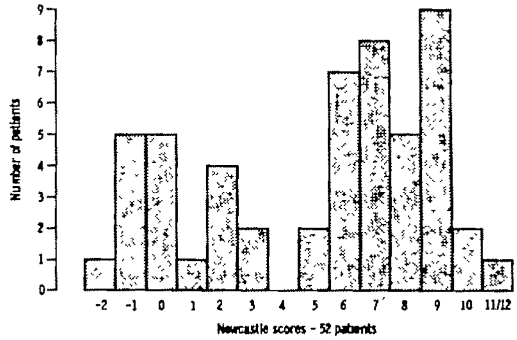

FIG. 2. Distribution of Newcastle scores: Northwick Park

We also examined the possibility that individual clinical features rather than the diagnosis endogenous depression was responsible for the difference in outcome between the depressive types. To do this we examined its relationship to the following subscales on the HRS; anxiety (psychic and somatic); insomnia (early, middle and terminal); delusions: weight loss (0,1 and 2); and agitation and retardation. None of the Hamilton subscales showed any significant association with a diagnosis of endogenous-neurotic depression as judged by Newcastle score and therefore could not have accounted for the difference between these groups.

TABLE II
Mean fall in HRS for various values of Newcastle Score: 58 patients

	'Neurotic'			*'Endogenous'*	
Newcastle score	<0	0–2	3–5	6–8	>8
No. of patients	6	9	12	20	11
Mean fall in HRS	1.2	2.9	2.9	9.7	5.6

TABLE III

Mean initial and mean falls in HRS scores for all 58 and 46 Northwick Park subsample patients (27 'endogenous' and 19 'neurotic') followed up for 2 weeks

			n	*Endogenous* (±*s.d.*)	*Neurotic* (±*s.d.*)	*t*	*P*
All patients	Mean initial	HRS	61	26.87±5.18	24.86±3.60	1.65	>0.05
	Mean fall in	HRS	58	9.97±8.72	3.06±6.15	3.32	<0.01
NPH patients	Mean initial	HRS	46	26.79±5.25	23.87±11.84	1.96	>0.05
	Mean fall in	HRS	46	10.59±8.67	2.13±5.32	3.63	<0.01

Very marked improvement

When those patients who had achieved 66% reduction in Hamilton's score are compared with those who had not achieved this reduction, eight of the 'endogenous' and no 'neurotics' were found to have achieved this standard of improvement. The difference is significant ($\chi^2 = 8.34$; d.f. = 1: $P<0.01$). Thus, 25% of those diagnosed 'endogenous' by the Newcastle score achieved this superior grade of improvement, whereas none of those categorised as 'neurotic' by the Newcastle did so.

Bipolar/unipolar classification

Northwick Park patients with whom there was historical evidence of one or more manic episodes of at least 2 weeks' duration each (these data were not available for the Kings College patients) were classified as 'bipolar' and those case histories which lacked such evidence, unipolar. The 46 patients for whom sufficient data were available to make this distinction comprised 12 bipolar and 34 unipolar patients, 17 of them with 'endogenous' Newcastle scores and 17 with 'neurotic' scores. The mean initial HRS scores of these groups (Table IV) did not differ—neither did the age, sex or SAM/placebo distributions—but the mean falls in HRS of both the bipolar and endogenous unipolar patients were significantly greater than that of the neurotic unipolar patients. Bipolar and endogenous unipolar groups, however, did not differ in this respect.

DSM-III classification

The DSM-III classification (DSM-III, 1980) specifies criteria for major depression of which bipolar psychosis and melancholia are subgroups. None of these definitions was entirely satisfactory for our design, as unlike ratings on the HRS and Newcastle scales, they are not quantifiable for the purposes of comparison or prognostic prediction. When we looked at the case histories of the above 46 Northwick Park Hospital depressives it was evident that they all satisfied the criteria for major depression. These comprise dysphoric mood or loss of pleasure continuously present for at least 2 weeks prior to assessment in the presence of four or more of the following: changes in appetite and weight; sleep disturbance; psychomotor retardation or agitation; loss of pleasure in usual activities including sexual interest; anergia; self-criticism or guilt; diminished ability to think or concentrate; ideas of suicide and death. By these criteria, all these patients were major depressives so that the diagnosis of major depression within the context of this subgroup did not further identify groups of contrasting prognoses.

We then looked at our patients in the light of DSM-III criteria for depression with melancholia. The discriminating criteria, in addition to other major depressive criteria, were at least three of the following: distinct quality of depressed mood; morning aggravation; early morning waking; psychomotor retardation and agitation; significant anorexia or weight loss: excessive or inappropriate guilt. There were 25 melancholic and 21 non-melancholic patients (Table V). They did not differ on mean initial HRS but both groups showed significant falls in HRS over the 14 days, the extent of these falls however not differing significantly ($t = 0.9$).

TABLE IV

Unipolar and bipolar patients: mean initial and mean fall in the HRS scores

		HRS score ± *s.d.*	
Type of patient	*n*	*Mean initial*	*Mean fall over trial period*
Bipolar	11	24.6±3.65	10.5±9.96
All unipolar	34	24.9±5.7	5.7±7.43
Unipolar endogenous	17	26.9±5.77	8.1±7.36
Unipolar neurotic	17	23.4±4.43	2.6±5.02

TABLE V
Mean initial and 14-day Hamilton's scores for melancholic and non-melancholic (DSM-III) patients completing 14 days' trial

	Number	*Mean HRS ± Standard deviation*		*Significance of fall*	
		Initial	*at 14 days*	*t*	*p*
Melancholia	25	26.9±4.35	19.6±11.08	3.00	<0.01
Non-melancholia	21	23.3±3.75	18.6±7.9	2.4	<0.05

Discussion

The investigation arose only incidentally out of a larger project—a series of trials of the anti-depressant efficacy of SAM. These were all severe, drug-free, primary depressives admitted for normal clinical reasons. It must be emphasised that the Newcastle scale was only one of the several administered on assessment for the trials before the results of treatment could have been predicted. Though a study of the distribution of Newcastle scores in depressed patients and its relationship to treatment outcome was not among the original purposes of the investigations, it early became apparent that the endogenous depressives were faring better than the non-endogenous patients so it was decided to investigate this aspect. For reasons quite beyond the original trial design, an arbitrary endpoint, short of the termination of the controlled trial, was selected and the data analysed after the completion of the controlled trial. This trial is now complete but all the data for all the trial patients have yet to be analysed. Nevertheless, because a study of the endogenous/neurotic dichotomy was not one of the original purposes of the investigation, the validity of the present result is thereby enhanced.

The endogenous and neurotic patients (as judged by Newcastle score) did not differ appreciably on our principal objective criterion of severity of depression—Hamilton's score. However, they did differ significantly on an independent variable, clinical outcome, patients with a Newcastle score of more than 5 being significantly better than those with a lower score. This finding was not accounted for by age, sex or treatment variations. The difference is so marked that the investigators have decided that a future trial should be confined to endogenous depression.

The evidence of the distribution curves, though not supporting the unitary view, does not constitute conclusive evidence for the binary thesis. The shapes of these curves depend upon several factors: the attitudes of the assessors, the populations being sampled and the method employed. Variations in these factors may have contributed to the different shapes of the curves from the Northwick Park subsample and the total sample, the former being clearly the more bimodal, and the latter doubtless being influenced by the characteristics of the King's patients. However, there was more than one assessor so that individual variations would have tended to cancel each other out: and the Northwick Park and King's catchment area populations differed in social class, structure and ethnic origin. Moreover, we were probably not looking at random samples of the general population, sources of potential bias being the mode of referral to hospital and the criteria of selection for the trials. Nevertheless, the difference in outcome between the endogenous and neurotic patients in both samples was equally marked and for reasons already given the evidence of distribution curves should not be awarded decisive importance in the context of this argument. It should not seriously detract from the case for the binary view, particularly in the light of the significant associations between diagnosis and outcome. Moreover, the Newcastle scale was only one of several rating scales applied on admission to the trial after a drug-free week, before injections were started or the outcome was predictable and the Registrars who carried out these investigations were not involved in the monist-dualist controversy. It was unlikely, therefore, that these ratings were influenced by preconceived nosological ideas on their part. Thus, the shape of these curves does not support a unitary explanation but to a great extent they confirm the original Newcastle findings (Carney *et al*, 1965). Firm evidence of a meaningful discontinuity (Kendell, 1982) in this patient population was provided by the abrupt break in mean reductions in HRS scores on either side of Newcastle score 5, the patients on the 'endogenous' (>5) side of this break faring significantly better than those on the non-endogenous side. Thus, the Newcastle scores constituted a non-linear series linked with an independent criterion, clinical outcome.

The whole question of prediction of outcome is of considerable interest. In the original paper (Carney *et al*, 1965) it was shown that outcome could be more accurately predicted by a set of features specifically related to ECT response than by diagnosis, and an ECT prediction index was based on these. The question arises—could not this device have been applied in the present work? It was not included in the trial design for these studies because at that time there seemed no good reason for doing so. Moreover, the wisdom of attempting to base conclusions about diagnosis and classification on the response to a particular treatment is basically unsound (Carney *et al*, 1965). Neither can it be automatically assumed that weighted features predictive of response to ECT will be the same for response to SAM. Finally, it was practically not possible to introduce the ECT Prediction Index at a late stage in the present work because it comprises a different set of 10 features with different weights from those of the diagnosis index. In respect of only two features—'anxiety' and 'weight loss'—do these inventories overlap. Both the other schemes of classification we examined depend on different sources of variation, the unipolar-bipolar on polarity, and DSM-III, a combination of severity of depression ('major depression') and criteria for distinguishing endogenous from non-endogenous depression (melancholia and non-melancholia). The unipolar–bipolar data indicate that there was only a modest difference between bipolar and all unipolar patients. However, when the HRS results for the bipolar and neurotic unipolar patients were compared there was a radical difference as there was between endogenous unipolar and neurotic unipolar patients.

However, when bipolar and endogenous unipolar patients were compared, there was no significant difference. Roth & Barnes (1981) have already stressed that in assessing the results of treatment in depression it is important to distinguish between endogenous and neurotic varieties of unipolar patient. Failure to make this distinction may result in valuable differences being submerged and possibly false conclusions being drawn.

The DSM system of classification is enjoying widespread popularity in the United States, perhaps not so enthusiastically shared by European psychiatrists. By DSM-III criteria all our patients would have been classified as major depressives. Subdividing into melancholics and non-melancholics however was not associated with differences in outcome so that the use of this system in this material would not have better predicted the outcome. Meaningful results from many drug trials will not be forthcoming until the endogenous–neurotic source of variation is taken into account (Montgomery, 1981). This is not to say that varieties of depressive disorder other than these two may not eventually be established, but the endogenous subgroup, in particular, is particularly stable and shows up consistently in other studies using various methods of analysis, especially cluster analysis (Paykel, 1971, 1981).

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References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) DSM-III. Washington, DC: Division of Public Affairs, APA.

Bech, P., Gram, L. F., Reisby, N. & Rafaelsen, O. J. (1980) The WHO depression scale: relationship to Newcastle Scales. *Acta Psychiatrica Scandinavica* **62**, 140–155.

Carney, M. W. P. (1986) The Newcastle Scale. In *Assessment of Depression* (ed. N. Sartorius & T. Ban), Berlin: Springer Verlag, on behalf of WHO).

—— & Sheffield, B. F. (1972) Depression and Newcastle Scale. Their relationship to Hamilton's scale. *British Journal of Psychiatry*, **121**, 35–40.

——, Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of E.C.T. response. *British Journal of Psychiatry*, **3**, 659–674.

——, Martin, R., Bottiglieri, T., Toone, B. K., Nissenbaum, H., Reynolds, E. H. & Sheffield, B. F. (1983) The switch mechanism in affective illness and SAM. *Lancet*, **1**, 820–821.

Checkley, S. A. (1978) A new distinction between the euphoric and antidepressant effects of methylamphetamine. *British Journal of Psychiatry*, **133**, 416–423.

Coppen, A., Abou-Saleh, P., Miller, P., Metcalf, M., Harwood, J. & Bailey, J. (1983) Dexamethasone Suppression Test in depression and other psychiatric illness. *British Journal of Psychiatry*, **142**, 498–504.

Davidson, J., Strickland, R., Turnbull, C., Belyea, M. & Miller, R. D. (1984) The Newcastle Endogenous Depression Diagnostic Index: validity and reliability. *Acta Psychiatrica Scandinvica*, **69**, 220–230.

Garside, R. F. (1973) Depressive illness in late life. *British Journal of Psychiatry*, **122**, 118–119.

—— & Roth, M. (1978) Multivariate statistical methods and problems of classification in psychiatry, *British Journal of Psychiatry*, **133**, 53–67.

Kendell, R. E. (1968) The problem of classification. In *Recent Developments in Affective Disorders* (eds A. Coppen & A. Walk), British Journal of Psychiatry Special Publication No. 1. London.

—— (1982) The choice of diagnostic criteria for biological research. *Archives of General Psychiatry*, **39**, 1334–1339.

Kiloh, L. G. & Garside, R. F. (1963) The independence of neurotic depression and endogenous depression. *British Journal of Psychiatry*, **109**, 451–463.

Montgomery, S. A. (1981) Measurement of serum drug levels in the assessment of antidepressants. In *Methods in Clinical Pharmacology—Central Nervous System* (ed. M. Lader & A. Richens), London: MacMillan.

Paykel, E. S. (1971) Classification of depressed patients: a cluster analysis derived grouping, *British Journal of Psychiatry*, **118**, 275–288.

—— (1981) Have multivariate statistics contributed to classification?, *British Journal of Psychiatry*, **139**, 357–362.

Post, F. (1972) The management and nature of depressive illness in later life: a follow through study. *British Journal of Psychiatry*, **121**, 393–404.

Reynolds, E. H. & Stramentinoli, G. (1984) S-adenosyl methionine and affective disorder. *Psychological Medicine*, 13, 705–710.

——, Carney, M. W. P. & Toone, B. K. (1984). Methylation and mood, *Lancet*, ii, 196–198.

Roth, M. & Barnes, T. R. E. (1981) The classification of affective disorders: the synthesis of old and new concepts. *Comprehensive Psychiatry*, 22, 54–77.

Sandifer, M., Wilson, T. C. & Green, L. (1966) The two-type thesis of depressive disorders, *American Journal of Psychiatry*, 123, 93–97.

Zimmerman, M., Pfhol, B., Stangl, D. & Coryell, W. (1986a) An American validation study of the Newcastle Diagnostic Scale. 1. Relationship with the Dexamethasone Suppression Test, *British Journal of Psychiatry*. (in press).

——, Coryell, W., Pfhol, B. & Stangl, D. (1986b). The validity of our definitions of endogenous depression: II clinical, demographic, familial and psychosocial correlates, *British Journal of Psychiatry*. (in press).

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Factor Study of a Psychiatric Child Rating Scale
Based on Ratings Made by Clinicians on Child and Adolescent Clinic Attenders

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Symptom rating sheets completed by psychiatrists over a 9-year period on 2602 intellectually normal child and adolescent clinic attenders were subjected to factor analysis (principal components). Two types of factor sets were derived, one for younger subjects (3–11 years) and another for older subjects (12–18). Both had three factors in common: conduct disturbance, relationship problems and emotionality, but differed on the fourth – developmental immaturity for the younger group, depression for the older. A factor solution was also derived for the whole clinic sample with three scales: conduct, emotionality and immaturity. Both types of factor solutions were subjected to MANOVA for age and sex effects, both of which proved to be of significant influence. Good correspondence was found between the symptom factors identified and conceptually similar clinical diagnoses.

Patterns of association between psychological symptoms have been investigated widely and have provided a basis for many classification schemes, diagnostic instruments and clinical rating scales for the assessment of psychopathological disturbance in children and adolescents. Such work has primarily involved the multivariate analysis of data and recent reviews (Achenbach, 1978; Quay, 1979) have suggested that certain dimensions appear consistently and are characterised by aggressive, delinquent, hyperactive, schizoid, anxious, depressed and withdrawn symptom domains (Achenbach, 1978).

These findings are particularly interesting given the heterogeneous nature of the studies involved, using subjects of different age-ranges, sex ratios and clinical groupings as well as employing different types of observer and assessment method. Although these broad findings are of value, several limitations are evident. In particular, differences which may exist between sexes and different age-ranges are obscured and the few studies which have investigated this (Jenkins & Glickman, 1946; Arnold & Smeltzer, 1974; Achenbach, 1978; Soli *et al*, 1981) do indicate that such effects are indeed present. In addition, most studies have employed parents or teachers to make ratings on subjects and only two studies have employed health service professionals to make direct clinical ratings on clinic attenders (Collins *et al*, 1962; Borgatta & Fanshel 1965).

If such analyses are to provide a sound basis for psychiatric diagnostic or classificatory schemes, it would seem important to use symptom ratings made on psychiatric clinic attenders by experienced clinicians, for ratings made by such groups as teachers, though widely employed (Thorley, 1983), have been shown in normal subjects to lack sensitivity, especially to emotional pathology (Green *et al*, 1980). Perhaps most important, as Achenbach (1978) has pointed out, is the lack of clinical validity for the symptom dimensions revealed by multivariate analysis of rating scale data, although a study by Neuchterlein *et al* (1981) has made a notable attempt to rectify this deficiency.

For these reasons, the present study was designed to provide a factor analysis of contemporary clinical symptom ratings made by psychiatrists on child and adolescent clinic attenders. Also, a systematic analysis of age and sex differences has been conducted as well as an attempt made to provide clinical support for the symptom factors by relating them to formal psychiatric diagnoses made on the same subjects.

A more limited aim of the study was simply to provide clinicians and researchers with a strategy to reduce the symptom data collected by the use of the clinical data sheets developed by the Bethlem Royal and Maudsley Hospitals to a more manageable and yet robust form.

Method

All intellectually normal (IQ > 70) child and adolescent clinic attenders at the Maudsley and Bethlem Royal Hospitals over

TABLE I

Symptom items rated by clinicians

Emotional symptoms

1. Abnormal suspiciousness or 'sensitivity'.
2. Morbid anxiety, worrying or panic.
3. Morbid depression, sadness, unhappiness, tearfulness.
4. Situation- or object-specific fears or phobias.
5. Ruminations, obsessions, rituals or compulsions (do not include faddiness, rigidity or other obsessive traits).
6. Suicidal ideas, attempt or threat.
7. Hypochondriasis.
8. Morbid irritability, screaming, tempers, breath-holding attacks.
9. School refusal, or phobia or crying on arrival at school.
10. Abnormally elevated mood (including hypomania).
11. Depersonalisation or derealisation.
12. 'Conversion' hysterical symptoms (do not include histrionic behaviour).

Somatic

13. Disturbance of eating (pica, refusal, abnormal faddiness etc.).
14. Disturbance of sleeping (insomnia, nightmares, sleepwalking etc.).
15. Pains of mental origin (headache, backache, abdominal pains, nausea, leg pains).
16. Encopresis or faecal soiling.
17. Enuresis.
18. Any non-epileptic disturbance of consciousness (fainting etc.).

Disturbance of relationships

19. Overt disturbance of child–mother relationship (including hostility, dependency etc.).
20. Overt disturbance of child–father relationship.
21. Overt disturbance of relationship with other adults (e.g. school teacher).
22. Overt disturbance of patient–sib relationship (including morbid rivalry or jealousy).
23. Overt disturbance of relationships with other children (including isolation, failure to make friends etc.).
24. Autism, social withdrawal, aloofness or detachment (not necessarily psychotic).
25. Socially disinhibited.

Speech and language

26. Disorder of rhythm (e.g. stuttering).
27. Disorder of articulation.
28. Disorder of comprehension of spoken language.
29. Disorder of production of spoken language (including simple retardation of language development).
30. Elective mutism.

Motor

31. Tics.
32. Other abnormal repetitive movement (whirling, flapping, twisting of hands, etc.).
33. Clumsiness or poor coordination.
34. Restlessness or fidgetiness.
35. Gross overactivity.
36. Hypoactivity.

Habitual manipulations

37. Thumb-sucking, tongue-sucking, rocking, masturbation, nailbiting, scratching, head-banging etc.

Antisocial behaviour or disorders of conduct

38. Disobedience (active or passive) or lying.
39. Stealing.
40. Destructiveness or malicious damage.
41. Fire-setting.
42. Truancy or staying out late.
43. Running or wandering away from home.
44. Sexual misbehaviour (assault, exposure etc.).
45. Fighting, bullying, aggression etc.
46. Violent assault (stabbing or use of other weapon or corrosives, severe physical attack etc.).
47. Taking drugs.
48. Other antisocial behaviour (raising fire alarms etc.).

Other

49. Disorder of sex role, sex object or gender identity (cross-dressing, abnormal feminine behaviour in boys, homosexual orientation etc.).
50. Hallucinations, delusions, ideas of reference or morbid persecutory ideas.

a 9-year period (1973–1981) formed the subject group ($n = 2602$). Clinical information was recorded by therapeutically involved psychiatrists within 2 weeks of admittance. This data gathering and storage system has been described in detail elsewhere (Thorley, 1982) but the principal features of it are that the clinician records the clinical diagnosis (ICD 9) and other elements on a multi-axial scheme (Rutter *et al*, 1979) together with Likert-type ratings labelled 'absent', 'minimal or dubious' and 'definitely present' (scored 0, 1 and 2) on 50 symptom items (see Table I). Other details recorded, but not used in the present study, include family, demographic and referral details. Of some importance is that no particular hypothesis is under consideration at the time these ratings are made. In fact, the system is operated routinely in conjunction with the recording of casenote information. The symptom ratings made are each intended to record a global clinical judgement, based on information from all sources, relating to the previous 12 months' functioning. In practice, the data sheets are completed by a very large number of clinicians, the majority being registrars who spend a 6-month period in the Children's Department. The symptom items used have been selected in an evolutionary manner over several decades of operating such a system to satisfy clinical experiences and research requirements and are set out on the rating sheets under the following headings: emotional symptoms, somatic, disturbance of relationships, speech and language, motor, habitual manipulations, antisocial behaviour, and other. This clinical information is stored on computer for convenient and rapid access.

The analyses were conducted with certain criteria and objectives in mind. The overall aim of the study was to produce clinically meaningful and robust (generalisable) factor scales. As a first step, to limit the error variance, all symptom items which had been responded to in less than 10% and in more than 90% of cases were excluded from the analysis. Factors were then extracted using a principal components analysis, but some symptom items in which there were a significant amount of missing data (e.g. relationship with siblings, child–father relationship) were excluded from analysis. However, other information relating to the same cases was retained and used appropriately. Each analysis produced a large number of factors: 34 for an analysis of the entire clinic population, 29 for clinic subjects under 11 years of age and 21 for subjects aged 12 years and over. However, most of these factors consisted of a single symptom item or weak loadings on several. To reduce this number, the factors were rotated orthogonally by the varimax method. The initial criterion for retention of the rotated factors was an eigenvalue of 1.0. If this failed to produce a scale of four symptom items or more with factor weights of 0.30 or better, the number of factors retained for rotation was reduced still further until an optimal factor set was found. The objective of this method was to avoid a second-order analysis of factors but instead to aim for a parsimonious and robust selection at the outset. Choosing an orthogonal rotation method in factor analysis is to make certain assumptions about the underlying pattern of the data. This method was chosen because discrete dimensions of symptoms were expected on the basis of previous work with this type of data (e.g. Achenbach, 1978; Miller, 1967); however, all analyses were repeated using oblique rotation which resulted in factor groupings that were substantially similar. Therefore, there was no reason to abandon the varimax (orthogonal) solution.

Methodological issues

There are particular problems of reliability and validity which need to be considered in a study of this type. The reliability of the clinical ratings are of particular concern because they are made by a constantly changing population of clinicians, usually registrars with, in all probability, differing degrees of accuracy. Although some training is given in the completion of the clinical data sheets, it is not possible to be sure that the judgement of one rater is comparable with that of another because consistency checks are not carried out routinely. This particular type of unreliability can usually be assessed by tests of inter-rater reliability. However, this is impractical to do on the present data and requires a prospective study which is beyond the scale of this present project. If inter-rater agreement *is* poor, it is likely to result in substantial levels of error variance and make the extrapolation of robust factors difficult to achieve. If meaningful factors fail to emerge, this may well be one explanation.

A second problem of reliability concerns the use of Likert-type ratings, which are ordinal rather than of interval quality. This affects the underlying assumptions of factor analytical techniques which require variables of the latter type. This requirement is often disregarded in practice, but if inferences are to be made of other clinic populations and of the relationship between symptoms in general, it is important that this possible source of error be investigated. To accomplish this, a series of sensitivity analyses were carried out on each of the derived factor solutions. The object was to see if the factors obtained were influenced unduly by the arithmetical nature of the variables rather than by their content.

A limited look at the validity of the symptom factors obtained was made possible by examining whether individual scores computed from each factor have some relationship to the eventual clinical diagnosis given by each clinician — in other words, whether the factor scores have the power to discriminate well between diagnostic categories.

Results

The analyses were carried out in several stages and are reported in sequential order.

Factor analysis of subjects divided into different age and sex groupings

For this procedure subjects were divided into four subgroups: 5–11-year old boys ($n = 703$, mean age 8.4 years, s.d. = 1.9); 5–11-year old girls ($n = 335$, mean age 8.6 years, s.d. = 1.9) and 12–18-year old girls ($n = 691$, mean age 14.0 years, s.d. = 1.3). The factor sets for each of the subgroups

TABLE II

Factor analysis of clinic symptom data in two subpopulations: younger subjects (5–11) and older subjects (12–18)

Factor labels	*Symptom item numbers and factor weights*	*% common variance*
5–11-year old boys and girls		
I. Conduct	38(0.58), 39(0.57), 40(0.51), 42(0.54), 43(0.55), 45(0.41)	45.4
II. Relationships	8(0.32), 19(0.43), 20(0.42), 21(0.56), 22(0.50), 23(0.58)	26.3
III. Emotionality	1(0.38), 2(0.72), 3(0.40), 4(0.50), 9(0.47), 13(0.32), 14(0.38), 15(0.33)	18.1
IV. Developmental immaturity	25(0.53), 27(0.31), 33(0.48), 34(0.66), 35(0.61)	10.3
12–18-year old boys and girls		
I. Relationships	1(0.30), 8(0.42), 19(0.44), 20(0.43), 21(0.54), 22(0.48), 23(0.57), 25(0.30), 40(0.38), 45(0.42)	42.0
II. Conduct	38(0.59), 39(0.55), 42(0.62), 43(0.43)	35.1
III. Emotionality	2(0.53), 4(0.51), 7(0.53), 9(0.46), 15(0.48)	12.7
IV. Depression	3(0.65), 6(0.60), 14(0.43), 13(0.30)	10.2

were generally similar. Thus, in all groups factors of conduct disturbance and emotionality appeared, accounting for between 53 and 85% of the total common variance (after rotation). For both of the male groups, factors reflecting disturbance of relationships were present (younger = 19.5% of variance; older = 37.7% of variance), but the same symptom items, though not forming a distinct factor, were present in the conduct disturbance factors of both female groups. A factor consisting of overactivity and developmentally related items appeared quite strongly for younger boys (11.2% of variance) and younger girls (15.1% of variance), not at all for older girls and weakly for older boys (9.1% of variance). Finally, items suggesting depression appeared as a strong factor for older girls but were contained in the emotionality factor for older boys.

A consideration of these patterns resulted in the decision to reduce the subgroups to just two, younger subjects and older ones (5–11 years and 12–18 years). The items and factors which emerged over the four subgroups suggested that differences between sexes were not as important as those between age-groups, for the chief differences lay in the presence of depression-related items in older subjects, contrasting with what might be called developmental immaturity-type items in younger subjects (overactivity, fidgetiness, social disinhibition, disorder of articulation etc.).

Factor analysis of subjects divided into older and younger age bands

The results of this are presented in Table II and show four factors emerging for each of the age-groups, labelled for the younger subjects, 'conduct disturbance', 'disturbance of relationships', 'emotionality' and 'developmental immaturity'. For the older subjects, the same factors occur except for 'developmental immaturity' which is replaced by one of 'depression'.

These two sets of factors seem the most useful for clinical and research use, providing the age-ranges of subjects is similar. However, because it seems that both factor sets have much in common and also because subjects in research do not always come conveniently in similarly convenient age-ranges, there seemed some justification in attempting to fit the whole clinical population to a single set of factors, even if these were fewer in number.

Factor analysis of the entire clinic population

Using 2602 subjects, the analysis produced three distinct factors labelled 'conduct disturbance', 'emotionality' and 'developmental immaturity', each accounting for 42.0, 33.9 and 24.1% of the common variance, and containing 12, 11 and 7 symptom items respectively (Table III).

Correlations between factor scores

Correlation analyses were carried out on the three factor structure for the whole clinic population and also on each of the four factor sets for the two clinic groups subdivided by age. The results are presented in Table IV and, in general, confirm the statistical independence of the factors. Factor scores were computed for each case simply by summing items which had the highest weights on each factor. The highest intercorrelation for the factor set of the youngest age-band subjects was between the conduct and relationship factors (0.32); the next, a negative one between conduct and emotionality of −0.24, then one of 0.20 between relationships and developmental immaturity with the other correlations being at a lower level of association.

For the older age-band children, the strongest association of factors was of 0.37 between conduct and relationships followed by one of 0.36 between depression and emotionality. The other correlations were of a much lower order.

Finally, the three-factor structure for both age-bands combined had inter-factor correlations of a very low order. The highest, 0.18, was between developmental immaturity and conduct, whilst those between conduct and anxiety, anxiety and developmental immaturity were 0.

It is perhaps worth pointing out that, with the very large subject numbers involved in these correlation analyses, though reaching statistical significance, these weak correlations are probably clinically irrelevant.

TABLE III

Factor analysis of symptom data on all clinic cases (item factor weights on each of the three factor scales)

	I. Conduct Factor				*II. Emotionality Factor*				*III. Immaturity Factor*		
Item	*I*	*II*	*III*	*Item*	*I*	*II*	*III*	*Item*	*I*	*II*	*III*
8	0.33	0.18	0.24	1	0.07	0.42	0.06	24	0.00	0.27	0.31
19	0.39	0.28	0.04	2	−0.12	0.67	0.00	25	0.27	−0.08	0.41
20	0.39	0.21	0.01	3	0.09	0.54	−0.13	26	−0.05	−0.03	0.32
21	0.49	0.07	0.17	4	−0.13	0.46	0.03	27	−0.02	−0.06	0.44
22	0.44	0.14	0.13	5	−0.12	0.30	0.18	29	−0.06	−0.04	0.49
23	0.34	0.25	0.30	6	0.10	0.31	−0.13	33	−0.05	0.00	0.44
38	0.68	−0.16	0.03	7	−0.06	0.36	−0.05	35	0.08	−0.05	0.46
39	0.49	−0.28	−0.15	9	−0.01	0.38	−0.14				
40	0.52	−0.14	0.17	13	−0.01	0.33	0.06				
42	0.46	−0.15	−0.23	14	0.02	0.40	0.04				
43	0.46	−0.05	−0.01	15	−0.07	0.34	−0.07				
45	0.53	−0.14	0.15								
Variance = 42.0%						33.9%				24.1%	

TABLE IV

Inter-correlations between factor scale items

Factor	*I*	*II*	*III*	*IV*
Factor set for younger subjects (ages 5–11)				
I. Conduct	1	0.32*	−0.24*	0.11*
II. Relationships	—	1	0.17	0.20*
III. Emotionality	—	—	1	0.02
IV. Developmental immaturity	—	—	—	1
Factor set for older subjects (ages 12–18)				
I. Relationships	1	0.37*	−0.01	0.15*
II. Conduct	—	1	−0.18*	−0.02
III. Emotionality	—	—	1	0.36*
IV. Depression	—	—	—	1
Factor set for all subjects (ages 5–18)				
I. Conduct	1	−0.01	0.18*	
II. Emotionality	—	1	−0.02	
III. Developmental immaturity	—	—	1	

*$P<0.001$.

Sensitivity analyses

When the factor structures had been obtained, the process was repeated, but this time the values of the symptom ratings were changed. The original rating values of 0, 1 and 2 were recoded to 0, 1 and 10 respectively and again to 0, 9 and 10 respectively. Each time the data were recorded in this way, repeats of the factor analyses were performed. Thus, a replication of the factor structures of (a) the entire clinic population, (b) the 5–11 year age-group and (c) the 12–18 year age-group was carried out.

Despite the gross distortion of the symptom rating values, the results of these procedures were that the original factor structures were replicated very closely with only minor variations being apparent. Therefore some confidence may be had in the factor structures reported, even though the symptom rating data was obtained from Likert-type ratings and thus did not conform to the usual assumptions of factor analysis. This stability is quite likely to be a consequence of the large number of subjects employed in this study.

Relationship of the symptom factor scales to diagnostic categories

This was carried out on the three factor scales derived from the whole clinic population and the hypothesis is that subjects diagnosed 'conduct disorder' should have higher scores on the conduct disturbance factor than either subjects diagnosed 'emotional disorder' or 'hyperkinetic syndrome'. In a similar way, subjects diagnosed 'emotional disorder' should have scores on the emotionality factor that significantly exceed those of the other two diagnostic groups. The same comparisons should also hold true for the 'hyperkinetic syndrome' subjects on the developmental immaturity factor scale.

The outcome of the comparisons (Table V) using one-way analysis of variance and Scheffe's *post-hoc* comparisons (0.10 α) supported the hypothesis without exception.

Age and sex trends

Factor set for younger subjects

Two-way analysis of variance was conducted to explore the effects of age (between the age-bands 5–6, 7–8, 9–10 and 11) and sex. The results are set out in Table VI.

TABLE V

Factor scale scores of conduct, emotionality and developmental immaturity of corresponding diagnostic groups (one-way ANOVA and Scheffe post-hoc comparisons)

	Conduct scale scores			*Emotionality scale scores*			*Developmental immaturity scale scores*		
Subject groups	*n*	$\bar{x}$	*s.d.*	*n*	$\bar{x}$	*s.d.*	*n*	$\bar{x}$	*s.d.*
1 Conduct disorder	872	9.32	4.9	908	3.01	3.13	926	1.41	2.17
2 Emotional disorder	371	6.30	3.7	384	5.52	3.74	383	1.51	2.45
3 Hyperkinetic syndrome	58	6.68	4.7	65	1.63	1.63	63	6.30	3.47
	1301								
One-way ANOVA									
F ratio	58.78			92.56			131.31		
F probability	$P<0.0000$			$P<0.0000$			$P<0.0000$		
Scheffe's *post-hoc* tests (0.10 α)	[1]Subset 1 = Group 1 Subset 2 = Groups 2, 3			[2]Subset 1 = Group 3 Subset 2 = Group 1 Subset 3 = Group 2			[1]Subset 1 = Groups 1, 2 Subset 2 = Group 3		

1. Statistically significant differences between subject groups in subset 1 and the groups in subset 2.
2. Statistically significant differences between all three subset groups.

TABLE VI

Analysis of variance carried out on the factor set for younger (5–11 years) subjects, exploring age and sex effects

	Sums of squares	*d.f.*	*Mean square*	*F*	*Significance (P)*
I. Conduct factor					
Main effects	364.2	4	91.0	11.5	0.001
Age	208.7	3	69.6	8.8	0.001
Sex	188.2	1	188.2	23.8	0.001
Two-way interactions	13.7	3	4.6	0.6	0.629
Explained	377.9	7	53.9	6.8	0.001
Residual	13 839.6	1748	7.9		
II. Social relationships factor					
Main effects	48.7	4	12.2	1.2	0.320
Age	38.7	3	12.9	1.2	0.293
Sex	10.3	1	10.3	0.9	0.320
Two-way interactions	3.0	3	0.9	0.09	0.962
Explained	51.7	7	7.3	0.7	0.662
Residual	17 350.5	1674	10.4		
III. Emotionality factor					
Main effects	688.8	4	172.2	20.5	0.001
Age	530.9	3	176.9	21.1	0.001
Sex	112.5	1	112.5	13.4	0.001
Two-way interactions	21.8	3	7.3	0.9	0.458
Explained	710.6	7	101.5	0.9	0.001
Residual	14 598.7	1741	8.4		
IV. Developmental immaturity factor					
Main effects	413.2	4	103.2	24.0	0.001
Age	340.7	3	113.6	26.4	0.001
Sex	48.8	1	48.8	11.3	0.001
Two-way interactions	15.0	3	5.0	1.2	0.324
Explained	428.2	7	61.2	14.2	0.001
Residual	7567.0	1757	4.3		

Conduct factor scale. Highly significant age and sex effects were found. Mean scores generally increased with age for both boys, particularly at age 11, but girls in every age-category returned lower mean scores.

Social relationships factor scale. No significant age, sex or interactive effects were found. The mean scores of both boys and girls were remarkably similar and were constant throughout all age-bands.

Emotionality factor scale. Highly significant age and sex effects but no interactive effects were found. Scores for boys and girls increased with age and in every age-band the mean scores of girls exceeded those of boys. The mean scores for both sexes increased sharply at age 11.

Developmental immaturity factor scale. Highly significant age and sex effects were found but no interactive ones. Mean scores for both sexes declined quite sharply with increasing age in every age-band. The mean scores for girls were considerably lower.

Factor set for older subjects

Similar analyses were carried out on the factor scales appropriate for subjects over 11 years of age. Two age-bands were employed: 12–13 years and 14–18 years. Results of the analysis of variance are set out in Table VIII.

Relationship factor scale. For this, as with the similarly named scale for younger subjects, no main or interactive effects were found. The mean scores for both boys and girls over the period of adolescence remained fairly constant.

Conduct factor scale. Moderately significant effects for age and sex were found but none for interaction. The mean scores for girls on both age bands were slightly lower and for both sexes the mean scores fell with increasing age.

Emotionality factor. Significant effects for sex but not for age or interaction were found. The mean scores for both age-bands were similar but were moderately elevated in both age categories for girls.

TABLE VII

Analysis of variance carried out on the factor set for older (12–18 years) subjects, exploring age and sex effects

	Sums of squares	*d.f.*	*Mean square*	*F*	*Significance (P)*
I. Relationship factor					
Main effects	53.8	2	26.9	1.6	0.212
Age	0	1	0	0	0.997
Sex	53.8	1	53.8	3.1	0.078
Two-way interactions	45.8	1	45.8	2.6	0.104
Explained	99.7	3	33.2	1.9	0.125
Residual	25 839.3	1491	17.3		
II. Conduct factor					
Main effects	70.7	2	35.3	4.1	0.017
Age	40.0	1	38.0	4.4	0.036
Sex	33.2	1	33.2	3.8	0.050
Two-way interactions	5.5	1	5.5	0.6	0.423
Explained	76.2	3	25.4	2.9	0.032
Residual	13 351.6	1547	8.6		
III. Emotionality factor					
Main effects	44.3	2	22.1	4.8	0.008
Age	14.4	1	14.4	3.1	0.077
Sex	29.6	1	29.6	6.4	0.011
Two-way interactions	9.1	1			
Explained	54.3	3	17.8	3.9	0.009
Residual	7184.3	1562	4.6		
IV. Depression factor					
Main effects	409.2	2	204.6	56.0	0.001
Age	44.9	1	44.9	12.3	0.001
Sex	365.3	1	365.3	99.9	0.001
Two-way interactions	40.7	1	40.7	11.1	0.001
Explained	449.8	3	149.92	41.0	0.001
Residual	5694.2	1557	3.7		

TABLE VIII
Age and sex effects on each of three major symptom factors of the clinic population

		Conduct			*Emotionality*			*Developmental immaturity*		
Age band		*n*	$\bar{x}$	*s.d.*	*n*	$\bar{x}$	*s.d.*	*n*	$\bar{x}$	*s.d.*
5–6	male	124	7.32	5.05	128	1.96	2.25	130	2.40	2.91
	female	57	5.68	4.55	61	3.32	3.47	61	1.49	2.60
7–8	male	194	6.86	5.27	204	2.85	3.06	205	2.13	2.51
	female	82	6.04	4.81	83	3.03	3.11	85	1.03	1.76
9–10	male	224	6.77	5.01	239	3.17	3.39	241	1.43	1.98
	female	110	5.76	4.55	111	3.46	2.96	115	0.74	1.77
11–12	male	262	7.65	5.55	271	4.08	3.78	276	1.06	1.69
	female	169	6.70	4.61	177	5.14	4.43	181	0.58	1.43
13–14	male	371	7.93	5.17	373	4.05	3.97	377	0.72	1.22
	female	295	7.23	5.30	301	4.93	3.72	311	0.55	1.10
15+	male	309	7.83	5.27	325	4.11	3.97	332	1.03	1.79
	female	234	6.37	4.53	248	5.46	3.56	255	0.61	1.19
ALL MALE		1484	7.49	5.25	1540	3.60	3.68	1561	1.28	1.99
ALL FEMALE		947	6.56	4.84	981	4.68	3.77	1008	0.69	1.47
TOTAL		2431	7.13	5.11	2521	4.02	3.75	2569	1.05	1.83

ANOVA	*Sums of squares*	*d.f.*	*F*	*P*	*Sums of squares*	*d.f.*	*F*	*P*	*Sums of squares*	*d.f.*	*F*	*P*
Main effects	1041.4	6	6.7	0.001	2038.0	6	25.5	0.001	690.7	6	37.3	0.001
Age	533.2	5	4.1	0.001	1335.5	5	20.1	0.001	478.9	5	31.0	0.001
Sex	615.7	1	23.7	0.001	498.8	1	37.4	0.001	146.8	1	47.6	0.001
Interaction	60.7	5	0.5	0.779	98.5	5	1.4	0.193	51.1	5	3.3	0.006

Depression factor. Highly significant age, sex and interactive effects were found. Mean scores for both girls and boys increased during the adolescent period but were notably higher for girls.

Factor set for all subjects

For each of the three factor scales, significant age and sex effects were found and, in addition, an interactive one on the developmental immaturity factor. Complete data are presented in Table VIII, in which mean values are given for the age- and sex-bands.

Conduct factor scale. Mean scores for both girls and boys increased with age and, in every age-band, were higher for boys.

Emotionality factor scale. Mean scores for both sexes increased with age quite consistently but, in contrast to the conduct factor scale, mean scores in every age-band were higher for girls.

Developmental immaturity factor scale. Mean scores generally declined with age and were lower in all categories for girls.

Summary of Results

Initial factoring of four subgroups, 5–11-year old girls and boys and 12–18 year old girls and boys revealed a factor pattern different essentially between age-groups rather than sexes. Two factor sets were then produced, each of four factors for the younger (conduct, relationships, emotionality and developmental immaturity) and older (relationships, conduct, emotionality and depression) subgroups. Nearly all of the factor scales revealed significant age and sex effects except for the social relationships scales for both the older and younger subjects, which showed neither, and the emotionality scale for the older subjects, which only showed a sex effect. Interactive effects were found on the depression (older subjects) and developmental (all subjects) factor scales.

Discussion

The results of the symptom factor analysis for both the younger (5–11 years) and older (12–18 years) clinic attenders are interesting in that, although factors labelled 'conduct', 'depression' and 'emotionality' are commonly found in other studies (Achenbach, 1978), ones labelled 'developmental immaturity' and 'social relationships' are not.

The developmental factor found in the younger group comprises symptoms reflecting overactive, socially disinhibited behaviour with poor coordination, speech and language delay. The factor is interesting in that it contains one of the central symptom features of hyperactive disorder. In reviewing factor studies of symptom data, Quay (1979) concluded that there was little evidence for a separate factor of hyperactivity as distinct from one of conduct disturbance and, indeed, in this study it seems likely that, if it were not for the presence of developmentally related items such as speech, language and motor dysfunctioning, it is doubtful whether the items related to hyperactivity would have remained independent of the conduct disturbance dimension. In conceptual terms this association of symptoms suggestive of developmental immaturity with those of hyperactivity is of interest and may explain why in some studies it emerges as a distinct dimension and in others it does not. Zukow *et al* (1978) confirmed the importance of this aspect of hyperactivity when, in attempting to devise a rating scale that would distinguish hyperactive from normal children, they found three major factors for a parent scale and two for a teacher scale. One factor which was common to both scales was one of motor coordination in which, interestingly, there was an item reflecting speech difficulties. In addition, Thorley (1984) found that disorder of articulation was one of the symptoms which distinguished the hyperkinetic syndrome children (ICD 9 diagnosis) from those with conduct disorder, although no differences in gross brain pathology were detected between the two groups. More broadly, Safer & Allen (1976) documented many studies in which numerous developmental features were associated with hyperactivity. These associations may thus explain why a separate factor with a hyperactivity-related item appeared in this, but less frequently in many other symptom factor studies.

The second factor of particular interest which appears in this study for both age-bands is the factor reflecting a disturbance of social relationships. This is satisfying from a conceptual and clinical viewpoint, since this type of disturbance, neglected in diagnostic schemes, seems to have important predictive value, as demonstrated in several studies (Robbins, 1966; Roff & Sells, 1968; Roff, 1969), and can be viewed as one of the more promising clinical predictors of outcome in adolescence and adulthood (Kohlberg *et al*, 1972).

The emergence of the factors of social relationships and developmental immaturity in this study and not others can be explained in part by the unique role that a clinican rather than a parent or teacher occupies. Symptom judgements about developmental pathology are difficult for parents or teachers to make and the clinician is in a uniquely advantageous position to assess disturbance of social functioning, because a judgement based on several sources of information can be employed in contrast to the narrower observational range of teachers or parents. Since all but two of the major symptom factor studies have employed respondents other than clinicians, it may not be too surprising that these factors have not emerged. Of course, an alternative explanation is that these factors are simply the result of a 'halo' effect: an artifact of certain clinical perceptions in which symptoms are recorded as being associated in a way that is not a true reflection of the real situation.

A further point of interest arising from the factor structures for the two broad age-bands is the appearance of a factor of depressive symptoms in adolescence. Mean scores on the emotionality factor scale for 5–11-year olds were higher for girls and increased with age. In adolescence, however, scores on the similarly labelled factor were more even between the sexes and constant over time. What changes is that a factor of depression appears, and on it girls score substantially higher, with their mean scores rising throughout the adolescent period. The processes involved are not clear. It could be that in some cases, diffuse emotional symptoms in childhood, particularly in girls, manifest themselves later as a depressive disorder or, perhaps in the postpubertal years, depressive symptoms arise without such precursors. Much of the longitudinal work has been concerned with prediction from childhood to adulthood (Kohlberg *et al*, 1972), but it would be valuable to know more of the pre-adult processes of emotional psychopathology.

An intriguing finding in the factor set relating to the younger (5–11 years) subjects is that, on the conduct and emotionality factor scales, the mean scores for both boys and girls rise quite strongly at 11 years of age. It is not clear why this should occur. Stress associated with change of school is perhaps an obvious factor, but clearly there could be others; ranging from intrapsychic processes (e.g. changes in ego development) to factors associated with the onset of puberty and to psychosocial disturbances (e.g. moving home, which many families choose to do in the school changeover period). Again, the topic seems worthy of investigation.

Reassuringly for the study as a whole, it does seem that the scales derived from the data sheets have some internal consistency and concurrent validity. Scores on the conduct scale distinguish between children diagnosed conduct disordered (ICD 9) and those diagnosed as emotional or hyperkinetic disorder. Similarly, scores on the emotionality scale distinguish those diagnosed emotional disorder (ICD 9) and each of the other two diagnostic groups, whilst scores on the developmental immaturity scale distinguish between those with a diagnosis of hyperkinetic syndrome and those with emotional or conduct disorder.

Finally, one of the most significant findings of this study is the importance of age and sex effects. Significant age effects are present in all three factor solutions on all scales except for that of 'social relationships'. This supports the findings of the few studies that have investigated such effects (Jenkins & Glickman, 1946; Arnold & Smeltzer, 1974; Achenbach, 1978; Soli *et al*, 1981) and taken together suggest that, in order to be useful, clinical rating scales require normative data along age-bands.

The importance of gender is also demonstrated. It has been suggested by Stewart *et al* (1981) that female clinic attenders may have specific syndromes (other than anorexia nervosa) unique to their sex. A study by these authors of 41 female inpatients found a weak suggestion of an elevation of somatic complaints in girls which may be attributed to hysteria. No evidence, at least in terms of broad symptom dimensions, was found in this present study to support this or any other qualitative difference between the sexes: the factor solution for both produced similar results. However, this is undisputedly influenced by the range of items put into the factor analysis. Where more items are included, the emergence of distinct sex-related factor structures is more likely (e.g. Soli *et al*, 1981). However, important quantitative differences are present. The three-factor solution for all age-groups showed that boys return higher mean scores in every age-band on the conduct disturbance scale. This pattern was not unexpected given the findings of several studies (e.g. Chazan & Jackson, 1971; Shepherd *et al*, 1971) which have demonstrated that boys more commonly than girls show oppositional and antisocial behaviour. Rutter *et al* (1970) also revealed from the Isle of Wight survey data that neurotic problems were much more common in girls. This is also supported by the gender differences on the emotionality factor scale where, on every age-band, girls returned higher mean scores than boys. On the third factor, developmental immaturity, boys returned higher mean scores on every age-band but, unlike the conduct or emotionality mean scores, the scores for both boys and girls decreased with age. This may be a true phenomenon—that such pathology decreases over time—or it may simply reflect a bias towards referral at an earlier rather than a later age. The finding of important effects for both age and sex as intervening variables suggest that clinical rating scales should be constructed within clearly defined age-ranges and that normative data should be available for both age- and sex-bands. From this aspect, the approach of Achenbach (1978) in constructing standardised scales for different age ranges and sex compositions seems justified.

In conclusion, the results seem to underline the potential value of this type of rating scale in which clinicians draw information from as many informants as possible by interview and perhaps questionnaires, and then make symptom ratings as a precursor to a diagnostic decision. Clearly, this is a conceptually different instrument than the psychiatric rating scale, a number of which are now available (e.g. Rutter & Graham, 1968; Kestenbaum & Bird, 1978; Hodges *et al*, 1981) but which are designed to collect information from the psychiatric interview alone. Since agreement between information from this source and that provided in a similar way by parents using similar scales is often poor (Reich *et al*, 1982), it is often difficult to quantify the process which eventually leads to a clinical diagnosis being given. The symptom rating scale of the Maudsley and Bethlem Royal Hospitals may be of value in research by providing symptom information more closely allied to the diagnostic decision than single source scales are able to do. From this point of view, and also as an integral part of a clinical data record system (Thorley, 1982), the symptom rating scales seem to have some value.

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References

ACHENBACH, T. M. (1978) The classification of child psychopathology: a review and analysis of empirical efforts. *Psychological Bulletin*, **85**, 1275–1301.

ARNOLD, L. E. & SMELTZER, D. J. (1974) Behaviour check list factor analysis for children and adolescents. *Archives of General Psychiatry*, **30**, 799–804.

BORGATTA, E. F. & FANSHEL, D. (1965) *Behavioural Characteristics of Children Known to Psychiatric Out-patient Clinics*. New York: Child Welfare League of America.

CHAZAN, M. & JACKSON, S. (1971) Behaviour problems in the infant school. *Journal of Child Psychology and Psychiatry*, **12**, 191–210.

COLLINS, L. F., MAXWELL, A. E. & CAMERON, K. A. (1962) A factor analysis of some child psychiatric clinic data. *Journal of Mental Sciences*, **108**, 275–285.

GREEN, K. D., BECK, S. J., FOREHAND, R. & VOSK, B. (1980) Validity of teacher nominations of child behavior problems. *Journal of Abnormal Child Psychology*, **8**, 397–402.

Hodges, K., Kline, J., Fitch, P. & McKnew, D. (1981) The child assessment schedule: a diagnostic interview for research and clinical use. *Catalogue of Selected Documents in Psychology*, **11**, 56.

Jenkins, R. L. & Glickman, S. (1946) Common syndromes in child psychiatry. I. Deviant behaviour traits: II. The schizoid child. *American Journal of Orthopsychiatry*, **16**, 244–261.

Kestenbaum, C. J. & Bird, H. R. (1978) A reliability study of the mental health assessment form for school age children. *Journal of the American Academy of Child Psychiatry*, 17, 338–344.

Kohlberg, L., LaCross, J. & Ricks, D. (1972) The predictability of adult mental health from childhood behaviour. In *Manual of Child Psychopathology* (ed. B. B. Wolman). New York: McGraw Hill.

Lorr, M. & Jenkins, R. (1953) Patterns of maladjustment in children. *Journal of Clinical Psychology*, **9**, 16–19.

Miller, L. C. (1967) Dimensions of psychopathology in middle childhood. *Psychological Reports*, **21**, 897–903.

Neuchterlein, K. H., Soli, S. D., Garmezy, N., Devine, V. T. & Schaefer, S. M. (1981) A classification system for research in childhood psychopathology: Part II. Validation research examining converging descriptions from the parents and from the child. In *Progress in Experimental Personality Research*, (ed. B. A. Maher) Vol. 10. New York: Academic Press.

Quay, H. C. (1979) Classification. In *Psychopathological Disorders of Childhood* (eds H. C. Quay & J. S. Werry). New York: Wiley.

Reich, W., Herjanic, B., Welner, Z. & Gandhy, P. R. (1982) Development of a structured interview for children: agreement on diagnosis comparing child and parent interviews. *Journal of Abnormal Child Psychology*, **10**, 325–336.

Robbins, L. N. (1966) *Deviant Children Grown Up: A Sociological and Psychiatric Study of Sociopathic Personality*. Baltimore: Williams & Wilkins.

Roff, M. (1969) Some life history factors in relation to various types of adult maladjustment. In *Life History Studies in Psychopathology* (eds M. Roff & D. F. Ricks). Minneapolis: University of Minnesota Press.

—— & Sells, S. B. (1968) Juvenile delinquency in relation to peer acceptance-rejection and socio-economic status. *Psychology in the Schools*, **5**, 3–18.

Rutter, M. & Graham, P. (1968) The reliability and validity of the psychiatric assessment of the child: I. Interview with the child. *British Journal of Psychiatry*, **114**, 563–569.

——, Tizard, J. & Whitmore, K. (eds) (1970) *Education, Health and Behaviour*. London: Longman.

——, Shaffer, D. & Sturge, C. (1979) *A Guide to a Multiaxial Classification Scheme for Psychiatric Disorders in Childhood and Adolescence*. London: Institute of Psychiatry.

Safer, D. J. & Allen, R. P. (1976) *Hyperactive Children: Diagnosis and Management*. Baltimore: Universal Park Press.

Shepherd, M., Oppenheim, B. & Mitchell, S. (1971) *Childhood Behaviour and Mental Health*. New York: Grune & Stratton.

Soli, S. D., Neuchterlein, K. H., Garmezy, N., Devine, V. T. & Schaefer, S. M. (1981) A classification system for research in childhood psychopathology: Part I. An empirical approach using factor and cluster analyses and conjunctive decision rules. In *Progress in Experimental Personality Research*, (ed. B. A. Maher) Vol. 10. New York: Academic Press.

Stewart, M. A., Gath, A. & Hierowski, E. (1981) Differences between girls and boys admitted to a child psychiatry ward. *Journal of Clinical Psychiatry*, **42**, 386–388.

Thorley, G. (1982) The Bethlem Royal and Maudsley Hospitals' clinical data register for children and adolescents. *Journal of Adolescence*, **5**, 179–189.

—— (1983) Data on the Conners' teacher rating scale from a British clinic population. *Journal of Behavioural Assessment*, **5**.

—— (1984) Hyperkinetic syndrome of childhood—clinical characteristics. *British Journal of Psychiatry*, **144**, 16–24.

World Health Organization (1970) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases*. Geneva: World Health Organization.

Zukow, P. G., Zukow, A. H. & Bentley, P. M. (1978) Rating scales for the identification of treatment of hyperkinesis. *Journal of Consulting Clinical Psychology*, **45**, 213–222.

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The Relative Stability of Positive and Negative Features in Chronic Schizophrenia

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The mental states of 92 inpatients with chronic schizophrenia were assessed on two occasions 4 years apart. The relationship between the two ratings was examined in the 84 patients who were either consistently on or consistently off neuroleptics. Negative features were more stable than positive, but were not irreversible. Every feature examined was shown to resolve in some cases.

It has become customary to classify the typical abnormalities of the mental state of schizophrenic patients into positive and negative features, with reference to behavioural excesses and deficits; positive features are pathological by their presence and negative features represent the loss of some normal function. The relationship between positive and negative features may not be simple (Wing, 1978) but the following generalisations have some support. Positive features are characteristic of earlier and negative of later phases of the illness (Pfohl & Winokur, 1982a). The effects of drugs upon positive features are greater than those upon negative features. Thus neuroleptics produce more marked improvement (Johnstone *et al*, 1978; Angrist *et al*, 1980) and amphetamine more marked exacerbation (Angrist *et al*, 1980) of positive than of negative features. Positive features have been said to be relatively variable and negative features relatively fixed (Ovchinnikov, 1968; Sneznevsky, 1968). There is evidence that the clinical (e.g. behavioural and neurological) correlates of the two groups of abnormalities are not the same (Owens & Johnstone, 1980). On the basis of such findings it is suggested that although manifestations of the same disease, these features may result from different biological processes (Crow, 1980) with that underlying the positive features being potentially reversible and that underlying negative features irreversible. The latter proposition finds some support in the fact that while clinical correlates of the cerebral ventricular enlargement sometimes found in schizophrenia are not constant, those that do occur have tended to concern abnormalities associated with negative rather than positive features. Although the question of the reversibility/stability of positive and negative features has assumed theoretical as well as practical interest (Crow, 1980) the progress, over a period of time, of positive and negative features has received little formal study. In general it is difficult to separate the question of reversibility/stability from that of responsivity to treatment, either in terms of neuroleptics or changing social factors. Negative features can be affected to some extent by social factors (Wing & Brown, 1970) although once established some impairment is likely to persist even in the most satisfactory social environment (Wing, 1978). The present study examines the reversibility/stability of positive and negative features over a 4-year period in a group of schizophrenic patients in a constant hospital environment, some of whom were and some of whom were not, receiving neuroleptic drugs throughout the period of investigation.

Method

The subjects of this study were part of a large cohort of patients who had participated in an earlier investigation (Owens & Johnstone, 1980) of schizophrenic patients all of whom fulfilled the St Louis (Feighner *et al*, 1972) criteria for schizophrenia and had been in hospital continuously for at least 1 year. The 92 patients taking part in the present study had continued as inpatients for a further 4 years and had remained in one of six wards caring for patients of medium dependency although not physically ill or frail. A standard therapeutic regime was in practice in those wards during the relevant 4 years and no major changes in the social circumstances or policy were made during that time. This investigation concerns the comparison of the mental states of these patients on two occasions 4 years apart using the rating scale for chronic psychotic patients devised by Krawiecka *et al* (1977) modified to separate flattening and incongruity of affect (Johnstone *et al*, 1978) so that the items rated each on a five-point scale were anxiety, depression, retardation, incongruity of affect, incoherence of speech, delusions, hallucinations, poverty of speech and flattening of affect. These ratings were conducted by E.C.J. and D.C.G.O.

A re-check of inter-rater and test-retest reliability was

TABLE I

	D.G.C.O. vs E.C.J. % agreement	*Fisher's exact P value*	*% of cases in whom feature is present at a score of more than 1*
Depression	100	<0.02	18%
Anxiety	82	NS	13.6%
Delusions	100	<0.002	54.5%
Hallucinations	100	<0.02	18%
Incoherence	91	<0.02	41%
Poverty of speech	100	<0.01	36%
Flattening	73	<0.002	54.5%
Incongruity	82	NS $P<0.06$	27%
Retardation	73	NS	9%

conducted for this investigation using videotapes of interviews with 11 patients with chronic schizophrenia (Table I).

This table shows the percentage agreements between the ratings. Because of the small numbers involved the significance of these agreements was assessed by applying Fisher's exact test to 2×2 contingency tables obtained by collapsing the five-point scale into the presence (score 2–4) or absence (score 0–1) of a pathological feature. This cut-off was chosen because a rating of 1 on the Krawiecka *et al* (1977) scale indicates a score which is not clearly pathological. In most cases the agreements between the ratings are satisfactory (Table I). The exceptions are anxiety incongruity and retardation and the lower level of significance was partly due to the scarcity of these features in the population on which reliability was checked (Table I).

The raters were blind to all treatment and historical aspects when they conducted both assessments on the 92 participants of this study and the second ratings were conducted blind to the results of the first. Details of past and current neuroleptic and other physical treatment had been obtained at the time of the first assessments and this information was brought up to date following the second interviews.

Results

The sample consisted of 31 females and 61 males. The mean ages, durations of illness and durations of stay in hospital at the time of the first rating were 58.7 s.d. 10.5 years, 32.2 s.d. 9.1 years, and 27.0 s.d. 11.9 years for females and 54.7 s.d. 12.5 years, 29.6 s.d. 10 years, and 25.8 s.d. 11.9 for males. The mean scores on each feature of the modified Krawiecka *et al* (1977) scale on the first and second occasions of rating are illustrated in Fig. 1.

Sixty-six patients were prescribed neuroleptics on both occasions on which they were rated; 18 patients were prescribed neuroleptics on neither occasion, four were taking neuroleptics the first time but not the second and the remaining four were not taking neuroleptics on the first occasion but were taking them on the second. The numbers in the groups of patients on neuroleptics on one occasion but not the other were too small to be examined usefully. Only the findings on the 84 patients consistently taking, or not taking neuroleptics have been considered and they are shown in Table II.

Significant relationships were found between the ratings on the first and second occasions of depression, delusions, hallucinations, poverty of speech and flattening of affect, but not in the case of anxiety, incongruity of affect or incoherence of speech. A significant relationship was established for retardation only in patients consistently on drugs. Since large numbers of subjects were involved in these comparisons χ^2 tests were applied to the contingency tables relating to the first and second occasion. Anxiety, incongruity of affect and retardation had relatively low inter-rater and test-retest reliabilities (Table I) and this may contribute to the lack of agreement between first and second ratings. By contrast incoherence of speech was rated very reliably (Table I). Thus the lack of agreement between the two occasions on this relatively common feature is likely to reflect its variability. The differences between the ratings in patients taking and not taking neuroleptics were examined using the Mann-Whitney U test (Table II). In this study the drugs were not randomly allocated but were prescribed on the basis of clinical need. The higher score on delusions of patients taking neuroleptics compared with those not taking them may reflect this fact. On the other hand, the fact that scores on retardation, poverty of speech and flattening of affect are higher in patients not taking neuroleptics is an indication that the abnormality being measured here is not a drug effect. This is of relevance because of the known difficulty in separating negative features from drug-induced akinesia (Rifkin *et al*, 1975). Scores of stability were calculated on the basis of percentage emergence/resolution relative to consistent presence and these are also shown in Table II.

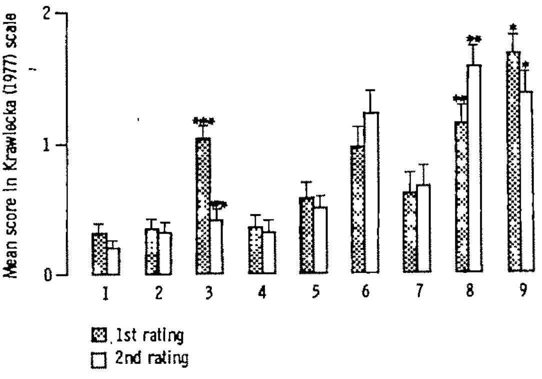

FIG. 1. Mean scores on the individual items of the Krawiecka *et al* (1977) scale in the total sample of 92 cases on the two occasions of rating. (*$P<0.05$, **$P<0.01$, ***$P<0.001$, McNemar test of changes.)
1 = Anxiety. 2 = Depression. 3 = Retardation. 4 = Incongruity of affect. 5 = Incoherence of speech. 6 = Delusions. 7 = Hallucinations. 8 = Poverty of speech. 9 = Flattening of affect.

TABLE II

	Within person comparisons			Number of cases				
	Patients on neuroleptics Significance of relationship between ratings	*Patients on and patients off neuroleptics Significance of relationship between ratings*	*Relationship between ratings of patients on and patients off neuroleptics > = worse*	*Never present*	*Consistently present*	*Emerged*	*Resolved*	*Percentage stability (rank)*
Anxiety	NS	NS	on>off NS $P<0.06$	68	1	5	10	12 (9)
Depression	$P<0.001$	$P<0.001$	on>off NS trend	64	6	6	8	46 (5)
Retardation	$P<0.001$	NS $P<0.06$	off>on $P<0.02$	50	9	3	22	42 (6)
Incongruity of affect	NS	NS	NS	65	3	7	9	27 (7)
Incoherence of speech	NS	NS	off>on NS $P<0.06$	57	4	9	14	26 (8)
Delusions	$P<0.003$	$P<0.01$	on>off $P<0.04$	45	13	16	10	50 (4)
Hallucinations	$P<0.01$	$P<0.0001$	NSD	55	13	5	11	62 (3)
Poverty of speech	$P<0.001$	$P<0.0001$	off>on $P<0.05$	35	25	18	6	68 (2)
Flattening of affect	$P<0.001$	$P<0.0001$	off>on NS $P<0.06$	27	31	9	17	70 (1)

Discussion

This study compared the stability of positive and negative features in a sample of long-stay hospital patients over a 4-year period. Of the nine features examined, anxiety, depression and retardation have been classified as non-specific features, incongruity of affect, incoherence of speech, delusions and hallucinations as positive features and poverty of speech and flattening of affect as negative features (Johnstone *et al*, 1978). In general the changes were not striking (Table II, Fig. 1). The patients showed no marked deterioration over a 4-year period but on the other hand the improvement in symptoms at ages above 55 years described by Bridge *et al* (1978) in their paper on burned out schizophrenia did not seem to be occurring.

The mean scores shown in Fig. 1 offer no information on the amount of change occurring in individual patients as changes in different directions in different patients would cancel the effect of one another. They demonstrate consistent trends across the whole population examined. Retardation showed the most change in mean score, this being significantly diminished on the second occasion (Fig. 1). The reliability of the scoring of this feature was relatively low (Table I) but while this could contribute to change it would not account for the consistent reduction in mean score. Over the 4 years of the study the patients had become better acquainted with the raters and this increased familiarity could be a plausible explanation for the lesser score on retardation and also for the less significant fall in flattening of affect. If this were so, however, it might be expected that poverty of speech would also decrease whereas it showed an increase in mean score (Fig. 1). Over the 4 years of the study the authors had discussed the difficulty of rating retardation and of separating it from poverty of speech and flattening of affect (Krawiecka *et al*, 1977). It may be that this concern led to more conservative rating of retardation on the second occasion than on the first.

Comparisons in Table II relate to the central issue of this study—the question of reversibility/stability of the features examined. The within-person ratings for retardation showed a significant relationship between first and second occasions only for patients taking neuroleptics and this feature was relatively low in the hierarchy of stability. Anxiety was the least stable of all features and although depression did show significant within-person relationships between occasions 1 and 2 it was placed at the midpoint in the hierarchy of stability.

Positive features showed a degree of variability, in that while delusions and hallucinations showed significant within-person relationships between occasions 1 and 2, and were placed fairly high in the hierarchy of stability, incongruity of affect and incoherence of speech showed no significant within-person relationships between occasions and were less stable than any other feature except anxiety.

By contrast, highly significant within-person associations between ratings 1 and 2 were found for both negative symptoms and they achieved the highest scores on stability. The study therefore provides support for the view that negative features are relatively stable and positive features relatively variable in circumstances where the effects of drug treatment and social change are likely to be minimal. It does, however, demonstrate that negative features are not entirely irreversible as they may be resolved (Table II). Indeed the frequency of resolution of flattening of affect found in this study is greater than the 6% noted by Pfohl & Winokur (1982b) in their study of 52 schizophrenic patients. It could be argued that the design of the present investigation may have led to underestimation of the difference in reversibility/stability between positive and negative features by including only patients who continued to require substantial care despite 25 years of inpatient treatment. Positive features show the greater response to drugs but there are some patients who do not show this response and continue to have such phenomena on a long-term basis (Paul, 1969). Such patients are likely to be over-represented in the present sample and indeed the frequency of positive symptoms (Table II) indicates that this is so. It is reasonable to suggest that such over-representation might reduce the amount of change in positive symptoms that would be found. This study took advantage of an unusual opportunity of minimising the effects of medication and social change. The patients are not a representative sample of schizophrenics in general and it is possible that in a more typical sample the differential reversibility/stability of positive and negative features would be greater than that found here. The difference demonstrated would be consistent with the idea that the pathological processes underlying positive and negative features may not be the same. The results do not, however, indicate that either group of symptoms is of necessity irreversible. Each of the symptoms rated were sometimes resolved in this group of patients with severe and chronic schizophrenia.

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References

Angrist, B., Rotrosen, J. & Gershon, S. (1980) Positive and negative symptoms in schizophrenia—differential response to amphetamine and neuroleptics. *Psychopharmacology*, **72**, 17–19.

Bridge, T. P., Cannon, E. & Wyatt, R. J. (1978) Burned-out Schizophrenia: Evidence for Age Effects on Schizophrenic Symptomatology. *Journal of Gerontology*, **33**, 835–839.

Crow, T. J. (1980) Molecular pathology of schizophrenia: more than one disease process? *British Medical Journal*, **280**, 66–68.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

Johnstone, E. C., Crow, T. J., Frith, C. D., Carney, M. W. P. & Price, J. S. (1978) Mechanism of the antipsychotic effect in the treatment of acute schizophrenia. *Lancet*, **i**, 848–851.

Krawiecka, M., Goldberg, D. & Vaughan, M. (1977) A standardised psychiatric assessment for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

Ovchinnikov. (1968) Problems of Philosophy 9. Cited by A. V. Snezhnevsky.

Owens, D. G. C. & Johnstone, E. C. (1980) The disabilities of chronic schizophrenia: their nature and the factors contributing to their development. *British Journal of Psychiatry*, **136**, 384–395.

Paul, G. L. (1969) Chronic mental patient: current status future direction. *Psychological Bulletin*, **71**, 81–94.

Pfohl, B. & Winokur, G. (1982a) Evolution of symptoms in institutionalised hebephrenic/catatonic schizophrenics. *British Journal of Psychiatry*, **141**, 567–572.

—— & —— (1982b) Schizophrenia: Course and Outcome. In *Schizophrenia as a Brain Disease* (eds F. A. Henn, H. A. Nasrallah). Oxford: Oxford University Press.

Rifkin, A., Quitkin, F. & Klein, D. F. (1975) Akinesia. *Archives of General Psychiatry*, **32**, 672–674.

Snezhnevsky, A. V. (1968) The symptomatology clinical forms and nosology of schizophrenia. In *Modern Perspectives in World Psychiatry* (ed. J. G. Howells). Edinburgh: Oliver & Boyd.

Wing, J. K. (1978) Clinical concepts of schizophrenia. In *Schizophrenia: Towards a New Synthesis* (ed. J. K. Wing). London: Academic Press.

—— & Brown, G. W. (1970) *Institutionalism and Schizophrenia*. London: Cambridge University Press.

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Assessing the Benefits of Psychotherapy:
The Economic Approach

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Recent debate about the place of psychotherapy in the National Health Service, while emphasising the importance of economic evaluation, has concentrated almost exclusively on issues of clinical outcome. This paper argues that the debate has focussed on too narrow a definition of psychotherapy and too limited a view of the possible effects of psychotherapy, both good and bad. It reviews evidence concerning the demand and need for psychotherapy services in a number of clinical settings, and discusses the economic implications of the provision of such services. Economic studies of psychotherapy are reviewed and a conclusion drawn that it might be possible to justify the provision of psychotherapy services on economic grounds, but that considerably more research needs to be done in this area. The directions such future research might take are outlined.

Within the last two years leading articles have appeared in four influential journals expressing serious doubts about the role of psychotherapy services within the National Health Service (NHS) (Shepherd, 1984; Wilkinson, 1984; Anonymous, 1984; Gwynne Jones, 1985). These critics, and those replying in defence of psychotherapy services (Aveline, 1984; Bloch & Lambert, 1985) imply that decisions about service provision in the NHS are primarily economic decisions; nonetheless, they all fail to meet the challenge of such a view and concentrate instead on the effectiveness of psychotherapy in terms of clinical outcome alone.

The purpose of this paper is to emphasise our belief that there is a further priority than the question 'is psychotherapy effective?'; namely, the economic appraisal of psychotherapy services. We are thus not concerned solely with whether psychotherapy works, but with whether it is worth spending scarce NHS resources on its provision. We wish to establish a different framework for considering psychotherapy than clinical outcome alone, and to this end we shall consider how economic issues could be approached in psychotherapy research, and suggest a protocol for future research in this area.

In the discussion below we shall adopt a fairly wide definition of psychotherapy, based on that of the Royal Australian and New Zealand College of Psychiatrists:

> Psychotherapy is the treatment of a patient by psychological processes in the setting of a patient–therapist relationship in which the involvement of the therapist is a clearly recognised factor. No specific theoretical orientation is implied, nor are specialist behavioural treatments specifically excluded.

Economic factors in assessment

Glass & Goldberg (1977) point out that just being 'cheaper' is not a sufficient reason for preferring a course of action; 'economising' is simply cost-cutting and ignores benefits. The criterion of economic efficiency ensures that a community's resources are allocated such that the maximum benefit is derived. Alternatives are compared by reference to both costs and benefits. Such a criterion implies that a given treatment or pattern of care will not be preferred over another solely on the basis of either being less costly or more beneficial. Decisions depend on both relative costs and relative benefits.

Drummond (1980) defines four questions to which economic appraisal can address itself:

1. What is the cost of a treatment?
2. What is the benefit gained from a treatment?
3. What is the most efficient way to treat a given condition?
4. Is the treatment worthwhile?

The first two questions each refer to only half of the problem of cost–benefit analysis, and hence each can yield only half the required information.

In asking the third question, it is assumed that treatment will be given (i.e. the treatment objective is not itself in question) and hence cost-effectiveness analysis (see appendix) can be used to compare alternative forms of treatment. Examples of such studies are the comparison of methods for delivering long-term domiciliary oxygen to chronic bronchitics (Lowson *et al*, 1981) and a comparison of day care against traditional inpatient treatment for inguinal hernias and haemorrhoids (Russell *et al*, 1977).

The answer to the fourth question is often taken for granted, but logically stands before question three. The question itself can refer to different issues. For example, one could ask how much *more* (or *less*) of an already existing treatment or screening programme can be justified. Neuhauser & Lewicki (1975) assessed the costs and benefits of a series of tests for colonic cancer by measuring the additional cost for each additional true positive result. In this case the application of average rather than marginal costs would have severely underestimated the true costs.

Alternatively, *total* costs and benefits can be measured as in the study by Hagard *et al* (1976) of the introduction of a programme to screen pregnant women for spina bifida cystica in foetuses. Even when some benefits prove difficult or impossible to measure

> if it can be shown that all costs are included and that there are other positive if intangible benefits, a benefit/cost ratio of 1 . . . is a sufficient justification for arguing for the implementation of the policy (Mooney *et al*, 1980).

So how can these economic principles be applied to an evaluation of psychotherapy services in the NHS? Questions that might be considered include: How much psychotherapy can be delivered? (i.e. how large should the service be?) and how best can it be delivered? These are questions that cannot be answered simply in terms of clinical effectiveness, but only in wider economic terms. The important question for a health service with limited resources, however, is what level of resources should be channelled into psychotherapy; should we reallocate resources already utilised in the treatment of existing psychiatric morbidity into psychotherapy or allocate additional resources? In other words, is psychotherapy merely an effective, and perhaps cost-effective way of delivering psychiatric treatment to those with co-existing physical illness, or more importantly, is it a cost-beneficial way of delivering treatment?

Economic aspects of psychotherapy

When attempting to answer such questions using an economic approach there are three steps that have to be considered. Firstly it is necessary to identify the *demand* (see appendix) for services. Technically, this is a demand, usually expressed as symptoms, for subjective better health. Such symptoms are often related to psychological distress. Demand is thus related to psychiatric morbidity as it presents to health professionals, without implying that a patient is demanding any specific form of treatment. The second step is to decide whether those making the demand on services have a *need* (see appendix) for medical intervention. This is a professional decision, as is the third step—the decision as to how this need is best met. For example, is a depressive episode best treated by psychotherapy, in some other way, or by a combination of psychotherapy with other treatments?

There is clear evidence of a large demand on the health services for treatment of symptoms related to psychological distress. Such symptoms are most commonly found in general practice, or general medical and surgical settings, and are closely related to other forms of morbidity (Maguire *et al*, 1974; Goldberg, 1979; Hoeper *et al*, 1979; Goldberg & Huxley, 1980; Goldberg, 1984, 1986; Bridges & Goldberg, 1984; Orleans *et al*, 1985). There is, therefore, a high level of psychiatry morbidity in the population served by the NHS and in the main it is very difficult to separate the two subpopulations suffering from emotional disorders and physical ailments. Depression, somatic representations of psychological distress and anxiety are the three most common problems; over 80% of those with recognisable disorders have affective illnesses, or alcohol or personality problems for which psychotherapy is a recognised treatment.

The evidence is that identified psychiatric illnesses are poorly managed in medical settings, whether hospital or general practice (Marks *et al*, 1979; Goldberg & Huxley, 1980; Bridges & Goldberg, 1984; Goldberg, 1984, 1986). Patients with associated physical and psychiatric disorders have a poorer prognosis if the psychiatric disorders are left untreated (Querido, 1959; Follette & Cummings, 1967; Goldberg *et al*, 1970; Cay *et al*, 1972; Kogan *et al*, 1975; Moffic & Paykel, 1975; Whitlock & Siskind, 1979; Hawton, 1981).

Johnstone & Goldberg (1976), however, found that psychotherapy shortened the duration of such disorders. Shepherd *et al* (1966) found that psychotherapy was the treatment of choice in most cases of minor illness in general practice patients.

The benefits of psychotherapy

Yates & Newman (1980a,b) have reviewed the findings of a number of American cost-effectiveness and cost–benefit studies of psychotherapy. An important part of their review is the discussion of the 'overutilisation' of medical services. The implication is that patients presenting with physical symptoms associated with psychiatric conditions are managed in ways that focus on physical rather than psychiatric symptoms. There is no reason to suspect that this is less true in Britain than the USA, and many Health Service resources such as special investigations, medication, and inpatient or outpatient care are utilised by these patients in an attempt to alleviate their physical symptoms.

Several studies (Follette & Cummings, 1967; Goldberg *et al*, 1970; Kogan *et al*, 1975; Levitan & Kornfeld, 1981; Mumford *et al*, 1984; Borus *et al*, 1985) emphasise the 'offset effect' of decreased utilisation of services when psychiatric treatment is provided. Not only is the duration of psychiatric morbidity reduced, but physical symptoms also improve, and the utilisation of other medical services (for example radiological and pathological investigations, outpatient attendances and inpatient stay) is decreased.

Mumford *et al* (1984) and Schlesinger *et al* (1981) have reviewed the evidence for the role of psychotherapy in such patients, concluding that as well as it being an effective treatment, there are measureable changes in the use of services. The former study suggests that psychotherapeutic intervention reduces the utilisation of medical services by up to 20%; the latter confirms this finding amongst such diverse populations as alcoholics and asthmatics.

It is important to point out that an 'offset effect' should not automatically be equated with a saving in resources. Firstly, such potential cost savings are calculated at average cost; the associated marginal costs (see appendix) are often considerably lower. Secondly, the savings are not always realisable, as a large proportion of costs are fixed in the short run, for example staff salaries and capital. Any released resources could be used to treat other patient groups, or additional numbers of similar patients. Thus for example, as the length of stay is reduced, extra patients may be treated; the cost per patient is reduced, but the total cost of providing treatment for all patients (including those extra) may remain the same or even increase. In this situation, additional benefits (measured as 'cured patients' or some other index of improved health) must be set against the maintained or increased costs.

In certain situations there can be a true saving, such as treatments that reduce the consumption of drugs. A number of studies (Koch, 1979; Robson *et al*, 1984; Catalan *et al*, 1984a,b) have examined the effect of different forms of psychotherapy on the prescribing of psychotropic medication in general practice. A clinical psychologist attached to a general practice reduced the prescription of psychotropic medication by 50% (Koch, 1979) or the equivalent of 28% of the salary of a senior clinical psychologist (Robson *et al*, 1984). Catalan *et al* (1984a,b) demonstrated that counselling offered by General Practitioners (GPs) significantly reduced the prescription of psychotropic medication without an increase in the time spent with patients. Counselling may also reduce the number of GP consultations (Koch, 1979; Ashurst, 1982). The Ashurst study implies that it may be possible to pick subgroups of patients where cost offsets are more likely to accrue.

Psychotherapy may also avert hospital admission. For example, in the study of Langsley *et al* (1971) patients receiving Family Crisis Therapy were less likely to be admitted to hospital in the 18-month follow-up period and their admissions were shorter. Crisis intervention was found to be cheaper, costing on average one-sixth as much as hospitalisation at 6 months. Rosser *et al* (1983) in a study of patients crippled by chronic obstructive airways disease found that patients who were given psychotherapy had fewer hospital admissions than a control group (31% against 73%).

Similar findings are also found in the treatment of the more seriously psychiatrically ill. Psychotherapy may reduce hospitalisation in schizophrenics (Sjostrom, 1985), and 'social intervention' (including family therapy) reduces relapses (Hogarty & Goldberg, 1973; Hogarty *et al*, 1979; Leff *et al*, 1982). Lesser (1983) showed that marital therapy in a selected group of manic-depressive patients offered benefits over lithium alone.

Cost-effectiveness and cost-benefit in psychotherapy

Yates & Newman (1980b) cite only one study where "crude benefit:cost ratios have been calculated", that of Cummings (1977). However, this study in fact measured the ratio of medical costs (using the number of visits as a crude proxy for such costs) before and after receipt of psychotherapy and did not specifically include measurement of benefits.

Psychotherapy could be provided in a number of ways; it is essential that the most cost-effective is selected. Psychotherapeutic treatment in the population served by the NHS can be delivered by clinical psychologists (Koch, 1979; Robson *et al*, 1984), social workers (Shepherd *et al*, 1979), counsellors (Anderson & Hasler, 1979) and nurses (Bird *et al*, 1979; Rosser *et al*, 1983) as well as general practitioners (Catalan *et al*, 1984a,b) and psychiatrists

(Wilson & Wilson, 1985). Karon & Van den Bos (1976) report on the varying cost-effectiveness of different professional therapists, the cost of the therapist varying with discipline, salary and degree of training.

Yates & Newman (1980b) in their review point out that many methodological problems exist in the comparison of delivery by different types of therapist. Therapists within and without the health service require and receive different types of training, yet the costs of training and set-up can be high and often are not included in assessment (Rubenstein *et al*, 1978). Further, thorough evaluation of newer forms of delivery of psychotherapy, which at first sight seem less costly, (for example therapy undertaken by the client alone using manuals or cassette tapes) is limited and most studies contain little or no cost information. Studies exist which examine the method of delivery; for example the amount of client contact (Yates & Newman, 1980b), intensive inpatient vs less intensive outpatient treatment (Binner *et al*, 1973), and inpatient vs daycase treatment (Washburn *et al*, 1976). These studies appear to favour psychotherapy, both clinically and economically, yet many are inconclusive and, as Yates & Newman point out, also methodologically weak. The American Psychiatric Association's Commission on the psychotherapies (APA, 1982) found that despite a great deal of interest in cost-effectiveness and cost-benefit in recent years, most studies ignore cost-analysis, and frequently even "those studies using 'cost-effectiveness' or 'cost-benefit' in their titles present no monetised data in their method or results sections", and estimate that as much as 95% of psychotherapy research neglects the cost of treatment.

Conclusions and implications for future research

We have reviewed a wide range of evidence which suggests (a) that psychotherapy has an effect: (b) that this effect can in some circumstances be beneficial and (c) that it may, in economic terms, be possible to justify the provision of a psychotherapy service within the NHS. We have made the point that outcome measures alone are of limited value in assessing a treatment, and the well recognised difficulties in measuring outcome (Bloch, 1983) do not need reiterating here. We have also argued that an economic approach is of considerably more use when asking questions about service provision.

Most of the studies cited are of short-term therapies of a non-psychodynamic kind. This reflects the absence in the literature of economic studies of intensive psychotherapy. It is, however, clear that 'psychotherapy' as practised in the NHS has a much wider scope and application than psychoanalysis, or even than specialist psychotherapy units offering a range of treatments. Any attempt to assess psychotherapy services in the NHS must take this into account. The question 'should psychotherapy be provided by the NHS?' has entirely different connotations if the word psychotherapy is replaced by 'psychoanalysis' or 'counselling in general practice'. Thus, the question 'is psychotherapy effective?' is both fundamental, as Shepherd (1985) asserts, and also as pointless (Bloch & Lambert, 1985) as the question 'is surgery effective?" would be if taken out of context. Before asking whether it is effective, it is first necessary to define clearly what are the techniques and aims to be assessed. What is therefore implied about the assessment of psychotherapy?

1. It remains essential to evaluate the clinical effectiveness of psychotherapy. This should be done in the context of the kind of service being delivered, with the techniques and aims clearly defined. Although specialist long-term psychotherapy may be the most expensive form of psychotherapy for an individual patient, it is probable that the patients placing the greatest financial burden on the NHS as a consequence of psychiatric illness are in general medical settings (due to their large numbers) or in mainstream psychiatry (for example the large cost of treating schizophrenia; Andrews *et al*, 1985). The most urgent economic studies of psychotherapy might therefore be in these settings. The cost-offset and cost-savings studies described above provide reasonable models for similar projects in the NHS. It is necessary to define the patient group involved, the aims of treatment, and the kinds of treatment being offered. A further point is that therapist training may be a substantial cost in the delivery of psychotherapy. Ideally, these costs should be included, but an alternative would be a comparison between trained and untrained therapists working with the same patient group.

2. Secondly, there is clear need for economic markers to be included in evaluation at whatever level it is performed, whether this be studying the clinical work of one therapist or the provision of a regional specialist unit. It is by no means an easy task, but it is nonetheless important, firstly, to define all *relevant* costs (e.g. marginal not average) and secondly, to include them in any evaluation. Even if both of these are achieved there may be great difficulties in accurately measuring or putting a value on many costs, especially the 'soft' social costs such as loss of leisure time (Goldberg & Jones, 1980). Measures of cost, when taken in isolation, are also of limited use in answering questions about service evaluation,

as benefits also have to be included in the equation. Benefits may also be measured in 'soft' terms such as 'wellbeing', which are difficult to measure and ascribe an economic value. The approaches to such measurement have recently been reviewed by the Office of Health Economics (OHE, 1985) and one fruitful area in psychotherapy research could be the designing and evaluating of disability scales or health indices sensitive to the changes produced by psychotherapy.

A further dimension to the application of cost-benefit analysis should be its extension to societal costs and benefits. Benefits, such as reductions in absenteeism, sickness benefit and lost productivity may be defined as true cost savings, though not directly accruing to the providers of the psychotherapy service. Such benefits are not well documented in the studies that have been performed (Yates & Newman, 1980b; APA, 1982).

3. Thirdly, such evaluation must be accompanied by comparable evaluation of alternative forms of treatment so that they can genuinely be compared in terms of overall costs and benefits. It must be remembered that offering no treatment at all is a real alternative; all treatments should be compared with the benchmark of doing nothing. This entails special difficulties in psychotherapy research where 'placebo' or research interventions may well contain a therapeutic component (Bloch, 1983), and studies should be designed to take this into account.

4. Finally, the search must continue, not only for the most cost-beneficial treatment or combination of treatments, but also for the most cost-effective way of delivering such treatments. Even if the benefits of psychotherapy were shown to outweigh the costs, this would not necessarily justify providing a service—other treatments may produce a more favourable ratio between benefits and costs. Should a treatment be shown to be both clinically effective and have a favourable cost-benefit ratio, this does not mean it should be provided in that form. It may be possible to alter delivery of the treatment to make it more cost-effective.

This approach seems complex, but need not dissuade researchers who are prepared to grasp the issues involved and plan studies carefully. Little useful work has been done in this important area and there is much scope for further study. Small-scale studies of local service provision, at the level of single units, or even groups of therapists, can be done without the need for large input of resources, but it is important to emphasise that major studies of service provision within district or regional health authorities, or even nationally, are expensive and will require funding. We consider that economic issues are of such importance that resources for research should be allocated preferentially to studies that include an economic evaluation alongside clinical research.

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Appendix

Brief definitions of some economic terms used. (In part modified from: Culyer, 1976; Drummond, 1980; APA, 1982; Office of Health Economics, 1985).

Cost-benefit analysis (CBA) aims to set all the costs involved in a procedure against all its direct and indirect economic benefits: CBA thus sets out to answer the question "Is the benefit of this treatment greater or smaller than its cost when both are measured in economic terms?"

Cost-effectiveness analysis (CEA) aims at determining the most efficient use of resources, when either the desired outcome or the available resources are fixed: CEA attempts to answer the question "Which of these alternative treatments provides better value for money given that they both achieve the same outcome?"

Cost-utility analysis attempts to measure the degree of well-being achieved by a treatment in relation to its cost. 'Utility' as a concept represents an attempt to measure and evaluate a change in quality of life. In the case of medical treatment, utility is an increase in the quality of life (Office of Health Economics, 1985).

Demand is an indication of desire to obtain a commodity. Utility represents the satisfaction gained, so the greater the utility the greater the demand. In the NHS the commodity in demand is 'better health'. Health is viewed by health economists as a 'fundamental commodity' for which demand is generated for its own sake. The demand for health care is considered to be secondary to the demand for better health.

Need is based on the assumption that the individual consumer of health care lacks relevant information on which to make judgements about the effects of treatment options open to him. The doctor decides on the quantity and type of health care required by the patient. Thus the patient's needs involve expert assessment and decision. Needs are therefore not absolute, but relate to the end being sought, involve the values of a third party (which must be made explicit in any assessment study) and can be ranked and costed.

Marginal cost is the cost of producing an extra unit of output. Fixed costs are those incurred irrespective of the quantity of output (e.g. land, buildings), variable costs vary with the level of output (e.g. special investigations performed) and average costs are fixed plus variable costs divided by the total units of output (e.g. number of patients treated). The marginal cost relates to the average cost in that if one additional unit of output costs less than the average then overall the average costs are reduced, and if it costs more then the average costs are increased. For example if patients are added into spare capacity then the marginal costs are low, and may be much less than the average if overheads have already been included. On the other hand if new buildings or equipment are required then the marginal cost may be very much greater than the existing average cost.

Marginal analysis implies that choices in health care are not usually couched in terms of whether or not one should devote any resources at all to the care of (say) neurotic patients in general practice, but whether we should give more or less resources, and to what form of treatment. In this case it is the incremental (or marginal) costs and benefits of expansion or contraction of a treatment programme that are relevant, not the costs and benefits of the whole existing programme. If the evaluation concerns setting up an entire service then marginal costs coincide with total costs.

References

American Psychiatric Association (1982) Commission on the Psychotherapies: *Psychotherapy Research—Methodological and Efficacy Issues*. Washington, DC: American Psychiatric Association.

Anderson, S. A. & Hasler, J. C. (1979) Counselling in General Practice *Journal of the Royal College of General Practitioners*, **29**, 352–356.

Andrews, G., Goldstein, G., Lapsley, H. *et al* (1985) The economic costs of schizophrenia. *Archives of General Psychiatry*, **42**, 537–543.

Anonymous (1984) Psychotherapy: effective treatment or expensive placebo? *Lancet*, **i**, 83–84.

Ashurst, P. M. (1982) Counselling in General Practice. In *Psychiatry in General Practice*, (eds A. W. Clare & M. Lader). London: Academic Press.

Aveline, M. (1984) What price psychiatry without psychotherapy? *Lancet*, **ii**, 856–858.

Binner, P. R., Holpen, J. & Potter, A. (1973) Patients, programs and results in a comprehensive mental health centre *Journal of Consulting and Clinical Psychology*, **41**, 148–156.

Bird, J., Marks, I. M. & Lindley, P. (1979) Nurse therapists in psychiatry: developments, controversies and implications. *British Journal of Psychiatry*, **135**, 321–330.

Bloch, S. (1983) Psychotherapy. In *Recent Advances in Clinical Psychiatry*, Vol. 4. London: Churchill Livingstone.

—— & Lambert, M. J. (1985) What price psychotherapy? A rejoinder. *British Journal of Psychiatry*, **146**, 95–98.

Borus, J. F., Olendzki, M. C., Kessler, L. *et al* (1985) The "offset effect" of mental health treatment on ambulatory medical care utilisation and charges: month-by month and grouped month analyses of a five year study *Archives of General Psychiatry*, **42**, 573.

Bridges, K. & Goldberg, D. (1984) Psychiatric illness in inpatients with neurological disorders: patients' views on discussion of emotional problems with neurologists. *British Medical Journal*, **289**, 656–658.

Catalan, J., Gath, D., Edmonds, G. *et al* (1984a) The effects of non-prescribing of anxiolytics in General Practice I. Controlled evaluation of psychiatric and social outcome. *British Journal of Psychiatry*, **144**, 593–602.

——, Bond, A. *et al* (1984b) The effects of non-prescribing of anxiolytics in General Practice II. Factors associated with outcome. *British Journal of Psychiatry*, **144**, 603–610.

Cay, E. L., Vetter, N., Philip, A. E. & Dugard, P. (1972) Psychological status during recovery from an acute heart attack *Journal of Psychosomatic Research*, **16**, 425–435.

Culyer, A. J. (1976) *Need and the National Health Service: Economics and Social Choice*. York Studies in Economics. Oxford: Martin Robertson.

Cummings, N. A. (1977) Prolonged (ideal) versus short-term (realistic) psychotherapy *Professional Psychology*, **8**, 491–501.

Drummond, M. F. (1980) Principles of economic appraisal in health care. London: Oxford University Press.

Follette, W. & Cummings, N. A. (1967) Psychiatric services and medical utilisation in a prepaid health plan setting. *Medical Care*, **5**, 25–35.

Glass, N. J. & Goldberg, D. (1977) Cost-benefit analysis and the evaluation of psychiatric services. *Psychological Medicine*, **7**, 701–707.

Goldberg, D. P. (1979) Detection and assessment of emotional disorder in a primary care setting. *International Journal of Mental Health*, **8**, 30–48.

—— (1984) The recognition of psychiatric illness by non-psychiatrists. *Australian and New Zealand Journal of Psychiatry*, **18**, 128–133.

—— (1986) Mental health aspects of general health care. In *Psychosocial Perspectives in Health*. WHO Monograph (eds N. Sartorius & D. Hamburg). (in press).

—— & Jones, R. (1980) The costs and benefits of psychiatric care. In *The Social Consequences of Psychiatric Illness* (eds L. N. Robins, P. J. Clayton & J. R. Wing) New York: Brunner/Mazel.

—— & Huxley, P. (1980) *Mental Illness in the Community*. London: Tavistock.

——, Krantz, G. & Locke, B. Z. (1970) Effect of a short-term outpatient psychotherapy benefit on the utilisation of medical services in a prepaid group practice medical program. *Medical Care*, **8**, 419–428.

Gwynne Jones, H. (1985) Psychotherapy research *Journal of the Royal Society of Medicine*, **78**, 3–5.

Hagard, S., Carter, F. & Milne, R. G. (1976) Screening for spina bifida cystica. *British Journal of Preventive and Social Medicine*, **30**(1), 40–53.

Hawton, K. (1981) The long-term outcome of psychiatric morbidity detected in general medical patients. *Journal of Psychosomatic Research*, **25**, 237–243.

Hoeper, E. W., Nycz, G., Cleary, P. *et al* (1979) Estimated prevalence of RDC mental disorder in primary medical care. *International Journal of Mental Health*, **8**, 6–15.

Hogarty, G. E. & Goldberg, S. C. (1973) Collaborative study group: drug and social therapy in the aftercare of schizophrenic patients. *Archives of General Psychiatry*, **28**, 54–64.

——, Schooler, N. R., Ulrich, R. *et al* (1979) Fluphenazine and Social therapy in the aftercare of schizophrenic patients. *Archives of General Psychiatry*, **36**, 1283–1294.

Johnstone, A. & Goldberg, D. (1976) Psychiatric screening in General Practice. *Lancet*, **i**, 605–608.

Karon, B. P. & Van Den Bos, G. R. (1976) Cost/benefit analysis: psychologist versus psychiatrist for schizophrenics *Professional Psychology*, **7**, 107–111.

Koch, H. C. H. (1979) Evaluation of behaviour therapy intervention in General Practice. *Journal of the Royal College of General Practitioners*, **29**, 337–340.

Kogan, W. S., Thompson, D. J., Brown, J. R. *et al* (1975) The impact on integration of mental health services and comprehensive medical care. *Medical Care*, **13**, 934–942.

Langsley, D. G., Machotka, P. & Flomenhaft, K. (1971) Avoiding mental hospital admission: a follow-up study. *American Journal of Psychiatry*, **127**, 1391–1394.

Leff, J., Kuipers, L. & Berkowitz, R. (1982) A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, **141**, 121–134.

Lesser, A. L. (1983) Hypomania and marital conflict. *Canadian Journal of Psychiatry*, **28**, 362–366.

Levitan, S. J. & Kornfeld, D. S. (1981) Clinical costs and benefits of liaison psychiatry. *American Journal of Psychiatry*, **138**, 790–793.

Lowson, K., Drummond, M. F. & Bishop, J. (1981) Costing new services: Long-term domiciliary oxygen therapy. *Lancet*, **ii**, 1146–1149.

Maguire, G. P., Julier, D., Hawton, K. *et al* (1974) Psychiatric morbidity and referral on two medical wards. *British Medical Journal*, **i**, 268–271.

——, Goldberg, D. P., Hobson, R. F. *et al* (1984) Evaluating the teaching of a method of psychotherapy. *British Journal of Psychiatry*, **144**, 575–580.

Marks, J., Goldberg, D. & Hillier, V. (1979) Determinants of the ability of General Practitioners to detect psychiatric illness. *Psychological Medicine*, **9**, 337–353.

MOFFIC, H. & PAYKEL, E. (1975) Depression in medical in-patients. *British Journal of Psychiatry*, **126**, 346–353.

MOONEY, G. H. & DRUMMOND, M. F. (1982) Essentials of health economics (series of six articles) *British Medical Journal*, **285**, 949.

——, RUSSELL, E. M. & WEIR, R. D. (1980). *Choices for Health Care*. London: Macmillan.

MORGAN, H. G. (1984) Do minor affective disorders need medication? *British Medical Journal*, **289**, 783.

MUMFORD, E., SCHLESINGER, H. J., GLASS, G. V. *et al* (1984) A new look at evidence about reduced cost of medical utilisation following mental health treatment. *American Journal of Psychiatry*, **141**, 1145–1158.

NABARRO, J. (1984) Unrecognised psychiatric illness in medical patients. *British Medical Journal (Leader)*, **289**, 635–636.

NEUHAUSER, D. & LEWICKI, A. M. (1975) What do we gain from the sixth stool guaiac? *New England Journal of Medicine*, **293**, 226–228.

OFFICE OF HEALTH ECONOMICS (1985) *The Measurement of Health*. London: Office of Health Economics.

ORLEANS, C. T., GEORGE, L. K., HOUPT, J. L. *et al* (1985) How primary care physicians treat psychiatric disorders: A national survey of family practitioners. *American Journal of Psychiatry*, **142**, 52–57.

QUERIDO, A. (1959) Forecast and follow-up. An investigation into the clinical, social and mental factors determining the results of hospital treatment. *British Journal of Preventitive and Social Medicine*, **16**, 13–49.

ROBSON, M. H., FRANCE, R. & BLAND, M. (1984) Clinical psychologist in primary care: controlled clinical and economic evaluation. *British Medical Journal*, **288**, 1805–1808.

ROSSER, R., DENFORD, J., HESLOP, A. *et al* (1983) Breathlessness and psychiatric morbidity in chronic bronchitis and emphysema: a study of psychotherapeutic management. *Psychological Medicine*, **13**, 93–110.

RUBENSTEIN, J. S., ARMENTROUT, J. A., LEVIN, S. & HERALD, B. (1978) The parent–therapist programme: alternative care for emotionally disturbed children. *American Journal of Orthopsychiatry*, **48**, 654–662.

RUSSELL, I. T., DEVLIN, H. B., FELL, M., GLASS, N. J. & NEWELL, D. J. (1977) Day case surgery for hernias and haemorrhoids: A clinical, social and economic evaluation. *Lancet*, **i**, 844–847.

SCHLESINGER, H. J., MUMFORD, E. & GLASS, G. V. (1981) Reducing medical costs through mental health treatment: research problems and recommendations. In *Linking Health and Mental Health* (eds A. Broskowski *et al*). Beverly Hills: Sage.

SHEPHERD, M. (1984) What price psychotherapy? *British Medical Journal*, **288**, 809–810.

—— (1985) What price psychotherapy? *British Journal of Psychiatry*. **146**, 555–557.

—— *et al* (1966) *Psychiatric Illness in General Practice*. London: Oxford University Press.

——, HARWIN, B. G., DEPLA, C. *et al* (1979) Social work and the primary care of mental disorder. *Psychological Medicine*, **9**, 661–669.

SJOSTROM, R. (1985) Effects of psychotherapy in schizophrenia. *Acta Psychiatrica Scandinavica*, **7**, 513–522.

WASHBURN, S., VANICELLI, M., LONGABAUGH, R. & SCHEFF, B. J. (1976) A controlled comparison of psychiatric day treatment and in-patient hospitalisation *Journal of Consulting and Clinical Psychology*, **44**, 665–675.

WHITLOCK, F. & SISKIND, M. (1979) Depression and cancer: a follow-up study. *Psychological Medicine*, **9**, 747–752.

WILKINSON, G. (1984) Psychotherapy in the market place. *Psychological Medicine*, **14**, 23–26.

WILSON, S. & WILSON, K. (1985) Close encounters in General Practice: experiences of a psychotherapy liaison team. *British Journal of Psychiatry*, **146**, 277–281.

YATES, B. T. & NEWMAN, F. L. (1980a) Approaches to cost-effectiveness analysis and cost-benefit analysis of psychotherapy. In *Psychotherapy: Practice, Research, Policy* (ed. G. R. Vandenbos). Sage Studies in Community Mental Health 1. London: Sage.

—— & NEWMAN, F. L. (1980b) Findings of cost-effectiveness and cost-benefit analyses of psychotherapy. In *Psychotherapy: Practice, Research, Policy* (ed. G. R. Vandenbos). Sage Studies in Community Mental Health 1. London: Sage.

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A Test of the Social Support Hypothesis

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As a test of the social support hypothesis, highly anxious primiparous mothers were assigned in the post-natal stage to either a professional intervention, a lay intervention or to a control group. It was hypothesised that those receiving an active intervention (be it lay or professional assistance) would become less anxious as a consequence of a central therapeutic ingredient – social support. Improvement was assessed by measuring state anxiety levels at baseline and at 12 months, while the degree to which therapists were incorporated into the social network was assessed by the Interview Schedule for Social Interaction (ISSI), given at baseline and at 12 months. While we established that anxiety levels were significantly lowered in those receiving the professional intervention and moderately (but not significantly) lowered in those receiving the lay intervention, ISSI scores for the separate groups appeared stable over the study. Reasons are considered why ISSI scores remained unchanged while intervention groups showed a reduction in anxiety levels.

Clearly, there is now a need for rigorous studies of the effect on non-psychotic disorders of providing prosthetic social relationships, either with professionals or others Henderson *et al* (1981).

There has been increasing research interest in the proposition that deficiencies in social support place the individual at risk for psychiatric disorder and particularly depression (Brown & Harris, 1978; Henderson *et al*, 1981; Costello, 1982; Harris & Brown, 1985). In terms of mechanisms, it seems that social support acts as a buffer against adversity but absence of social support does not by itself act as a pathogen. Theoretical views differ about the relative importance of actual and perceived deficiencies in social support, and the degree to which these two components are related, as well as the degree to which need for support determines any perceived lack of support.

If perceived lack of support is a risk factor to neurotic disorders (particularly anxiety and depression) it might be expected that both the inception and maintenance of such disorders might be decreased during a time of adversity by ensuring the availability of adequate social support. We examine that proposition within a controlled, intervention study described in an earlier publication (Barnett & Parker, 1985) so that only key details will be noted here and in the next section.

A consecutive series of 630 primiparous women, married or with a live-in partner, were approached on the third or fourth day post-partum at two Sydney obstetric hospitals and asked to complete trait and state anxiety scales (Spielberger *et al*, 1970), with several exclusion criteria (e.g. poor English, seriously ill baby) being specified. All but three women accepted, and assignment was made to distinct subgroups on the basis of trait anxiety scores, with a score of 40 or more reflecting 'high anxiety', 32 or 33 'moderate anxiety', and 25 or less 'low anxiety'. As those in the high anxiety group were to be further subclassified, a larger sample of 'high anxiety' subjects ($n = 89$) was obtained than for the 'moderate anxiety' ($n = 29$) and 'low anxiety' ($n = 29$) groups, (with 11 mothers having refused, and three not completing the initial interview). Interventions were offered to two-thirds of the highly anxious subjects while the remainder acted as controls.

Thirty of the 'high anxiety' mothers were allocated to a 'lay intervention' group, which comprised the provision of experienced mothers who had volunteered to assist, and who met our requirements for a support figure who, in essence, would offer commonsense advice, general support and practical assistance. Thirty-one of the 'high anxiety' mothers were allocated to a 'professional intervention' group which comprised assistance from social workers experienced in working with mothers and children, and who would provide social support but, additionally, would consider specific anti-anxiety measures, the promotion of self-esteem and confidence, and attend to any anomalies in mother–infant, father–infant and marital interactions. Thus all those receiving active intervention were to be assigned a support-figure, which was the key treatment variable for the 'lay intervention' group and a component of the treatment package for the 'professional intervention' group. Twenty-eight mothers were allocated to the 'high anxiety' control group, receiving no intervention over the year.

Both the lay and professional support workers were provided with a suggested schedule of contact, organised so that contact (visits and by telephone) was more frequent in the early weeks but maintained over the whole year. A contact diary was maintained for each subject by the support workers. Confirmation of the social support hypothesis was tested by examining for: (a) improvement in social support scores for those receiving an active intervention, and (b) a reduction in anxiety scores in those receiving an active intervention.

Method

Members of the sample were visited in their homes by the same rater (B.B.) within 3 weeks of hospital discharge. In arranging the visits, the mothers were informed that the researchers were interested in the difficulties mothers faced with new babies and that the initial visit by the rater was to explore such issues. Following a semi-structured interview a number of personality questionnaires were administered, together with the measure of social support—the Interview Schedule for Social Interaction (ISSI). The ISSI is a measure of the perceived availability and adequacy of support from intimates and the wider social network (Henderson *et al*, 1981), and generated four scale scores for each of our subjects: 'availability of attachment' (AVAT), 'adequacy of attachment' (ADAT), 'availability of social integration' (AVSI) and 'adequacy of social integration' (ADSI). The interviewer remained unaware of each subject's group allocation until the interview and baseline ratings were completed when an envelope listing the assignment was opened. Those in the 'high anxiety' group had previously been randomly allocated to the three subgroups: lay or professional intervention or control.

Subjects were asked to complete and return questionnaires at 3, 6 and 9 months, and to attend a 1-year follow-up interview, with trait and state anxiety scores being measured on each occasion. At the second interview at 12 months the ISSI was readministered to generate a second set of social support scores.

Results

Of the 147 mothers entered in the study, 139 attended the 12-month follow-up, so ISSI details are limited to the 139 subjects completing the interview schedule on both occasions (Table I). The test–retest coefficients vary somewhat across the subgroups, with the coefficients for the total sample being 0.55 (AVAT), 0.57 (ADAT), 0.72 (AVSI) and 0.63 (ADSI), perhaps suggesting somewhat greater stability in the social integration subscale scores. We next examined whether the 'high anxiety' subjects (comprising those in groups 1, 2, and 3 and therefore irrespective of treatment status) differed from the remaining subjects allocated to the 'moderate anxiety' and 'low anxiety' group, both at baseline and at follow-up. At baseline, the 'high anxiety' subjects reported significantly less adequacy of attachment (ADAT) and significantly less availability of social integration (AVSI). At follow-up, the 'high anxiety' subjects differed significantly from the remaining subjects on all four ISSI measures, in the direction of reduced availability and less adequacy in their attachments. Thus, we established that anxious subjects returned ISSI scores suggesting certain deficiencies in social support, before and after our intervention.

The next two broad sets of analyses sought to determine whether scores on the ISSI differed for the high anxiety subjects receiving intervention as against the high anxiety controls who received no intervention. At baseline, there were no significant differences, establishing that random allocation to treatment or control group was not associated with differences in attachment scores. At follow-up, the professional intervention and control groups differed on one scale, with the professional intervention group reporting greater adequacy of attachment (ADAT). As both these groups, however, showed a rather similar reduction in scores on this measure (5% and 8% respectively), the follow-up difference would appear to be more a reflection of a pre-existing trend, which was just short of significance at baseline, reaching significance at follow-up. When the lay intervention and control groups were compared at follow-up, no significant differences were demonstrated on the ISSI scales.

In addition, a series of planned contrast analyses, using the PSY programme (Hewson & Bird, 1977), examined for changes in ISSI scores over the 12 months. Such analyses were important to control for group differences at baseline and to avoid the risks of multiple inference associated with the large number of tests used in the initial analysis. In undertaking the analyses of variance with repeated measures, the technique accepted distinction between two types of factors, namely, independent groups (i.e. treatment and control) and repeated measurement (i.e. baseline and follow-up scores). The Bonferroni procedure (see Hall & Bird, 1985) was used to control the Type 1 error rate for each family of contrasts at 0.05 and we calculated that a critical F ratio of 8.53 would be required to establish significant differences between groups and for interactions of groups with repeat measurements. Firstly, we examined for group differences on ISSI scale scores averaged over time. When all high anxiety subjects were contrasted with remaining subjects, there were significant differences for ADAT ($F=13.46$) and AVSI ($F=11.84$), but not for ADSI ($F=4.46$) or AVAT ($F=3.01$). No differences were found on any scale when lay intervention and high anxiety controls, or when professional intervention and high anxiety controls were contrasted on scores averaged over time. Secondly, interaction contrasts examined whether there were significant changes between groups over time, examining in part the ADAT difference noted in the previous paragraph. When all high anxiety and remaining subjects were contrasted there were no significant differences in change for AVAT ($F=1.42$), ADAT ($F=2.17$), AVSI ($F=0.39$) or ADSI ($F=2.51$). When lay intervention and high anxiety control scores were contrasted, no significant differences in improvement were found over time for AVAT

TABLE I

ISSI Scale	Assessment	*High anxiety*			*Moderate anxiety*	*Low anxiety*	*Comparison of groups*		
		Lay intervention 1	*Professional intervention 2*	*Control 3*	*4*	*5*	*All high anxiety groups compared with remaining groups*	*Lay intervention compared with anxiety controls*	*Professional intervention compared with anxiety controls*
AVAT	Baseline	7.3	7.2	7.4	7.4	7.6	$t=0.96$	$t=0.28$	$t=0.78$
	Follow-up	6.8	7.4	7.3	7.5	7.6	$t=2.12$*	$t=1.60$	$t=0.40$
	Reliability	0.61	0.77	0.45	0.21	0.21			
ADAT	Baseline	8.4	9.8	8.4	10.1	9.9	$t=2.52$**	$t=0.07$	$t=1.92$
	Follow-up	7.6	9.3	7.7	9.9	10.0	$t=3.86$***	$t=0.08$	$t=2.33$**
	Reliability	0.57	0.67	0.68	0.21	0.21			
AVSI	Baseline	6.6	7.0	7.2	8.4	8.8	$t=3.40$***	$t=0.78$	$t=0.35$
	Follow-up	6.0	6.4	6.7	7.2	8.4	$t=2.93$***	$t=0.98$	$t=0.37$
	Reliability	0.55	0.87	0.74	0.64	0.64			
ADSI	Baseline	12.4	12.4	12.6	13.3	13.0	$t=1.38$	$t=0.20$	$t=0.26$
	Follow-up	11.1	12.5	11.7	13.0	13.5	$t=2.39$**	$t=0.57$	$t=0.84$
	Reliability	0.70	0.56	0.71	0.68	0.68			

*$P<0.05$ **$P<0.025$ ***$P<0.001$

($F=1.81$), ADAT ($F=2.17$), AVSI ($F=0.47$) or ADSI ($F=0.26$). Similarly, when professional intervention and high anxiety control scores were contrasted over time, no significant differences were found for AVAT ($F=1.60$), ADAT ($F=0.23$), AVSI ($F=0.004$) and ADSI ($F=1.76$).

One item (Q.48), which contributes to the AVSI scale, appeared particularly pertinent to our inquiry in that it questions whether, apart from those at home, there are people available "(in this town) to whom you can turn in times of difficulty? Someone you can see fairly easily whom you could trust and whom you could expect real help from in times of trouble". Only three high anxiety subjects (two professional intervention and one control) were unable to nominate any such individual before the study, and two (both lay intervention subjects) at 12 months. Over the interval 29% of the lay intervention, 34% of the professional intervention and 30% of the controls reported an increased number of such people being available, while a decrease was reported by 50% of the lay intervention, 41% of the professional intervention and 41% of the controls. This analysis suggests that the provision of a social support figure to two of the groups was not associated with a differential increase in such nominated support-figures by the subjects.

In a previous report (Barnett & Parker, 1985), we examined whether intervention was successful in reducing anxiety. When mean scores at baseline and at 12 month follow-up were compared, both intervention groups had shown a significant reduction in state anxiety levels while the change in the control group was non-significant. In a more comprehensive analysis, assessing state anxiety levels every 3 months over the 12-month study, and using planned contrast analyses again, we established that only professional intervention had a significant effect with state anxiety levels being reduced by 19%, while lay intervention was associated with a non-significant reduction of 12%, as against the 3% reduction in state anxiety levels in the controls.

Thus, while the variable of importance—high anxiety—was decreased in those receiving intervention in comparison to the controls, such an improvement was not associated with any change in the four measures of both close and diffuse social relationships.

Discussion

Our study was designed principally to assess further the proposition that a high level of maternal anxiety is a key risk factor to women in the pre-, peri- and post-natal period. In a non-clinical group of primiparous mothers, subjects were assigned to subgroups on the basis of trait anxiety scores. In a separate publication (Barnett & Parker, 1986) we report the greater morbidity of the highly anxious subgroup. At baseline they scored as more neurotic and depressed (suggesting broad dysfunction not merely expressed as anxiety), they reported more life events in the previous year and they had more negative interpretations of their pregnancy, the hospital experience and the hospital staff. They had more delivery complications, while their babies were more likely to be dysmature and slow to suckle. In the post-partum period these mothers were more depressed, had more concerns about their baby, themselves and their marriage, and were less confident about coping and their parenting capacities. Thus we argue that this subgroup of mothers had rather diffuse psychosocial morbidity and were an appropriate sample to test the effectiveness of an intervention programme with an emphasis on social support.

Two interventions were designed for highly anxious mothers, using respectively professional and lay therapists, with the key objective of lowering anxiety levels in the mothers and with the secondary objective of improving mother–child interaction. Theoretically, the social support component was integral to both interventions, being the key component to the lay therapist group while, for the professional therapist group, the orientation was more to specific therapeutic strategies. In practice, at the initial visit by the interviewer each mother received a general explanation before the results of the random assignment was known, with the interviewer suggesting that having a baby makes everyone anxious and that ways to help mothers were being explored. Mothers were told that chance would decide whether "an experienced mother with children of her own" or a social worker "accustomed to working with mothers and children" would be the "helper" for up to 1 year for those assigned a "support person", or whether they would simply be followed over the 1 year. While a clear statement was therefore made about the potential for allocation of a support-figure, we did not assess in any detail the mothers' perception of the support-figure other than that they rated the therapists in a universally complimentary way (Barnett & Parker, 1985).

While we demonstrated that state anxiety levels were distinctly reduced in the intervention groups, as against the non-significant reduction in the controls, we find no support for the view that improvement might have been a consequence of the therapist becoming a key social supportive figure. There are three broad possible explanations for these findings. Firstly, that the highly anxious subjects had optimal social networks, whether actual or perceived, disallowing improvement. Secondly, that the therapists did effect change via a social support mechanism but that the ISSI did not measure that contribution. Thirdly, that the therapists were effective for reasons that were independent of any social support contribution, and a psychotherapeutic effect must be considered when the greater success of the professionals was demonstrated.

If the therapists were incorporated into the social network of the subjects, we would expect their presence to be recorded both in the AVAT scale (assessing the presence of others who know the individual very well, who allow demands for emotional support, and with whom private feelings may be shared) and in the AVSI scale which assesses the extent of, and interaction with less intimate contacts in the social network. At baseline, the three high anxiety groups scored 7.2 or more on the AVAT scale which suggests, since the maximum score of the AVAT scale is 8.0, that the group scored as having almost optimal 'availability of attachment' before the trial. This could be an accurate assessment of the sample or a reflection of the scale tending to generate skewed scores. As the community sample surveyed by Henderson *et al* (1981) returned a mean score of 6.9 on the AVAT scale, we suspect that high scores are relatively easy to generate, perhaps because subjects find it too threatening to acknowledge certain deficiencies in their intimate relationships and/or that they judge their need for an intimate as being met. Our inclusion criteria (i.e. women with husbands or live-in partners) would favour such an effect. The ISSI has a subsidiary scale (NONAT), contributed to by questions from the AVAT scale, in which the respondent indicates that *no* intimate figure is available for those particular situations and she is *not* dissatisfied with this. Only seven subjects (5%) made such a response. The highest possible NONAT score is 7 and only two of the seven subjects scored 2, the others scoring 1, out of seven. Whether as a consequence of such sample characteristics and/or some incapacity of the AVAT scale to overcome defensive responses by subjects, such a ceiling phenomenon in our sample suggested that improvement on the AVAT scale could hardly be expected. In fact, the opposite occurred, with AVAT scores for all high anxiety subjects being lower than for remaining subjects at 12 months, although such a difference was not observed at baseline. As AVAT scores for the separate subgroups of high anxiety subjects did not change over the trial, any involvement by the therapists (be they lay or professional) was not observable in AVAT scores.

No such ceiling phenomenon was evident for our sample on the AVSI scale, with the high anxiety subgroups having mean scores of 6.6–7.2, and the maximum score for the scale being 16. These mean AVSI scores for our high anxiety subgroups are lower than the mean of 9.4 established for married community subjects by Henderson *et al* (1981). Further, we established significantly lower AVSI scores for this group in comparison to the less anxious subjects in our total sample, at baseline, follow-up and averaged over time, suggesting that our highly anxious subjects had impaired availability of social integration, with the controlled design of the study establishing that this was not merely a consequence of pregnancy status in the preceding year. Several items of the AVSI scale (e.g. respondent being asked to nominate if there are people available who may be turned to in times of difficulty or trouble, or who can be talked to if the respondent is irritated by things) clearly have the potential to incorporate a social support figure. Thus, if a social support intervention is effective as a consequence of the therapists providing social support, we would expect higher follow-up AVSI scores in subjects receiving intervention, particularly when decrements were demonstrated at baseline. However, not only did AVSI scores fail to increase for high anxiety subjects receiving intervention, they showed a general trend to decrease over the trial. As this phenomenon was relatively constant across all groups, including the moderate and low anxiety subjects, it would appear to be a reflection of the narrowing of the social world of the new mother as well as the insensitivity of the scale to the social support figure. When we specifically examined whether the number of support-figures outside the home and available in times of trouble (Q.48) had increased in those receiving intervention, we found no evidence to support incorporation of the lay or professional therapist on this AVSI item. Again, mean scores for the high anxiety subjects of 8–9 on the ADAT scale (the allowed maximum being 12) and an overall mean of 12.4 on the ADSI scale (the allowed maximum being 17) suggest that the possibility that the subjects already had relatively supportive social networks is unlikely. Not only did the therapists fail to be incorporated in the availability scales, there is no suggestion that they contributed to the adequacy measures, with adequacy scores tending to decrease rather than increase over the trial for those in the high anxiety subgroups. Clearly, these findings might not hold up if intervention were offered to a socially isolated group.

The possibilities that the therapists were effective as social support figures (but that improved social support was not detected by the ISSI) or that they were effective independent of any social support role (e.g. by psychotherapeutic effects) must be explored indirectly. At baseline assessment, the high anxiety subjects reported significantly less adequacy of attachment and a trend for less adequacy of social integration compared to less anxious subjects, differences which were significant in both scales at follow-up. We interpret this as a phenomenon associated with high anxiety, in that such subjects have at least a strong negative response bias or 'plaintive set' (Henderson *et al*, 1981) in perceiving and reporting dissatisfaction with their attachment figures, or that their social network is indeed deficient. Henderson *et al* (1981) found that higher neuroticism scores were linked with lower availability and adequacy ISSI scores, and suggested that neuroticism might affect the individual's capacity to form and maintain social relationships, as well as cause the individual to view his social relationships as inadequate. It might be anticipated that if anxiety levels were reduced in such subjects, they would be less inclined to judge social support negatively and that their social relationships would be improved. Although we established (Barnett & Parker, 1985) that subjects were universally complimentary about their assigned therapist and that contact hours were quite extensive, our failure to demonstrate improved ISSI scores in those receiving an intervention suggests an insensitivity of the ISSI to the therapists' contribution, whether as an intrinsic weakness of the measure in such situations or the subjects not 'perceiving' the therapist as relevant to the questions. The general worsening in perceived adequacy of attachment over the study across most groups suggests that the ISSI was sufficiently sensitive, however, to detect the narrowed social world of the new mother.

High test–retest coefficients are generally interpreted as suggesting stability in scores. Henderson *et al* (1981) reported 12-month correlation coefficients of around 0.75 for the ISSI scales but, after correcting for measurement error, found a very high ("almost constant") correlation for the 'availability' measures, but somewhat greater fluctuations for the 'adequacy' measures. They suggested that the availability measures have "characteristics normally attributed to personality traits . . . as though each person had his own 'natural' level of availability". Our 12-month test–retest data (with a mean coefficient of 0.62) suggest less stability than found by Henderson's group, which might be anticipated for a sample facing social adaptation such as ours, and perhaps argues against any view that the ISSI measures fixed attributes which might be insensitive to imposed changes in the social network.

Such issues bear on the validity of the ISSI measure. As a test of the concurrent validity of the measure, Henderson *et al* (1981) correlated scores by 114 community subjects with scores returned by informants about the respondents. The mean correlation coefficients were 0.42 (AVAT) 1, 0.39 (ADAT), 0.59 (AVSI) and 0.26 (ADSI). These correlations, and particularly those for the adequacy scales, are not impressive and suggest relatively discordant views between the subjects and informants. Low agreement between subjects' and informants' reports is not an uncommon finding, while high agreement may not necessarily constitute validation, particularly if reports are not truly independent (Platt, 1980). Low agreement does suggest, however, that ISSI scores may be very much influenced by subjective judgments with their attendant biases. If so, it might be expected that the provision of assistance to a group of highly anxious women would have changed their perception of social support for the better. Either the provision of a therapeutic social support figure was of little moment to the subjects or the ISSI is insensitive to such changes.

Finally, it must be conceded that even if deficiencies in social support induce psychosocial morbidity, it cannot be argued that the provision of social support will necessarily relieve that morbidity, for causal and reparative pathways are not necessarily linked. Failure to demonstrate improved ISSI scores in a sample of highly anxious women whose anxiety level was reduced cannot be interpreted as the ideal challenge to those aspects of the social support hypothesis relating to aetiology and causal mechanisms. As Henderson *et al* (1981) observed, aetiological and outcome studies require different hypotheses and research strategies. Further, in arguing for intervention trials, Brugha *et al* (1982) noted that successful intervention (to deficiencies in social relationships) does not guarantee proof of a causal link. Our failure to demonstrate successful modifications of ISSI scores does not, by contrast, then disallow a causal process linking levels of social support with the inception, maintenance and resolution of neurotic disorder. These several explanations must be conceded and assessed in further longitudinal studies.

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References

BARNETT, B. & PARKER, G. (1985) Professional and non-professional intervention for highly anxious primiparous mothers. *British Journal of Psychiatry*, **146**, 287–293.

——, —— (1986) Possible determinants, correlates and consequences of high levels of anxiety in primiparous mothers. *Psychological Medicine*, **16**, 177–185.

BROWN, G. W. & HARRIS, T. (1978) *Social Origins of Depression*. London: Tavistock.

BRUGHA, T., CONROY, R., WALSH, N., DELANEY, W., O'HANLON, J., DONDERO, E., DALY, L., HICKEY, N. & BOURKE, G. (1982) Social networks, attachments and support in minor affective disorders: a replication. *British Journal of Psychiatry*, **141**, 249–255.

COSTELLO, C. G. (1982) Social factors associated with depression: a retrospective community study. *Psychological Medicine*, **12**, 329–339.

HALL, W. & BIRD, K. (1985) The problem of multiple inference in psychiatric research. *Australian & New Zealand Journal of Psychiatry*, **19**, 265–274.

HARRIS, T. & BROWN, G. W. (1985) Interpreting data in aetiological studies of affective disorder: some pitfalls and ambiguities. *British Journal of Psychiatry*, **147**, 5–15.

HENDERSON, S., BYRNE, D. G. & DUNCAN-JONES, P. (1981) *Neurosis and the Social Environment*. Sydney: Academic Press.

HEWSON, D. & BIRD, K. (1977) *Psy Packaged Program*. Typescript obtainable from the School of Psychology, University of New South Wales, Sydney.

PLATT, S. (1980) On establishing the validity of "objective" data: Can we rely on cross-interview agreement? *Psychological Medicine*, **10**, 573–581.

SPIELBERGER, C. D., GORSUCH, R. L. & LUSHENE, R. E. (1970) Manual for the State-Trait Anxiety Inventory (Self-Evaluation Questionnaire). Palo Alto, California: Consulting Psychologists Press.

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Suicide Risk Factors:

A Comparison of Suicide Attempters with Suicide Completers

K. MICHEL

Fifty cases of attempted suicide were compared with 50 cases of completed suicide on variables reported to measure suicide risk: the seriousness of the attempt, and the circumstances in which the act occurred discriminated best. Depressive symptoms were more frequent in the suicide group and in the serious attempters' group than in the non-serious attempters' group.

Because those who attempt suicide differ in certain typical characteristics from those who complete it, the assessment of risk can be based on the assumption that the closer a person gets to killing himself, the more he resembles a person who has killed himself. The variables found to be useful in measuring suicide risk include personal and clinical characteristics (Pallis & Sainsbury, 1976; Pallis *et al*, 1982) and certain aspects of the way a suicidal act is carried out (Beck *et al*, 1974; Pallis & Pierce, 1979).

Several scales for estimating suicide risk have been developed. However, it is difficult to measure their predictive value: large numbers of patients need to be followed up in order to include at least a few cases of suicide, and important characteristics are likely to change in the course of time. Furthermore, special care given to patients thought to be at risk may invalidate an original prediction. Therefore, validation of risk factors and scales has been based on comparisons between those who attempt and those who complete suicide. Such comparisons, however, face certain methodological problems. For example, information about suicide attempters can be obtained from the individual himself, whereas information about people who commit suicide can only be collected from relatives, friends or case records. Moreover, scales have not so far been validated in populations of different socio-economic structure and culture.

The aim of the present study was to test in a Swiss urban population findings based on a population in the South of England (Pallis *et al*, 1982). Characteristics of low-intent attempters, high-intent attempters (intent is measured by scales based on the *circumstances* of the suicidal act) and suicide completers were compared. We expected the findings of Pallis *et al* to be valid also for a Swiss population, although the two countries differ in terms of social structure and suicide rate (Switzerland's rate is three times higher than that of England & Wales). To overcome some of the methodological difficulties described above, we collected all the data from relatives and friends, i.e. from similar sources for all the three groups, and used a semi-structured interviewing method.

Method

Study samples

The study samples consisted of 50 cases of attempted and 50 of completed suicide. Both samples were drawn from a catchment area covering Bern and the close surroundings, within a time span of 15 months (October 1982 to January 1984).

Suicide attempters

Patients referred to the psychiatrist on duty by the casualty department of the University Hospital of Bern (comprising about 90% of all admitted suicide attempters) were routinely reported to the research psychiatrist. Those who had clearly carried out a deliberate and potentially life-threatening act were accepted for the study, and relatives or friends were contacted. In 83 cases (62%) a contact person was either untraceable or refused to co-operate, and to complete 50 interviews it was necessary to collect 133 cases.

Suicide completers

The Institute of Forensic Medicine of the University of Bern, to which all suicides from the catchment area are referred, reported names of the cases "in which a cause of death other than suicide could reasonably be excluded". For each case permission to contact the relatives by letter had to be sought from the Coroner. The letter had been approved by an ethical committee, and it had been agreed that the relatives were not to be contacted until 4 weeks after the suicide. In 31 cases (38%) relatives refused an interview or could not be traced, and 81 cases had to be collected until 50 interviews were completed.

The interviews in the attempters' group took place between 2 days and 6 weeks (mean 21 days) and in the suicide group between 4 and 12 weeks (mean 53 days) after the event.

Data collection

A set of 37 written questions was asked in a semi-structured way. Questions covered the following areas:

Medical seriousness of the method used in the suicide act: if it represented an actual danger to life it was classified as medically serious, otherwise as not serious.

Circumstances of the suicidal act: questions covered the items of the Modified Intent Scale (Pallis *et al*, 1984), and answers were rated according to the definitions and guidelines provided by the authors.

Personal characteristics: the six items of the Risk Scale (Pallis *et al*, 1982, 1984) were applied (see Table II).

Clinical characteristics: 12 questions covering symptoms of depression and signs of alcohol and drug dependence taken from the Present State Examination (PSE) (Wing *et al*, 1974) were asked and rated accordingly (the author is trained in the use of the PSE).

Items concerning circumstances of the suicidal act and mental state which could not be rated with confidence were classified as 'unclear'; this possibility was applied relatively freely, as it seemed preferable to have no information rather than unreliable information.

The reliability of the ratings

The reliability was measured by comparing ratings of the research psychiatrist with ratings made by an independent observer psychiatrist (B.R.) from 13 randomly tape-recorded interviews. In the Modified Intent Scale there was disagreement in 15% and in the 12 PSE questions in 8% of the ratings.

In nine cases of attempted suicide, the attempter was separately interviewed in addition to the contact person. The two sets of ratings differed as follows: in the Intent Scale there was disagreement in 17% and in the clinical symptoms in 23%; in 79% of the discordant ratings the attempters reported higher levels of intent and more depressive symptoms than the relatives.

Results

There were no significant differences in sex or age (below/above 45) distribution between the 214 reported cases and the two study samples. There were more women than men in the attempters' group (30 vs 20) and more men in the suicide group (16 vs 34). Most suicide attempters and most male suicide cases were under 45 years; most female suicide cases were over 45. The mean ages in the study samples for attempters were: men 36.2, women 36.9; and for suicide completers: men 41.2, women 50.1 years.

Methods used for the suicide act

The most frequent method used for suicide attempts was overdosing: 74%. In the suicide group the most frequent method was shooting (32%), followed by jumping from high places (20%); only 18% used drugs: 22 suicide attempts were, independently from other information, classified as 'medically not serious'. Examples of serious methods were deliberate poisoning from car exhaust, shooting in the chest, jumping from the fourth floor, overdose with 70 tablets of bromazepam 3 mg. Examples of not serious methods were overdose with 20 tablets of flunitrazepam 2 mg, superficial cuts on the wrists and threatening to jump from the window while being held back by the family.

Circumstances of the suicidal act

The seven items of the Modified Intent Scale give a range of scores from 0 to 14. The mean score in the attempters'

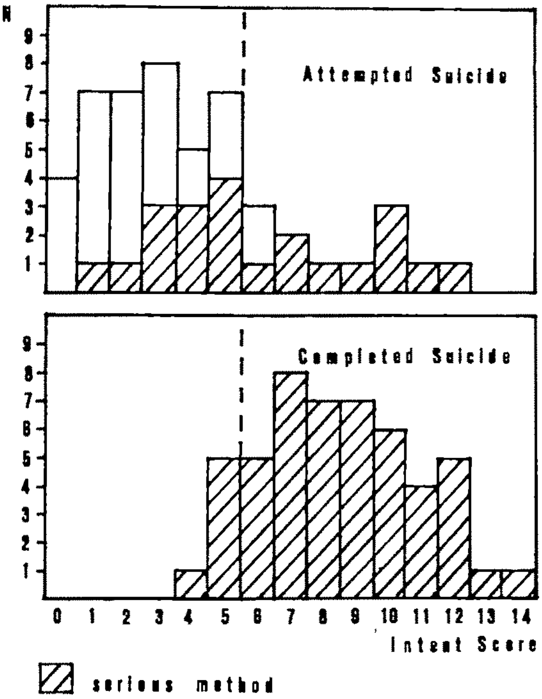

Fig 1. Modified Intent Scale: distribution of scores for the two study samples (difference between the two groups, Mann-Whitney U-test, two-tailed $P<0.001$) and their relationship to seriousness of method. Optimal cut-off line between scores 5 and 6.

group was 4.0 and in the completers' group 8.5. The distribution of scores is shown in Fig. 1.

The optimal 'cut-off line' based on the minimum percentage overlap of attempters and suicide completers lies between the scores 5 and 6 and correctly discriminates 82% of all the cases. This cut-off line was used to divide attempters into a 'low-intent' (38 cases) and a 'high-intent' (12 cases) group. Figure 1 also illustrates the association of intent with medical seriousness. In 31.5% of low-intent and 83.2% of high-intent attempters, the method was classified as serious. The mean intent score was 2.4 for the 'not serious' group, and 6.1 for the 'serious' group (the difference in the Mann-Whitney U-test was highly significant: $P<0.001$).

The distribution of scores showed statistically significant differences in six of the seven items: 1) proximity of others during act, 2) timing, 3) precautions against discovery, 4) acting to gain help during or after act, 5) final act in anticipation of death, 6) degree of planning for the act. Item 7 (presence or absence of suicide note) did not reach statistical significance.

Personal characteristics

Figure 2 illustrates the distribution of scores on the six-item Risk Scale.

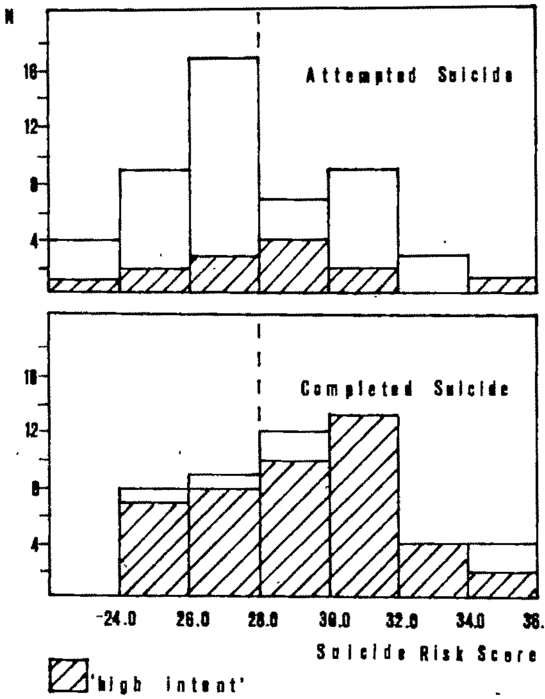

FIG. 2. Six-item Risk Scale: distribution of scores for attempted suicides and complete suicides (difference between the two groups: Mann-Whitney U-test, two-tailed $P<0.01$) and their relationship to low-intent and high-intent. Optimal cut-off point: 28.0.

TABLE I

Items of Short Risk Scale: frequency distribution of 'risk answers'

	Attempted suicide				*Completed suicide*	
	Low intent (n=38)		*High intent (n=12)*		*(n=50)*	
	N	%	N	%	N	%
Age over 45	12	31	2	16	20	40
[1]Male sex	12	31	7	58	34	68
Unemployed	6	15	1	8	15	30
Living alone	12	31	3	25	13	26
[1]Social class I+II	7	18	4	33	25	50
Suicide communications in the last year	24	63	7	58	40	80

1. Items in which the differences in the distribution of yes/no answers were significant in the χ^2-test.

The optimal cut-off line of 28.0 correctly discriminated the two samples in 64%. There was no correlation between scores on the 'Risk' Scale and the Intent Scale—the mean scores for low and high-intent attempters did not differ.

The 'risk items' appear in Table I. 'Risk answers' were found more often in the suicide group, but the differences were significant in only two of the six items.

Clinical characteristics

The ten symptoms of depression give a range of scores from 0 to 20. The man scores were 5.7 for low-intent attempters, 8.5 for high-intent attempters and 8.0 for suicide completers. Alcohol and drug abuse was associated with low depression scores, so were schizophrenia, personality disorder and, interestingly, interpersonal conflicts in adolescence (three suicide cases).

TABLE II

Symptoms of depression; percentages of score '2' answers (ratings 0–2)

	Attempted suicide		*Completed suicide*
	Low intent	*High intent*	
Symptom	%	%	%
Delayed sleep	23	36	20
[1]Early waking	3	20	55
Worrying	17	67	44
Depressed mood	26	54	30
Poor concentration	3	0	14
[1]Loss of interest	6	42	35
Social withdrawal	9	33	26
Self depreciation	14	18	26
Weight loss	9	25	31
Anergia and retardation	12	33	20

1. Significant differences in the χ^2-test.

Table II shows the percentages of score '2' answers. Differences between the three groups were significant in two of the ten symptoms.

Discussion

The aim of the study was to test the findings of Pallis *et al* in a Swiss urban population: although the study is mainly a replication of previous work, it introduces new methodological elements. To avoid the problem of having to compare data from different sources collected in different ways, information on suicide attempters as well as completers was collected by interviewing relatives or close friends of the individuals in a semi-structured way. Both study samples came from the same catchment area and were consecutively collected within the same time-span. The main problem we encountered was the refusal of a high proportion of contact persons to be interviewed. However, because we had case notes of all the cases accepted for the study, we were able to compare them with the sex- and age-distribution of the study samples, and we found no significant differences in the χ^2 test. We can therefore assume that our samples were fairly representative of the total group.

Firstly, we found a strong association between medical seriousness and scores of the Intent Scale. People who made careful preparations to kill themselves used methods presenting a high threat to life. This association has been described by Pallis & Sainsbury (1976).

Secondly, the Modified Intent Scale discriminated well between suicide attempts and completed suicides: based on the optimal cut-off point the accuracy was 82%. The results are similar to those of Beck *et al* (1974) who compared suicide attempters and completers on eight items of the Suicidal Intent Scale.

Thirdly, although the differences of the scores of the six-item Risk Scale were significant in the Mann-Whitney U-test and clearly followed the same tendency as the samples of Pallis *et al*, they were somewhat less than expected. It is interesting, however, that the cut-off point of 28.0, which accurately discriminated 64% of the cases, lies between the cut-off points of Pallis and Sainsbury's original group (28.5) and their validation group (27.5). We could not find an association between personal characteristics and intent scores as had been reported by Pallis & Sainsbury. Because the data of this scale are of an objective nature (age, sex, work status, etc.) the differences in the results cannot be due to differences in the method of data collection. They could, however, be due to differences in the sampling method (our suicide attempters had all been sent to hospital, most of them by General Practitioners and therefore represented the rather more serious cases; Pallis & Sainsbury had found medically serious attempts in 15% of their cases, while in our study this proportion was 44%), or they could reflect differences between populations in the South of England and Switzerland.

Fourthly, suicide attempters and completers differed in their amount of depressive pathology. High-intent attempters had as much pathology as suicide completers, which supports the original hypothesis that individuals at risk resemble persons who have committed suicide. It would be interesting to know which symptoms of depression are of special importance in the assessment of suicide risk. In our samples only two of the symptoms reached differences large enough to be statistically significant, while Pallis *et al* had described larger differences in more symptoms. Measuring depressive pathology 'second hand' in an objective and comparable way is difficult (the PSE-questions we used were designed to evaluate the symptoms and signs of a person being interviewed directly), and although we know that a diagnosis of depression is frequent in individuals who commit suicide (Robins, 1981; Barraclough *et al*, 1974), to our knowledge depressive symptoms in suicidal persons have not so far been measured and compared in a standardised way.

The results have clear implications on the assessment of suicide risk in Switzerland: the seriousness of method, the Intent Scale, the six-item Risk Scale and the amount of depressive pathology all can differentiate, in an urban population, between those suicide attempters who are not at risk and those who do accomplish suicide and should therefore be applied in clinical practice. The findings are similar to those in the South of England (and in Philadelphia), although Switzerland differs in many ways. The Swiss population has few social problems and little unemployment (under 1%) but has one of the highest suicide rates (27/100 000 in 1982) in the Western world. Also, the proportions of the various methods used for suicide are different in Switzerland: more than half of our suicide completers used firearms or jumped from high places: methods that are rare in England and Wales. A comparison of suicide statistics (for England and Wales: McClure, 1984) e.g. shows that our study samples have a relatively high proportion of cases from the younger age-group. Such factors might be a reason why the six-item Risk Scale was less powerful in discriminating between suicide attempts and completed suicides than expected from the findings of others. A comparison of social factors associated with suicidal behaviour might shed more light on the driving forces of suicidal behaviour and thus lead to more effective prevention.

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References

BARRACLOUGH, B. M., BUNCH, J., NELSON, B. & SAINSBURY, P. (1974) A hundred cases of suicide: clinical aspects. *British Journal of Psychiatry*, **125**, 355–372.

BECK, R. W., MORRIS, J. B., BECK, A. T. (1974) Cross-Validation of the suicidal intent scale. *Psychological Reports*, **34**, 445–446.

BECK, A. T., SCHUYTER, D. & HERMAN, I. (1974) Development of suicidal intent scales. In *The Prediction of Suicide*, Bowie, Maryland: The Charles Press.

MCCLURE, G. M. (1984) Trends in suicide rate for England and Wales 1975–80. *British Journal of Psychiatry*, **144**, 119–126.

PALLIS, D. J. & SAINSBURY, P. (1976) The value of assessing intent in attempted suicides. *Psychological Medicine*, **6**, 487–492.

—— & PIERCE, D. W. (1979) Recognising the suicidal overdose. *Journal of the Royal Society of Medicine*, **72**, 565–571.

——, BARRACLOUGH, B. M., LEVEY, A. B., JENKINS, J. S. & SAINSBURY, P. (1982) Estimating suicide risk among attempted suicides: I. The development of new clinical scales. *British Journal of Psychiatry*, 141, 37–44.

——, GIBBONS, J. S. & PIERCE, D. W. (1984) Estimating suicide risk among attempted suicides: II. Efficiency of predictive scales after the attempt. *British Journal of Psychiatry*, **144**, 139–148.

ROBINS, E. (1981) *The final months. A study of the lives of 134 persons who committed suicide.* New York: Oxford University Press.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms.* London: Cambridge University Press.

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The Impact of a Liaison Psychiatry Service on Patterns of Referral in a General Hospital

A. BROWN and A. F. COOPER

A retrospective study of psychiatric referrals from a general hospital inpatient population was carried out for three separate years, 1973, 1976 and 1979. Reorganisation of the liaison service to the responsibility of one consultant team was associated with changes in referral rate and disposal. In particular, there was a significant increase in the referral rate of patients from medical and surgical wards who were not involved in acts of deliberate self-harm.

The association between physical illness and psychiatric morbidity is now well recognised (Lloyd, 1977) but the extent to which psychiatrists are involved in the management of inpatients in general hospitals is very variable. Historically, the uncertain relationship between general medicine and psychiatry in Britain has passed through several phases. Traditional psychosomatic medicine had little impact on clinical psychiatry earlier this century (Gath & Mayou, 1983). In the 1950s, psychosomatic concepts proposed by Alexander (1952) and others attracted attention for a time but soon fell into disfavour on account of claims which were over-inclusive and theories which were difficult to substantiate (Lipowski, 1977; Gomez, 1981). However, in the 1960s the trend appeared to change following the development of consultation–liaison psychiatry, particularly in the United States (Lloyd, 1980). With a few exceptions (Crisp, 1968; McLeod & Walton, 1969) the concept of liaison psychiatry in its fullest sense i.e. the psychiatrist's integral involvement in the management of general hospital patients, has never developed as fully in the UK as in the USA, in contrast to the advances that have taken place in the management of deliberate self-harm.

In most general hospitals, psychiatrists offer a regular consultative service to their non-psychiatric colleagues but the types of service offered and the referral rates vary greatly (Brooks & Walton, 1981). Referral rates are consistently higher in the USA than in the UK (Lloyd, 1980) but in neither case are they commensurate with the levels of psychiatric morbidity found amongst general hospital in-patients (Maguire *et al*, 1974). Analysis of the type of referrals made to liaison services has been carried out at a number of centres (Fleminger & Mallett, 1962; Anstee, 1972; Fava & Pavan, 1980) but few recent studies have examined the trends in referral patterns over time. In October 1974, a liaison psychiatry service, staffed by one consultant team, was introduced to the general wards of the Victoria Infirmary, Glasgow. In the course of the next few years, a rise in the annual referral rate was noted (Fig. 1) and this paper sets out to examine the nature of the changing pattern of referrals to the liaison service.

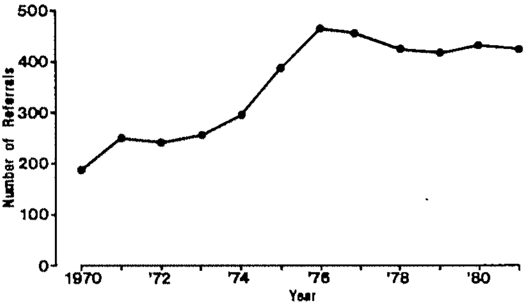

FIG. 1. Annual referral rate of Victoria Infirmary inpatients to the liaison psychiatry service.

Description

The Victoria Infirmary is a 500-bed general hospital, without a psychiatric in-patient unit which, during the time of the study, served the population of Greater Glasgow Health Board, South Eastern District, approximately 200,000 people. Prior to October, 1974 patients referred by physicians and surgeons were seen in the wards by psychiatrists from the associated psychiatric hospital, Leverndale Hospital, situated 6 miles away. Consultations usually took place on a twice-weekly rota basis during the course of outpatient sessions held at the Victoria Infirmary. Since that date, referrals from the general wards have been the responsibility of one of the Leverndale clinical teams consisting of the consultant and one trainee psychiatrist who have devoted three sessions per week to liaison psychiatry.

Initially, all patients were seen in the medical or surgical wards, but in 1976, accommodation adjacent to the medical wards became available for psychiatric staff to interview patients in privacy. Referrals are made by letter and an appointment system is operated. Patients are brought to the Department of Psychiatry by ward staff and the management of each is discussed with appropriate medical or nursing staff before the end of each liaison session.

Method

A retrospective case-record study was carried out on all patients referred for a psychiatric opinion in 1973 (the last full year prior to the introduction of the liaison service); and also those referred during 1976 and 1979. Information was obtained from both general hospital and psychiatric hospital case records and from contact sheets used by the local psychiatric service. The following data were recorded: age and sex of referred patient, source of referral, previous contact with the psychiatric services, presence of self-poisoning or injury, primary psychiatric diagnosis and disposal.

Information relating to admissions to the Victoria Infirmary and to Leverndale Hospital and the number of referrals to the psychiatric outpatient clinics in the catchment area was obtained from the Information Services Division, Greater Glasgow Health Board. The significance of differences between the samples was assessed by χ^2 analysis and in view of the large number of patients and tests of significance involved, $P<0.01$ was taken as the criterion for statistical significance (Altman *et al*, 1983).

Results

Demographic features

The mean ages of the referred patients were 35.7 years (1973), 40.6 years (1976), 40.9 years (1979). Consistently the highest number of referrals was in the age-range 20–29 years and in each year, approximately 75% of the patients were between 20 and 59 years of age. The ratio of male to female patients remained fairly constant (1/1.85 in 1973 and 1976; and 1/1.91 in 1979).

Diagnostic categories

Diagnoses were made initially according to the ICD-9 classification (WHO, 1978) and were then grouped into nine categories. The results relative to the primary diagnoses are shown in Table I.

In several categories, e.g. alcohol and drug addiction and organic brain disorders, the percentage of patients diagnosed remained approximately constant, whereas in others, e.g. personality disorder and situational disturbance, there was a significant change. This probably represents a variation in the fashion and priorities for psychiatric diagnosis rather than an alteration in the patient population. This conclusion is supported by observing the results obtained when the percentages in diagnostic categories 1 and 2 were combined and those in categories 3 and 4 were also combined (Table I). Comparable totals for each of the three years studied were obtained for each category. Thus, 41–45% of patients were diagnosed as personality disorder and situational disturbance, and 28–32% as suffering from depressive illnesses.

A separate analysis was carried out for the group of patients who had not harmed themselves (50 in 1973; 127 in 1976; 112 in 1979). Whilst there was a decline in the frequency of a primary diagnosis of personality disorder over the years similar to that in the total group, the diagnosis of depressive illness increased from 4 (8%) in 1973 to 34 (27%) in 1976 and 31 (28%) in 1979. The diagnosis of neuroses (other than depressive) also increased from 4% in 1973 to 8% in both 1976 and 1979 whilst that of alcohol and drug-related problems decreased from 26% in 1973 to

TABLE I

Primary psychiatric diagnoses (approximate % in parenthesis)

Diagnosis	*1973*	*1976*	*1979*	*P(d.f. = 2)*
1. Personality disorder	56 (22)	53 (11)	20 (5)	<0.001
2. Situational disturbance	47 (19)	147 (32)	166 (40)	<0.001
3. Neurotic depression	66 (26)	80 (17)	68 (17)	<0.01
4. Endogenous depression	14 (6)	68 (15)	48 (11)	<0.01
5. Alcohol and drug addiction	34 (13)	56 (12)	42 (10)	NS
6. Organic brain disease	16 (6)	31 (7)	26 (6)	NS
7. Other psychosis	12 (5)	16 (3)	14 (3)	NS
8. Other neurosis	5 (2)	11 (2)	18 (4)	NS
9. No diagnosis	2 (1)	6 (1)	18 (4)	<0.01
Total	252	468	420	

TABLE II
Disposal (% in parenthesis)

	1973	*1976*	*1979*
GP only	93 (37)	130 (38)	180 (43)
Outpatient follow-up	111 (44)	195 (41)	131 (31)
Inpatient care (Leverndale)	33 (13)	64 (14)	50 (12)
Other	15 (6)	79 (17)	59 (14)

21% in 1976 and 12% in 1979. The diagnosis of organic psychosyndromes in this group was high but relatively constant at around 25%.

Disposal

Table II shows the pattern of disposal of patients following consultation. The percentage followed up as outpatients fell significantly during the period 1976–1979 ($P<0.001$) and this was entirely due to a reduction in the follow-up rate of the deliberate self-harm group ($P<0.001$). The proportion of patients transferred to psychiatric inpatient care, however, remained constant. A consistent majority in the latter group were patients previously known to the psychiatric service (55–60%) and most had harmed themselves (1973:79%; 1976:64%; 1979/76%).

TABLE III
Number, source and type of referral (% in parenthesis)

Referrals	*1973*	*1976*	*1979*
Total referrals	252	468	420
Referred by physicians	227 (90)	418 (89)	365 (87)
Deliberate self-harm (DSH)	202 (80)	341 (73)	308 (73)
Non-DSH	50 (20)	127 (27)	112 (27)

Referral rates

Table III shows the total number of referrals to liaison psychiatry in each of the three years studied, the number referred by physicians and the numbers of cases of deliberate self-harm (DSH) and non-DSH patients. The vast majority of patients referred were cases of DSH and from physicians, but an increased proportion of non-DSH referrals was noted after 1973.

Table IV shows the referral rates of various categories of admission i.e. the number of patients referred divided by the total number of admissions to the Victoria Infirmary in each category. There was a significant increase in the referral rates of all admissions to the Victoria Infirmary, of admissions to the medical wards and of non-DSH patients. However, there was no significant change in the referral rates of patients indulging in self-harm despite the fact that the commitment to liaison psychiatry was increased by one session per week.

TABLE IV
Referral rate of various categories of admission to Victoria Infirmary

	1973	*1976*	*1979*	*P (d.f. = 2)*
Total referrals	252	468	420	
Total admissions	13594	14811	14991	
Referral rate	1.85%	3.16%	2.80%	<0.001
Referrals from physicians	227	418	365	
Total medical admissions	3388	3686	3922	
Referral rate	6.7%	11.3%	9.3%	<0.001
DSH referrals	202	341	308	
DSH admissions	347	536	551	
Referral rate	58.2%	63.6%	55.9%	NS
Non-DSH referrals	50	127	112	
Non-DSH admissions	13247	14275	14440	
Referral rate	0.38%	0.89%	0.78%	<0.001

The possibility that the rise in referral rate was merely a reflection of rising psychiatric morbidity in the community was investigated by comparing total liaison referrals and non-DSH liaison referrals in each year with the number of referrals made to the associated district psychiatric outpatient clinics and inpatient psychiatric wards. The results in Table V show that the increase in both total and non-DSH liaison referrals was significantly different from these other indicators of psychiatric morbidity.

TABLE V
Comparison of liaison referrals, new outpatient psychiatric referrals and psychiatric admissions

	1973	*1976*	*1979*
A. Total liaison referrals	252	468	420
B. Non-DSH liaison referrals	50	127	112
C. Total new outpatient psychiatric referrals	959	1148	692
D. Total psychiatric admissions	973	1074	1086

For comparisons A vs C, A vs D, B vs C and B vs D: $P<0.001$, d.f. = 2.

Discussion

Reorganisation of the liaison psychiatry service to a general hospital provided a natural experiment in which the impact of the work of a single liaison team on patterns of referral could be investigated. There appears to be only one recent study in which changes in the pattern of referrals to a liaison service over time have been studied. Lipowski & Wolston (1981) compared two consecutive populations of 1000 patients referred to a liaison service during an 8-year period and emphasised the stability of the populations in relation to demographic characteristics, diagnostic categories and patterns of referral and intervention.

Despite the widely differing proportions of patients indulging in deliberate self-harm (8% of Lipowski & Wolston's patients and 73–80% in the present study) the demographic characteristics of the two populations are similar; 75% of the present sample being between 20 and 59 years in each year examined, compared with 70% in the other study. The ratio of female to male patients is also slightly higher in the present series, 1.85–1.91:1 compared to 1.6–1.8:1. These results are similar to the findings of Fleminger & Mallett (1962) and Fava & Pavan (1980).

Comparison of the distribution of the psychiatric diagnoses in the two studies is limited by the use of different classifications. For example, the first two categories are not specifically represented in Lipowski & Wolston's paper. The proportion of depressive disorders found in our patients is markedly less (28–32% compared to 43–50%) as is the percentage of patients with organic brain syndromes (6% compared to 14–15%). The latter finding may be a reflection of the much higher proportion of patients presenting with deliberate self-harm in the present study since, when these are excluded, the percentage of organic disorders rises to 22–28%.

The very high percentage of referrals from physicians in this study (87–90%) is probably related to the high proportion of cases of deliberate self-harm. However, when these are excluded, the referral rate from physicians (58–66%) is still higher than from other specialties and is similar to the 60% reported by Lipowski & Wolston (1981).

The change in pattern of disposal, particularly the significant reduction in the follow-up rate of those who had harmed themselves, is probably the result of a conscious alteration in the management of these patients. There is increasing awareness that even intensive follow-up does not significantly alter repetition rates (Chowdhury *et al*, 1973; Gibbons *et al*, 1978; Hawton *et al*, 1981), and that a reduction in repetition rates is more likely to occur when psychiatric assessment and advice takes place before discharge from hospital (Greer & Bagley, 1971; Kennedy, 1972). The high default rate of these patients at subsequent outpatient appointments is well recognised (Morgan *et al*, 1976; Blake and Mitchell, 1978). In contrast, however, a relatively constant proportion of patients require psychiatric inpatient care for further assessment and treatment.

Referral rates to the liaison service are comparable to those elsewhere in the UK (Lloyd, 1980) but these rates are much lower than the reported rates of psychiatric morbidity found in medical wards (Maguire *et al*, 1974). There are several possible reasons for this. Estimates of psychiatric morbidity may be excessive, the psychiatric services may be inadequate, psychiatric disorder may not be recognised or, if recognised, may be treated by the non-psychiatric specialist or general practitioner (Mezey & Kellett, 1971). Several authors have commented on the resistance of other specialists to referring their patients to psychiatrists. The problem is often attributed to the patient but may in fact reflect reluctance on the part of the referring specialists themselves (Steinberg *et al*, 1980).

Unlike the increase in referral rates observed in another British study (Anstee, 1972), the rise in referral rate over the period of this investigation is not attributable to increased referral of cases of deliberate self-harm. It is noteworthy that the maximum rate of referral in this group was only 63.6%, despite government recommendations (Ministry of Health, 1961) that all such cases should have formal psychiatric assessment.

The most striking finding in this study is the significant increase in the referral rate of medical and surgical inpatients who are not involved in acts of deliberate self-harm. This does not appear to be associated with a rise in psychiatric morbidity generally, as measured by the rate of admissions to the local psychiatric hospital and outpatient referrals to the psychiatric service at the time. It is encouraging to note that the rise in referral rate is associated particularly with an increase in the diagnosis of potentially treatable conditions such as depressive illness and neurotic disorders.

The results suggest that the changes observed in the pattern of referrals are related to the interaction between the reorganised liaison service and the referring departments. The identification of a specific liaison team may lead to improved interprofessional relationships, an increased awareness of the importance of psychological factors in physical illness and a more well defined and realistic expectation of the service provided. Crisp (1968) reported a referral rate of 10% of inpatients from two medical wards to his liaison service and emphasised that personal contact with the physicians was a major factor determining the extent of referrals of patients for psychiatric opinion. Other authors have attributed changes in both rate (Torem *et al*, 1979) and specificity (Davis & Nelson, 1980) of referral to liaison services, to increased contact with the referring departments. The present study demonstrates that even a modest reorganisation of the liaison psychiatry services to a general hospital can be associated with a significant increase in the referral rate of patients in medical and surgical wards.

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References

Alexander, F. (1952) *Psychosomatic Medicine; Its Principles and Applications*. London: Allen and Unwin.

Altman, D. G., Gore, S. M., Gardner, M. J. & Pocock, S. J. (1983) Statistical guidelines for contributors to medical journal. *British Medical Journal*, **286**, 1489–1492.

Anstee, B. H. (1972) The pattern of psychiatric referrals in a general hospital. *British Journal of Psychiatry*, **120**, 631–634.

Blake, D. R. & Mitchell, J. R. A. (1978) Self-poisoning: management of patients in Nottingham, 1976. *British Medical Journal*, **1**, 1032–1035.

Brooks, P. & Walton, H. J. (1981) Liaison psychiatry in Scotland. *Health Bulletin*, **39**, 218–227.

Chowdhury, N., Hicks, R. & Kreitman, N. (1973) Evaluation of an aftercare service for parasuicide (attempted suicide) patients. *Social Psychiatry*, **8**, 67–81.

Crisp, A. H. (1968) The role of the psychiatrist in the general hospital. *Postgraduate Medical Journal*, **44**, 267–276.

Davies, D. S. & Nelson, J. K. N. (1980) Referrals to psychiatric liaison nurses: changes in characteristics over a limited time period. *General Hospital Psychiatry*, **2**, 41–45.

Fava, G. A. & Pavan, L. (1980) Consultation psychiatry in an Italian general hospital. *General Hospital Psychiatry*, **2**, 35–40.

Fleminger, J. J. & Mallett, B. L. (1962) Psychiatric referrals from medical and surgical wards. *Journal of Mental Science*, **108**, 183–190.

Gath, D. & Mayou, R. (1983) Consultation—liaison psychiatry in the United Kingdom. *Advances in Psychosomatic Medicine*, **11**, 109–126.

Gibbons, J. S., Butler, J., Urwin, P. & Gibbons, J. L. (1978) Evaluation of a social work service for self-poisoning patients. *British Journal of Psychiatry*, **133**, 111–118.

Gomez, J. (1981) Liaison psychiatry. *British Journal of Hospital Medicine*, **26**, 242–246.

Greer, S. & Bagley, C. (1971) Effect of psychiatric intervention in attempted suicide: a controlled study. *British Medical Journal*, **i**, 310–312.

Hawton, K. E., Bancroft, J., Catalan, J., Kingston, B., Stedeford, A. & Welch, N. (1981) Domiciliary and outpatient treatment of self-poisoning patients by medical and non-medical staff. *Psychological Medicine*, **11**, 169–177.

Kennedy, P. (1972) Efficacy of a regional poisoning treatment centre in preventing further suicidal behaviour. *British Medical Journal*, **4**, 255–257.

Lipowski, Z. J. (1977) Psychosomatic medicine in the seventies: an overview. *American Journal of Psychiatry*, **134**, 233–244.

—— & Wolston, E. J. (1981) Liaison psychiatry: Referral patterns and their stability over time. *American Journal of Psychiatry*, **138**, 1608–1611.

Lloyd, G. G. (1977) Psychological reactions to physical illness. *British Journal of Hospital Medicine*, **18**, 352–358.

—— (1980) Liaison psychiatry from a British perspective. *General Hospital Psychiatry*, **2**, 46–51.

McLeod, J. G. & Walton, H. J. (1969) Liaison between physicians and psychiatrists in a teaching hospital. *Lancet*, **ii**, 789–792.

Maguire, G. P., Julier, D. L., Hawton, K. E. & Bancroft, J. H. J. (1974) Psychiatric morbidity and referral on two general medical wards. *British Medical Journal*, **1**, 268–270.

Mezey, A. G. & Kellet, J. M. (1971) Reasons against referral to the psychiatrist. *Postgraduate Medical Journal*, **47**, 315–319.

Ministry Of Health (1961) *Circular HM* (61) 94. London: HMSO.

Morgan, H. G., Barton, J., Pottle, S., Pocock, H. & Burns-Cox, C. J. (1976) Deliberate self-harm: a follow-up study of 279 patients. *British Journal of Psychiatry*, **128**, 361–368.

Steinberg, H., Torem, M. & Saravay, S. M. (1980) An analysis of physician resistance to psychiatric consultations. *Archives of General Psychiatry*, **37**, 1007–1012.

Torem, M., Saravay, S. M. & Steinberg, H. (1979) Psychiatric liaison: benefits of an "active" approach. *Psychosomatics*, **20**, 604–611.

World Health Organisation (1978) Mental disorders: Glossary and guide to their classification in accordance with the *Ninth Revision of the International Classification of Diseases*. Geneva: WHO.

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The Persistence of Prescribing Habits:
A Survey and Follow-up of Prescribing to Chronic Hospital In-patients

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This study describes a survey of prescribing to psychiatric patients on long-stay wards, together with a follow-up survey carried out after a 2-year period. Our findings show that there had been no significant improvement in polypharmacy and overprescribing of psychotropic drugs over that period, 1983–1985. Guidelines for improvement are suggested.

The theoretical aspects of rational pharmacotherapy have been discussed extensively (e.g. Ayd, 1973). Recently, with increasing interest in medical audit, there have been a number of surveys of prescribing in psychiatric settings. The first British study, carried out in an Oxford hospital, was reported by Michel & Kolakowska (1981) and highlighted the problems of polypharmacy and illogical prescribing.

A further study of patients in Malvern (Morgan & Gopalaswamy, 1984) found similar results, but criticised the earlier survey by attempting to show that illogical polypharmacy could be explained when patients' diagnoses and individual needs were reviewed in conjunction with the survey. However, although this approach may partly explain the reasons for polypharmacy, it does not lessen the value of identifying extensive overprescribing (Holden, 1984). The findings of the Oxford survey have been replicated by Edwards & Kumar (1984) in a survey carried out on a similar patient population in Birmingham. This present study was carried out to see if similar results would be found in a survey of prescribing to a dissimilar population of psychiatric patients, and to investigate the stability of prescribing over a 2-year period in the light of the above research and publicity.

Method

The population studied was of patients on long-stay wards of a large psychiatric hospital, serving the catchment area of the whole of Newcastle. This hospital is a designated teaching hospital for undergraduate and postgraduate psychiatric teaching. The wards involved have high bed occupancy (in the region of 98%) and low turnover (10–15 new admissions per quarter with dementia, and 2–4 with 'chronic functional' illnesses) in common with such wards elsewhere in the country. All patients on these wards are reviewed fully, at least at 6-monthly intervals, as well as receiving more frequent medical attention when necessary.

The patients were divided into two groups, those suffering from 'chronic functional disorders' (predominantly schizophrenia and affective disorders) and those with 'organic mental disorder' (predominantly middle- and late-stage of dementia of mixed types). Division into these groups was done by reference to the patient's casenote diagnosis.

The initial survey was carried out within a single day in March 1983, and the follow-up survey within a single day in July 1985. Ward staff were not forewarned prior to either study. All medication listed in the British National Formulary within the section on 'Drugs acting on the central nervous system' was recorded as psychotropic medication. 'As-required' medication was only included if it had been administered within the immediately preceding 24 hours.

During the 2-year period between the surveys, the results of the initial survey were published briefly (Holden, 1984). In addition, further attention was drawn to the problems of prescribing by the publication of the surveys mentioned above. However, no attempt was made to approach care-workers directly over the individual patients' findings.

Results of the two surveys were compared using the chi-squared test. However, it should be made clear that this test depends on the assumption of the independence of the observations in the two surveys, and as there were some patients and therapists common to both, this is not strictly the case. However, since no other simple statistical test is more applicable to this situation, and the chi-squared test underestimates the likelihood of findings being significant in this case, its use was felt to be acceptable.

Results

In the 1983 survey, there were 139 patients in the chronic functional disorders ('chronic') group — 60 males (age range 37–85, mean = 67.9, s.d. = 9.2) and 79 females (age range 34–85, mean = 63.1, s.d. = 12.1) — and 174 patients in the organic mental disorder ('organic') group — 33 males (age range 53–89, mean = 73.2, s.d. = 9.0) and 141 females (age range 46–100, mean = 80.3, s.d. = 7.9). In the second survey, there were 156 patients in the 'chronic' group — 61

TABLE I
Psychotropic prescribing

	'Chronic' patients[1]				*'Organic' patients*[2]			
	1983		*1985*		*1983*		*1985*	
Patients receiving	*n* *139*	*%* *100*	*n* *156*	*%* *100*	*n* *174*	*%* *100*	*n* *137*	*%* *100*
Total no. of drugs: 0	12	9	24	15	68	39	55	40
1	24	17	27	17	62	36	46	34
2	39	28	39	25	37	21	19	14
3	26	19	33	21	7	4	12	9
4	27	19	19	12	0	0	5	4
5	5	4	10	6	0	0	0	0
6	6	4	4	3	0	0	0	0
Neuroleptics	117	84	119	76	51	29	47	34
Anticholinergic agents	78	56	67	43	15	9	12	9
Hypnotic	29	21	39	25	58	33	42	31
Anxiolytics	18	13	12	8	3	2	2	1
Antidepressives	17	12	15	10	13	7	8	6
Lithium	6	4	5	3	0	0	0	0
Anticonvulsants	12	9	15	10	6	3	11	8
Sinemet	6	4	6	4	5	3	7	5
On neuroleptics	*117*	*100*	*119*	*100*	*51*	*100*	*47*	*100*
Depot alone	9	8	7	6	0	0	0	0
Depot and oral	29	25	36	30	0	0	4	9
No. of neuroleptics: 1	72	62	68	57	48	94	40	85
2	30	26	35	29	3	6	7	15
3	13	11	14	12	0	0	0	0
4	2	2	2	2	0	0	0	0
With anticholinergic	74	63	65	55	9	18	11	23
On hypnotics	*29*	*100*	*39*	*100*	*58*	*100*	*42*	*100*
With nocturnal neuroleptic or antidepressive	25	86	31	79	24	41	17	40

1. χ^2 showed no significant differences for all comparisons 1983–1985.
2. χ^2 showed no significant differences for all comparisons 1983–1985 except total drugs prescribed increased, $P<0.05$.

males (age range 52–88, mean = 69.6, s.d. = 7.2) and 95 females (age range 31–105, mean = 67.7, s.d. = 14.7)—and 137 patients in the 'organic' group—42 males (age range 27–90, mean = 69.5, s.d. = 12.6) and 95 females (age range 52–100, mean = 79.9, s.d. = 11.0). Almost all of the patients were white Caucasian (>99%).

In the 'chronic' group (Table I), 9% of patients were receiving no psychotropic medication in 1983 and 15% in 1985, with 46% receiving three or more psychotropic drugs in 1983 and 42% in 1985. In 1983, 84% of patients were receiving neuroleptics and 76% in 1985. Of those prescribed neuroleptics, 39% were receiving two or more and 25% both depot and oral forms in 1983, increasing to 43% and 30% respectively in 1985. In the 1983 survey, 56% of the whole group and 63% of those who were receiving neuroleptics were also receiving anticholinergic anti-parkinsonian agents, in contrast with 43% and 55% in the 1985 survey. Those prescribed multiple neuroleptic medication were significantly more likely to receive anticholinergics ($\chi^2 = 20.4$, d.f. = 2, $P<0.001$).

Hypnotic drugs were prescribed to 21% of the 'chronic' patients in 1983 and to 25% in 1985. These were combined with an antidepressant or a neuroleptic in 86% in 1983 and 79% in 1985. The percentages of patients receiving other groups of psychotropic drugs in both surveys were low. There were no significant changes between 1983 and 1985 in any statistical comparison.

Drugs for physical problems were given to 60% of 'chronic' patients, the most common being laxatives (23% of all patients), diuretics (15%), vitamins (13%) and topical preparations (13%). Drugs were administered four times per day in 39%, three times in 40%, twice in 15% and once in 4%.

In the 'organic' group (Table I), 39% were receiving no psychotropic medication and only 4% three or more psychotropics in the first survey, these figures being 40% and 13% in the second. Approximately one-third of patients were receiving neuroleptics in both surveys and most of these were given orally.

Only 6% received two or more neuroleptics in the 1983 survey, increasing to 15% in 1985. In both surveys, 9% of patients received anticholinergic antiparkinsonian agents and these drugs were given to approximately 20% of those receiving neuroleptics. Just over 30% of patients received hypnotics, and of these 40% were also given a nocturnal neuroleptic or antidepressant. Only small numbers of patients were receiving other groups of psychotropic drugs, and no patients received lithium therapy. Total psychotropic prescribing for the 'organic' group increased significantly from 1983 to 1985 ($\chi^2 = 9.39$, d.f. $= 3$, $P < 0.05$), but there was no other statistically significant change.

Drugs for physical ailments were given to 73% of 'organic' patients, the most common being laxatives (45% of all patients), diuretics (25%), topical preparations (13%), vitamins and nutrients (11%) and analgesics (10%). Drugs were administered four times per day in 11%, three times in 30%, twice in 22% and once in 28%.

Comparing the prescribing patterns of both psychotropic and physical medication for the two groups in the 1983 survey, 'organic' patients were prescribed significantly fewer drugs — 82% being on three or less drugs compared with 48% of 'chronic' patients ($\chi^2 = 40.7$, $P < 0.001$). Significantly fewer 'organic' patients (41%) were administered drugs three or more times per day compared with 'chronic' patients (73%) ($\chi^2 = 30.58$, $P < 0.001$) and drugs for physical illness were prescribed to the 'organic' group more frequently than to the chronic group ($\chi^2 = 5.5$, $P < 0.05$), especially diuretics ($\chi^2 = 4.87$, $P < 0.05$), laxatives ($\chi^2 = 16.12$, $P < 0.001$) and anticonvulsants ($\chi^2 = 3.85$, $P < 0.05$).

The 'chronic' group were given significantly more neuroleptic medication ($\chi^2 = 93.5$, $P < 0.001$) and anticholinergic antiparkinsonian ($\chi^2 = 83.46$, $P < 0.001$) than did the 'organic' group. Of patients on neuroleptics only, 20% were receiving them once daily and 60% more frequently than twice daily. The most commonly chosen neuroleptic in both groups was thioridazine. More than one-half of both groups were given anticholinergic antiparkinsonian drugs as a three-times-daily dosage. The 'organic' group received more hypnotic medication than did the 'chronic' group ($\chi^2 = 5.99$, $P < 0.05$) but fewer minor tranquillisers ($\chi^2 = 15.56$, $P < 0.001$). Similar differences were found for the 1985 survey.

Discussion

Extensive polypharmacy was found in both the surveys, mainly in the 'chronic' group, but also in the 'organic' group. In the 'chronic' group, of the three-quarters of patients who were receiving neuroleptics, over one-third were given more than one type, and over 10% were receiving three or more types of neuroleptic. Between 20 and 30% of these 'chronic' patients received both depot and oral forms. The prescribing of two or more neuroleptics to stable chronic patients is hard to justify (Morgan & Cheadle, 1974; Kimball & Glass, 1983).

Anticholinergic antiparkinsonian agents (anticholinergics) were prescribed to a large number of patients, especially those who were receiving multiple neuroleptics. In a study of the withdrawal of these drugs in patients on neuroleptics in this same hospital, McClelland *et al* 1974 found that all but 8% of patients could have anticholinergics discontinued after the acute phase. Similar findings have been found by Klett & Caffey (1972), but Rifkin *et al* (1978) found that over 50% of patients withdrawn from procyclidine developed extra-pyramidal symptoms. Perris *et al* (1979) have suggested that the prescription of combined neuroleptics and anticholinergics increases the likelihood of tardive dyskinesia, and Kane & Smith (1982), whilst concluding that there was currently insufficient clinical data to confirm this, still emphasised the importance of their judicious use.

About one-quarter of 'chronic' patients were receiving nocturnal hypnotics as were approximately 30% of 'organic' patients. In addition, 80% of these 'chronic' patients and 40% of these 'organic' patients were prescribed sedative antidepressants and neuroleptics. The use of nocturnal doses of the latter drugs might allow minimisation of the use of hypnotics.

Large percentages of both groups of patients received drugs for physical ailments. They were given to 60% of 'chronic' patients and 73% of 'organic' patients. The high percentage of patients receiving laxatives probably reflects the constipating anticholinergic side-effects of the polypharmacy. Particularly unusual was the high level of regular topical medication given to patients. The fact that more than 10% of patients were on vitamins or other nutrients makes one wonder about the nutritional value of diet provided in an NHS hospital.

This survey showed that drugs were administered an excessive number of times per day to the majority of patients, and that drugs with long half-lives were given in divided dosages, often three or four times daily. Although patients may show fewer side-effects from divided doses of drugs when initially prescribed, Ayd (1973) suggested that it is illogical to prescribe drugs with a long half-life in divided doses after this initial phase. This would apply to neuroleptics, antidepressants and anticholinergics. Administering the majority of drugs once daily would save nursing time, decrease the chance of errors in administration and improve patients' willingness to comply.

The differences found between the 'chronic' and 'organic' groups were as expected. The 'organic' patients were receiving significantly fewer psychotropic drugs including fewer neuroleptics, but significantly more drugs for physical ailments. All these findings are consistent with the 'organic' group being less psychotic and having more age-related illness and deterioration.

In the population studied, patient turnover was low. The decrease in numbers in the 'organic' group in 1985 was due to the closure of approximately 50 beds for financial and administrative reasons, and may have resulted in the 1985 patients being generally more dependent. This might explain the increase in medication levels in this group. However, there remains the fact that there was no evidence of reduction of levels of prescribing over the period of study. Long-stay hospital inpatients are likely to be on stable medication for long periods, as there is undoubtedly a tendency not to change a patient's medication unless there is a change in his mental state, and administration of drugs by nurses allows medical staff to be more confident of compliance when multiple drugs are prescribed. As the effect of rapid changes in medication is minimised, the results cannot be explained as artifacts caused by transition between acute and maintenance medication. The lack of change, in spite of the publication of some of the results of the first survey (Holden, 1984) and the findings of other drug surveys, is somewhat surprising and highlights the difficulties of medical audit and resistance to change.

Comparison with the previous British studies of Michel & Kolakowska (1981) in Oxford and of Edwards & Kumar (1984) in Birmingham is difficult, as both studies looked at all patients, including day-patients. Compared with this study of long-stay patients, they showed more patients receiving depot neuroleptics, and higher levels of prescribing of anti-depressants and lithium. However, this is expected from the nature of the present sample—chronic inpatients dominated by the diagnoses of chronic schizophrenia and dementia.

Morgan & Gopalaswamy (1984) have criticised the approach used in this and other studies which does not look at the individual patient's diagnosis and needs for medication. However, we would maintain that surveys of prescribing indicate areas that can be improved. In the light of this survey, we would like to see more drugs being given as once-daily doses, with fewer drug administrations by nurses per day, and decreased chronic prescribing of anticholinergics with neuroleptics. Chronic patients should be given only one neuroleptic, either by depot or oral administration. Reorganisation of medication so that it is given mainly at night might avoid the need to give hypnotic medication. Changes along these lines would save expenditure on drugs, decrease side-effects and errors, and allow more nursing time to be spent with patients instead of administering drugs.

We would agree with Diamond *et al* (1976) and Edwards & Kumar (1984) that prescribing surveys form a useful part of medical audit, but it seems from this follow-up study that publication of the results of surveys is not enough to overcome the inertia of prescribing habits. Further follow-up studies are needed, associated with planned programmes of information and discussion.

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References

AYD, F. J. (1973) Rational pharmacotherapy: Once-a-day drug dosage. *Diseases of the Nervous System*, 34, 371–378.

DIAMOND, H., TISLOW, R., SNYDER, T. & RICHELS, K. (1976) Peer review of prescribing patterns in a CHMC. *American Journal of Psychiatry*, 133, 697–699.

EDWARDS, S. & KUMAR, V. (1984) A survey of prescribing of psychotropic drugs in a Birmingham psychiatric hospital. *British Journal of Psychiatry*, 145, 502–507.

HOLDEN, N. L. (1984) Prescribing psychotropic drugs. *British Journal of Psychiatry*, 145, 93–94.

KANE, J. R. & SMITH, J. R. (1982) Tardive dyskinesia. *Archives of General Psychiatry*, 39, 473–481.

KIMBALL, C. P. & GLASS, R. M. (1983) Concommitant anti-psychotic drug use. *Journal of the American Medical Association*, 250, 1332.

KLETT, C. J. & CAFFEY, E. (1972) Evaluating the long-term need for antiparkinsonian drugs by chronic schizophrenics. *Archives of General Psychiatry*, 26, 374–379.

MCCLELLAND, H. A. BLESSED, G., BHATE, S., ALI, N. & CLARKE, P. A. (1974) The abrupt withdrawal of antiparkinsonian drugs in schizophrenic patients. *British Journal of Psychiatry*, 124, 151–159.

MICHEL, K. & KOLAKOWSKA, T. (1981) A survey of psychotropic drug prescribing in two psychiatric hospitals. *British Journal of Psychiatry*, 138, 217–221.

MORGAN, R. & CHEADLE, J. (1974) Maintenance treatment of chronic schizophrenia with neuroleptic drugs. *Acta Psychiatrica Scandinavica*, 50, 78–85.

—— & GOPALASWAMY, A. K. (1984) Psychotropic drugs: another survey of prescribing patterns. *British Journal of Psychiatry*, 144, 298–302.

PERRIS, C., DIMITRIJEVIC, P., JACOBSSON, L., PAULSON, P., RAPP, W. & FROBERG, H. (1979) Tardive dyskinesia in psychiatric patients treated with neuroleptics. *British Journal of Psychiatry*, 135, 509–514.

RIFKIN, A. QUITKEN, F., KANE, J., STRUVE, F. & KLEIN, D. F. (1978) Are prophylactic anti-parkinsonian drugs necessary? *Archives of General Psychiatry*, 35, 483–489.

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Manic Depressive Psychosis with Mental Retardation and Flexion Deformities:
A Clinical and Cytogenetic Study

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Five mentally handicapped patients are described in whom a bipolar manic depressive psychosis was associated with flexion deformities, involving principally the fingers. The effect of increasing degrees of retardation on the clinical presentation of the affective psychosis is discussed. Surgical treatment of the flexion deformity brought about considerable improvement in one patient. These five patients were further investigated cytogenetically using high resolution banding techniques. The results obtained were interesting but inconclusive. There would seem to be a definite place for further cytogenetic investigations of some of the more distinctive psychotic disorders using this technique.

One of the authors (A.H.R) has had a longstanding interest in affective disorders in mentally handicapped people (Reid, 1972, 1982), and had been curious to note the development of flexion deformities of the hands in two mentally handicapped siblings in Strathmartine Hospital (mental handicap), both of whom suffered from a bipolar affective psychosis. These flexion deformities had proved remorselessly progressive over the years, despite physiotherapy and even the use of spring-loaded splints.

It seemed possible that the mental retardation, affective disorder and flexion deformities might be causally related, so the authors decided to survey the hands of all the significantly mentally retarded (IQ<50) bipolar manic depressive patients in Strathmartine Hospital to see if they too were affected. In all, five such patients were known to A.H.R. The authors were intrigued to find that each of these five patients showed varying degrees of flexion deformities of the fingers, which had sometimes been misdiagnosed as Dupuytren's contracture. Therefore, we decided to investigate these five patients further by recording the nature and extent of their flexion deformities, preparing a careful psychiatric history and examining their chromosomal findings in some detail. We carried out these chromosome studies in collaboration with the MRC Clinical and Population Cytogenetics Unit in Edinburgh.

Method

Chromosomal preparations were made from cultures of peripheral blood. Extended chromosomes were obtained either by the method of Yunis (1976) or by the method of Ikeuchi (1984) and were G-banded by the ASG method of Sumner *et al* (1971).

Clinical psychiatric and orthopaedic findings

Case 1

Male, aged 58, profoundly mentally retarded, suffers from occasional *grand mal* fits.

This patient can feed himself with a fork and spoon but needs prompting in matters of personal hygiene, dressing and undressing. He has no language but some comprehension.

Prior to admission to hospital at the age of 39 he had been reported as having episodes of restlessness accompanied by insomnia. Since admission he has been noted to have repeated episodes of overactivity, sometimes very severe and accompanied by insomnia, noisiness, unpredictability and aggressiveness, lasting a few days to a few weeks and usually around 1 month. Occasionally these episodes of restlessness are followed by brief periods of unusually quiet, withdrawn behaviour.

At the age of 39, he was described by the physiotherapist as having "excessive tension in many muscles". There were no observable changes at this time but shortly afterwards these began to appear and he has now gone on to develop moderate-to-severe flexion deformities of the middle, ring and little fingers of each hand, with minor involvement of the index finger of the right hand also. In addition there is some limitation of the range of extension of the thumbs on both sides at the metacarpophalangeal joints. There are flexion deformities of the elbows but both wrists have a full range of dorsiflexion and palmar flexion. With the wrists in palmar flexion, it is possible to increase the extension of all the finger joints of the little, middle and ring fingers by some 5–10°. He has little use of his hands but attempts to undo buttons with his thumbs. He can lift objects such as boxes with his thumbs opposing his flexed index fingers and with the wrists held in palmar flexion. There is some wasting of the left leg, involving the calf muscles in particular, but little functional weakness and only a slight diminution of dorsiflexion of the left foot. There is also marked thickening of the outer helix of each ear. There is no evidence of Dupuytren's contracture in either hands or feet and no evidence of Peyronie's disease.

Treatment over the years has included phenothiazines, thiothixenes, butyrophenones, lithium, anticonvulsants and carbamazepine. He is now on carbamazepine 200 mg *t.i.d.*, haloperidol 3 mg *b.i.d.*, procyclidine 5 mg *b.i.d.* and Priadel 1200 mg daily.

Case 2

Female, aged 53, moderately mentally retarded: the sister of Case 1.

She is able to cope with dressing, undressing and most matters of personal hygiene although with some supervision; however, she is persistently inclined to abrade her skin by rubbing it excessively hard with a flannel. Her feeding habits are poor and she tends to bolt her food. In the main she is continent unless mentally disturbed. She is illiterate but able to carry on a rudimentary conversation.

She was described as intermittently sullen and moody at home prior to admission to hospital at the age of 34, but following admission she appeared well settled and posed little in the way of behaviour problems until she was aged 40 when she became quite suddenly quiet, withdrawn, lethargic and hypochondriacal and lost weight. This lasted a few weeks when, over the course of a few days, she displayed a complete change of mood and activity becoming restless, talkative with flight of ideas and an unusual willingness to explore new activities. She expressed wishful fantasies of impending marriage and childbearing. Since then she has shown repeated swings of mood lasting a few weeks to a few months interspersed with periods of normality. When depressed she is described as lethargic, withdrawn, 'confused' and at times preoccupied with thoughts of death or contamination, or with nihilistic ideas of having no clothes. On one occasion she was described as verging on stupor. Manic swings are characterised by restlessness, noisiness, agitation and insomnia. Elation of mood is sometimes present, but more often she becomes excited and the clinical picture is then of a mixed affective state, in which morbid themes and preoccupations co-exist with marked restlessness and impulsiveness. She is sometimes incontinent during episodes of affective disorder.

At the age of 40 she was described as having some 'stiffness' of the fingers. There were no observable changes at the time, but at the age of 47 these were noted to be developing and she was referred for an orthopaedic opinion. She has now gone on to develop flexion deformities affecting particularly the little and ring fingers of the right hand, the index and middle fingers of that hand to a lesser extent, and recently also the little finger of the left hand. In addition, there is some limitation of the range of extension of the right thumb at the metacarpophalangeal joint of the order of some 20°. The wrists and elbows are fully mobile and there is no evidence of Dupuytren's contracture in either hands or feet. Repeated attempts have been made over the years to prevent the development and progression of these finger deformities with help from orthopaedic surgeons, physiotherapy and a variety of spring-loaded splinting devices, all to no avail. She has also had for many years a smooth, non-toxic goitre, not requiring thyroid supplements and antedating the prescription of lithium.

It was felt that she might benefit from surgical lengthening of the common tendons of the flexor digitorum sublimis group and this was carried out via a longitudinal incision in her right forearm. Post-operatively, a considerable increase in extension of the distal and proximal interphalangeal joints of the right hand was achieved.

Treatment over the years has included phenothiazines and butyrophenones, to which she has developed marked side-effects including rigidity and drooling, all of them ceasing on reduction of medication to a low level. She has also had one episode of jaundice which was thought to be due to phenothiazine sensitivity. She was treated with lithium between 1973 and 1979, but this was discontinued because it did not appear to be effective in controlling mood swings and there was concern about its effects on her goitre and renal function. She has also had tricyclic antidepressants as required and two short courses of ECT when at the bottom of a depressive mood swing. She is now on chlorpromazine 25 mg *b.i.d.* and benzhexol 2 mg *b.i.d.*

Case 3

Male, aged 58, severely mentally retarded.

This patient was admitted to Strathmartine Hospital at the age of 11. He is clean, toilet-trained and able to feed and dress himself to some extent. He has a few words but cannot communicate meaningfully.

At the age of 24, he was described as catatonic with *flexibilitas cerea*, and thereafter he went through a phase of negativism and food refusal during which it was difficult to sustain his weight. Around the age of 40, he began to have episodes of noisy, restless, active behaviour, lasting from a few days to a few weeks and accompanied by disturbed sleep. In these episodes, he chatters away volubly in disconnected words, apparently devoid of any communication component. He appears elated in mood and he can become very excited indeed, and latterly aggressive. At other times, he is reported as having had episodes in which he is quiet, passive, withdrawn and unemotional.

In earlier years, he was noted to have particularly long, thin and mobile fingers and toes, suggestive of Marfan's syndrome but with no other symptoms of that disorder. At the age of 55, he was noted to be holding his left arm and hand in an unusual, 'stiff' posture: this proved persistent and progressed rapidly to involve the right arm and hand. He was referred for neurological and orthopaedic opinions, and a variety of medications, physiotherapy and splinting was suggested but to no avail. The flexion deformities progressed rapidly and he has now gone on to develop severe and virtually fixed flexion of the fingers of both hands, with palmar maceration due to the finger-nails digging in. A small degree of passive extension of the little finger is possible and both thumbs are freely mobile. The patient tends to hold his wrists in palmar flexion but passive dorsiflexion is possible with difficulty. There is some flexion deformity of the elbows but the degree is hard to assess. There is no evidence of Dupuytren's contracture in either hands or feet, and no evidence of Peyronie's disease. He is very uncooperative with examination of his hands, and thus no detailed measurements were possible.

Treatment over the years has included phenothiazines, butyrophenones, lithium and carbamazepine, the latter prescribed in an endeavour to control restlessness (Reid *et al*, 1981). He is now on lithium carbonate 800 mg and carbamazepine 500 mg, each at 8 p.m., with an as-required dose of chlorpromazine 100 mg which is hardly ever used.

Case 4

Female, aged 46, moderately mentally retarded.

This patient is competent in most matters of self-care skills within a protected setting and can communicate verbally in a childish way.

She had fits in childhood and adolescence but has had none since the age of 14. She was exploited sexually in earlier years and was admitted to hospital at the age of 17. She is a friendly woman but rather impulsive and quick-tempered. At the age of 28, she had an episode of depressive psychosis in which she appeared depressed, quiet and sullen. She was hypochondriacal and unpredictably aggressive towards nursing staff. Around 1 year later, she became acutely mentally disturbed, restless, talkative and expressing grandiose and wishful delusions of a sexual and pregnancy nature. She entertained the belief that she knew everything, could teach the doctors and nurses and was possessed of great wealth. She claimed to be in communication with God. Her sleep was disturbed with very early morning wakening. This episode of hypomania subsided but subsequently there have been further episodes of affective disorder, although mood swings have been very much less since she was started on lithium in 1971.

On examination of her hands, she has no measurable flexion deformities. However, with the hands held in the position of prayer with the fingers dorsiflexed, there is a clear tendency for the fingers to curl into minor flexion such that the palmar surfaces of the fingers cannot be approximated. There is also the impression of some tightness of the long flexors.

In earlier years, she was treated with phenothiazines, antidepressants, one course of ECT and butyrophenones as appropriate for episodes of affective disorder. Since 1971 she has been maintained on lithium carbonate which has been substantially successful in controlling her affective disorder. Her present medication consists of haloperidol 3 mg *t.i.d.*, benzhexol 4 mg *t.i.d.* and lithium carbonate 500 mg *b.i.d.*

Case 5

Female, aged 57, moderately mentally retarded, suffers from very occasional *grand mal* fits.

This patient is competent in most matters of self-care skills within a protected environment.

She was exploited sexually in earlier years and was admitted to hospital at the age of 26. At the age of 29 she was recorded as having episodes of noisy, quarrelsome behaviour, but her first episode of clear affective illness as such was at the age of 37, when she was described as restless, noisy, elated and talkative for around 4 months, swinging down to a state of quite severe depression with disturbed sleep, early morning wakening, emotional lability and aggressiveness. There was a further episode lasting a few weeks at the age of 41, in which she was restless, noisy, irritable and impulsively aggressive, again followed by a clear depressive downswing with emotional lability, much crying, psychomotor retardation and disturbed sleep. There have been further similar episodes of affective illness over the years and these continue up to the present.

On examination of her hands, she has no measurable flexion deformities but again, with the hands held in the position of prayer, there is a clear tendency for the fingers to curl into minor flexion, such that the palmar surfaces of the fingers cannot be approximated. There is also the impression of some tightness of the long flexors.

Treatment over the years has included phenothiazines, butyrophenones and antidepressants as required for episodes of affective disorder. Since 1971 she has been on lithium carbonate which has been partially successful in controlling her manic depressive psychosis. Her present medication consists of chlorpromazine 50 mg *b.i.d.*, prothiaden 75 mg *nocte* and lithium carbonate 500 mg *nocte*.

The clinical data on these five patients are summarised in Table I and the degrees of the flexion deformities and the results of surgical treatment in Table II.

TABLE I
Clinical data on the five patients

Case	*Sex*	*Retardation range*[1]	*Epilepsy*	*Approximate age at onset of flexion deformities (to nearest 5 years)*
1	M	Profound	Grand mal	40
2	F	Moderate	—	45
3	M	Severe	—	55
4	F	Moderate	In childhood and adolescence	45
5	F	Moderate	Grand mal	55

1. According to ICD-9 (World Health Organization, 1978).

Cytogenetic analysis

G-banded extended chromosomes were examined in all five cases. One observer considered that three of the cases showed a difference between homologues in the region 2q22.1 to 2q24.1. Further examination by other observers led to the conclusion that the differences observed were not inconsistent with the variation observed in control cases. The two other probands showed no abnormality.

TABLE II

Degrees of flexion deformity (0° = full extension)

	Metacarpophalangeal joints degrees	*Proximal interphalangeal joints degrees*	*Distal interphalangeal joints degrees*
Case 1			
Left hand			
Little	35	60	5
Ring	5	55	0
Middle	0	50	0
Index	0	0	0
Thumb	0–15		
Right hand			
Little	65	50	15
Ring	60	95	5
Middle	55	75	5
Index	5	30	0
Thumb	0–20		
Case 2			
Left hand			
Little	0	20	0
Ring	0	0	0
Middle	0	0	0
Index	0	0	10
Right hand (pre-op.)			
Little	0	90	15
Ring	5	60	10
Middle	0	0	10
Index	0	0	10
Right hand (pre-op.)			
Little	0	35	0
Ring	0	35	0
Middle	0	0	0
Index	0	0	0

Discussion

These five patients, only two of whom are related, appear to share some features in common. Firstly, they are all very significantly mentally retarded and in each case the retardation is of unknown origin and appears to be congenitally determined. The presence of such associated phenomena as minor congenital abnormalities in Case 1, and of *grand mal* epilepsy in Cases 1, 4 and 5, is in keeping with this conclusion.

Secondly, all five patients suffer from an associated psychotic illness clinically suggestive of a bipolar manic depressive psychosis. The diagnosis of manic depressive psychosis can be difficult in mentally retarded patients, particularly in those with more severe degrees of handicap such as Cases 1 and 3. In Case 1, however, there was a family history of manic depressive psychosis affecting his sister (Case 2) and, in addition, another younger brother of dull/normal intelligence suffered from a bipolar manic depressive psychosis. This younger brother lived independently, ran the family farm and had been admitted to inpatient psychiatric care in a different hospital in a different catchment area on three occasions with episodes of mental illness. These episodes of illness were diagnosed independently as manic depressive psychosis in nature and treated with lithium in the absence of knowledge of the history of mental illness involving his two siblings. Furthermore, the mother of Case 3 had spent many years in a mental hospital and died there, suffering from a mental illness characterised in earlier years by depression and critical auditory hallucinations, although latterly it had been complicated by severe deafness and was regarded as paraphrenic in nature. Finally, the sister of Case 5 also had a history of affective disorder, treated independently with ECT in another hospital. Therefore, we feel that, notwithstanding the difficulties in diagnosing affective disorder in retarded patients, a good case can be made out for suggesting that these five patients were suffering from a bipolar affective disorder.

Thirdly, these five patients also suffered from flexion deformities of varying degrees involving the fingers in all cases, the thumbs in Cases 1 and 2 and the elbows in Cases 1 and 3. These flexion deformities were associated with a sensation of 'stiffness' or 'tightness' in the long flexor muscles of the fingers. This stiffness had been noted in Cases 1 and 2 some years prior to the onset of obvious flexion deformities. Some of these flexion deformities had been described, wrongly, as Dupuytren's contractures in the notes. Dupuytren's contracture is a pathological condition of the palmar fascia and cannot be overcome partially by flexion of the wrist, or by a flexor tendon lengthening operation in the forearm. These flexion deformities were certainly of variable severity. In Cases 4 and 5, the deformities were mild and possibly of arguable significance, but in Cases 1, 2 and 3 they were obvious, gross and caused considerable nursing problems in relation to feeding, dressing and hygiene. This tendency towards flexor hypertonus is one of the features that Rogers (1985) remarked on in his comprehensive and scholarly review of the motor disorders of severe psychiatric illness. He concluded that these motor disorders lead one inescapably to the unfashionable conclusion that severe psychiatric illness is regarded most appropriately as a form of neurological disorder, and he casts doubt on psychogenic and institutional theories.

There is the possibility that these flexion deformities were drug-induced and related to the prescription over years of phenothiazines, butyrophenones, anticonvulsants or lithium. However, if that is so, it is difficult to explain why they developed in these five patients and not in so many other mentally retarded patients, who were also treated with phenothiazines or butyrophenones but did not suffer from manic depressive psychosis. An increased incidence of Dupuytren's contracture has been reported in epileptics, and Critchley *et al* (1976) suggested a link with long-term phenobarbitone treatment. In the five cases in the present series, however, the deformities were not typical of Dupuytren's contracture, and in two of the cases there was no history of fits. Moreover, these two cases included both the most severely affected patient (Case 3) and one member of the sibling pair (Case 2). There is nothing in the literature to suggest that lithium induces flexion deformities, but it is the one drug these five patients did have in common. However, a survey of 100 consecutive attenders at the lithium clinic in Dundee showed only one hand deformity, and that was traumatic (S. E. Hopwood, personal communication). Therefore, we feel it is unlikely that either phenothiazines, butyrophenones, anticonvulsants or lithium preparations were instrumental in promoting these flexion deformities.

Thus, it seemed that in these five cases, the diagnosis of idiopathic mental retardation, sometimes accompanied by idiopathic epilepsy, manic depressive psychosis and flexion deformities might be meaningfully linked. This was an intriguing possibility and we sought through chromosomal studies to see if a chromosomal marker could be identified.

With the advent of high resolution banding (HRB) techniques, it has become possible to identify up to 850 bands in human chromosomal preparations obtained at the prophase or prometaphase stages of mitosis (ISCN, 1985). This has significantly increased the resolution of cytogenetic techniques for the identification of chromosomal aberrations giving rise to genetic abnormalities. Therefore, HRB studies were used to identify chromosomal aberrations in this sample of patients. The techniques are time-consuming and require considerable expertise, but it was considered important to exclude abnormality at this level of resolution, since the identification of such an abnormality could lead to the localisation of a major gene conferring susceptibility to affective disorder. Genes responsible for retinoblastoma, Wilms' tumour, Duchenne muscular dystrophy, chronic granulomatous disease and other diseases (HGM8, 1985) have been localised following the identification of specific aberrations in this way. In the case of affective disorders, where there is a relatively high general population frequency (*c.* 0.1% for bipolar disorders), the coincidence of a chromosomal aberration with the clinical abnormality could arise by chance. This might occur in about 0.1% of all mentally handicapped individuals, if the two conditions are unrelated. Alternatively, the existence of patients exhibiting both clear-cut cyclical manic-depressive mood swings, together with non-specific features such as mental handicap and/or dysmorphism that are found almost invariably in patients with moderate-sized chromosomal deletions and unbalanced rearrangements, might imply deletion (and loss of function) of a major gene responsible for affective disorder, in addition to loss of other genes causing non-specific developmental abnormality.

There is considerable evidence for the existence of genetic factors in bipolar illness (Gershon, 1983), which is consistent with the presence of a major gene influence showing reduced penetrance (O'Rourke *et al*, 1983). In the case of the patients studied here, there is a third common factor, namely the occurrence of delayed onset, progressive flexion deformities affecting the hands and forearms to varying degrees. This condition has not been described as a specific genetic syndrome either on its own, or in combination with the other features observed here. It could be postulated either that these are pleiotropic effects of a single, rare mutant gene or that they are all consequent on the simultaneous loss of large numbers of linked genes due to deletion of a chromosomal segment. Visible deletions probably only represent a small proportion of all deletions (see Vogel & Rathenberg, 1975), many more of which are now being detected at the DNA level by means of gene probes (Monaco *et al*, 1985). Although some variation in the dark G-bands was observed in three of the patients in a region of chromosome 2 (2q22.1-2q24.1), it was felt that this degree of variation was not outside the range of variation seen in normal controls. This emphasises the difficulty of positively identifying small deletions using HRB, where small differences in the degree of contraction of G bands can occur even between apparently normal chromosomal homologues. Some of the variation of this sort may be of a heritable nature, representing banding polymorphisms; in other cases, it may be due to small differences in the rates of condensation of chromosomal homologues during the progression from prophase to metaphase.

Therefore, our chromosome studies were unfortunately inconclusive. However, we do feel that this technique could be of very substantial potential interest in psychiatry, particularly in the investigation of the more distinctive and identifiable psychotic disorders, and further studies would be very worthwhile.

Addendum

It has just come to light that the sister of Case 4 suffers from a bipolar manic depressive psychosis, independently diagnosed and treated with lithium in another hospital in the absence of knowledge of the relationship. She is also mentally retarded and said to have the same incurving fingers with inability to approximate the palmar surfaces.

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References

Critchley, E. M. R., Vakil, S. D., Hayward, H. W. & Owen, V. M. H. (1976) Dupuytren's disease in epilepsy: result of prolonged administration of anticonvulsants. *Journal of Neurology, Neurosurgery & Psychiatry*, 39, 498–503.

Gershon, E. S. (1983) Genetics of the major psychoses. In *Genetics of Neurological and Psychiatric Disorders* (eds S. S. Kety, L. P. Rowland, R. L. Sidman & S. W. Matthysse). New York: Raven Press.

HGM 8 (1985) Eighth International Workshop on Human Gene Mapping. *Cytogenetics and Cell Genetics*, 40, 1–4.

ISCN (1985) An international system for human cytogenetic nomenclature. *Birth Defects: Original Article Series*, 21 (1), 1–117.

Ikeuchi, T. (1984) Inhibitory effect of ethidium bromide on mitotic chromosome condensation and its application to high-resolution chromosome banding. *Cytogenetics and Cell Genetics*, 38, 56–61.

Monaco, A. P., Bertelson, C. J., Middlesworth, W., Colletti, C., Aldridge, J. Fischbeck, K. H., Bartlett, R., Periczak-Fance, M. A., Roses, A. D. & Kunkel, L. M. (1985) Detection of deletions spanning the Duchenne muscular dystrophy locus using a tightly linked DNA segment. *Nature*, 316, 842–845.

O'Rourke, D. H., McGuffin, P. & Reich, T. (1983) Genetic analysis of manic-depressive illness. *American Journal of Physical Anthropology*, 62, 51–59.

Reid, A. H. (1972) Psychoses in adult mental defectives: I. Manic depressive psychosis. *British Journal of Psychiatry*, 120, 205–212.

—— (1982) *The Psychiatry of Mental Handicap*. London: Blackwell Scientific Publications.

—— Naylor, G. J. & Kay, D. S. G. (1981) A double-blind, placebo controlled, crossover trial of carbamazepine in over-active, severely mentally handicapped patients. *Psychological Medicine*, 11, 109–113.

Rogers, D. (1985) The motor disorders of severe psychiatric illness: a conflict of paradigms. *British Journal of Psychiatry*, 147, 221–232.

Sumner, A. T., Evans, H. J. & Buckland, R. A. (1971) New techniques for distinguishing between human chromosomes. *Nature New Biology*, 232, 31–32.

Vogel, F. & Rathenberg, R. (1975) Spontaneous mutation in man. *Advances in Human Genetics*, 5, 223–318.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to the Classification in Accordance with the 9th Revision of the International Classification of Diseases*. Geneva: WHO.

Yunis, J. J. (1976) High resolution of human chromosomes. *Science* 191, 1268–1270.

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The Influence of Depression on the Processing of Personal Attributes

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Depressed, schizophrenic and normal subjects were tested for incidental recall and recognition of lists of positive and negative personal attributes. It was hypothesised that depressives would show a deficit in recall and recognition of words of which they had been asked a self-referential encoding question, but would show equivalent performance to controls on words of which they had been asked an other-referential, semantic or structural encoding question. The experiment was designed to enable a decision to be made between two possible explanations of the expected deficit: Davis's (1979) suggestion that it is due to disorganisation of the self-schema in depression, and the hypothesis of Beck *et al* (1979) that depression is characterised by the predominance of a negative self-schema. The expected deficit was observed on the recall but not on the recognition task. However, the precise pattern of the results raises problems for both of the above interpretations, and alternative explanations are considered.

There has been a growth of interest in the role of cognitive processes in psychological disorders, stimulated on one hand by the emergence of cognitive theories of clinically observed phenomena, such as Beck's (1967) theory of depression, and on the other by attempts to apply information-processing models to the deficits or symptoms observed in clinical populations (e.g. Hemsley, 1982). The present study investigated the processing of personal attributes by depressed patients, attempting to clarify the role of depressives' self-schema in their psychological functioning.

There has been a number of attempts to relate cognitive functioning to a psychological theory of depression, Beck's being the most influential. The central premise is that the personal paradigm of the patient when he is in a depressed state produces a distorted view of himself and his world (Beck *et al*, 1979). Distortion is of a negative kind such that persistent negative evaluations are made of the self, world and future. Negative affect is explained as a consequence of negative cognitions, and other depressive deficits, such as in motivation or concentration, are regarded as secondary to the primary cognitive dysfunction. Thus, according to Beck, the depressive subjects incoming information to a systematic schema-mediated processing bias, the self-schema being the origin of such bias.

Beck's concept of the self-schema is similar to that put forward by Rogers *et al* (1977), especially in its implication that the self is active in at least some forms of information processing. Rogers *et al* (1977) set out to investigate "the construct of self and how it is implicated in the organisation of personal data". The self was defined as "an abstract representation of past experience with personal data", which is "activated when a person encounters a situation involving personal information, becoming part of the available information-processing system". To test this they adopted Craik & Lockhart's (1972) proposal that there is a hierarchical organisation of levels of processing, and that the 'depth' of such processing predicts memory trace strength and, in turn, retention, applying this to verbal information. Lockhart *et al* (1976) suggested that the physical, phonemic and semantic characteristics of words exist in what Sutherland (1972) refers to as different domains "proceeding from shallow structural domains to deep semantic domains". A domain in which the self-concept is active is therefore 'deep' in two senses: first, that it is higher in the hierarchy than physical or phonemic domains and, secondly, that *within* the set of semantic domains it would be expected to be of particular significance. To test this, Rogers *et al* (1977) required subjects to encode personal adjectives under different orienting conditions, using a variant of the incidental recall paradigm (Craik & Tulving, 1975). The self-referential question (i.e. describes you?) produced superior recall to the other three encoding questions (semantic, phonemic and structural). They claimed that "involvement of the self in the interpretation of new stimuli imparts a degree of fullness and richness to the input" and that during recall "subjects probably use the self as a retrieval cue'' (Rogers *et al*, 1977). A later study (Rogers *et al*, 1979) employed a similar recognition task, exploring the possibility that the earlier results were not a function of the self-schema but simply an effect of the involvement of a person in the rating task. Though not unambiguous, they concluded that different processes occur in self- and other-referential coding and therefore that the concept of the self-schema is justified.

Other investigators have implicated the self in information processing (e.g. Markus, 1977; Bower & Gilligan, 1979) and for a long time personal construct theorists have had a concept of the self-schema similar to that proposed by Rogers *et al*. However, the first study to employ the methodology of Rogers *et al* (1977) within a clinical population was reported by Davis (1979). He argued that not all depressives respond according to a negative self-schema. Noting that some depressed patients describe themselves inconsistently, he proposed that these had a less well integrated self-schema, comparable to the subjective phenomenon of uncertainty about the self. Davis's (1979) findings demonstrated that depressives' recall was significantly worse on the self-referential coding task than that of the controls, although their performance was equivalent on the other tasks. A problem with the study is that, of the 48 personal attributes employed in his study, 31 were rated by 10 colleagues of the present authors as positive. Hence depressives' performance might be attributable to their tendency to screen out positive input related to the self. A more balanced list, as was to be employed in the present study, could have resulted in equivalent recall on the self-referential question for depressives and controls.

It might also be the case that, irrespective of whether the attributes were regarded as self-descriptive, depressives would have a tendency to recall predominantly negative words, as found by Bradley & Mathews (1983). Most experiments have inferred an adjective's self-descriptive status from a 'yes' response in the self-referential coding condition. This prejudges the issue of whether the self-schema is in fact involved in recall. However, if the operation of the self concept were a major factor influencing recall, one would expect more negative self-descriptive words to be recalled than negative non-self-descriptive words for the depressives, and the converse for the control group. To assess this, the present study required that subjects rate all the words on the list presented, according to a self-descriptive question, after completing the recall task. One can then examine the words recalled to see if more self-descriptive words are recalled than expected by chance. On Davis's theory, since depressives are characterised by ''non schema based responding'', a self-schema effect would not be predicted for the depressives, whereas on Beck's theory it would be dependent on whether the words were positive or negative. A lack of self-schema effect for both groups would challenge both Beck's and Davis's theories, since they agree in implicating the self-schema in non-depressed responding, and only disagree on its role in depressed responses.

There were three further extensions to Davis's study: first, the addition of a recognition task similar to that employed by Rogers *et al*; secondly, the inclusion within this task of both other- and familiar other-referential questions; thirdly, the inclusion of a clinical control group. A sample of schizophrenic patients was assessed in view of several authors' theories of the nature of the self-schema in schizophrenia (e.g. Laing, 1960).

Method

All subjects were to carry out two tasks, one involving recall, the other recognition. For the recall task, three forms of coding were to be employed, for the recognition task four forms.

Materials

A set of positive and negative personal attributes was generated. From this set a subset consisting of half positive and half negative attributes was selected for each of the tasks. The subset was then ordered in a number of ways corresponding to the number of forms of questions being presented in each task and a question order was assigned, each word having each question asked of it on only one of the lists.

The recall list consisted of 24 words, the recognition list of 32 words. In addition, 64 distractor words were required to construct the lists from which subjects were to select the words they recognised. To construct the word lists, a group of 20 psychology students were asked to rate 150 personal attributes as either good, bad or uncertain, and in addition to describe whether it was of long, short or medium length. The recall task set comprised six short positive, six short negative, six long positive and six long negative words. The recognition task set consisted of eight of each of these categories, and the distractor set of these 32 words plus 32 positive and 32 negative words.

For the recall task lists, three word orders were generated randomly. Each word was then assigned one of the three coding questions for each list, such that on any one presentation of a particular list, each coding question would be presented eight times, four times to positive words, and four times to negative words. This was done in such a way as to obviate any possible order effect.

The recognition task list was drawn up in a similar way, except that four word orders were generated. Four recognition distractor lists were constructed of 24 words each. Each consisted of eight words from the list to be remembered (four positive and four negative) and 16 distractor words (eight positive and eight negative).

Coding questions

The coding questions used for the recall task were:

(1) Does the word . . . describe you? (*self-referential question*)

(2) Would you expect a 7-year old child to understand the meaning of . . .? (*semantic question*)

(3) Is the word . . . a long word? (*structural question*)

For the recognition task, questions 1 and 2 from the recall task were employed, plus:

(4) Does the word . . . describe your mother? (*familiar other referential question*)

(5) Does the word . . . describe the person in the photograph? (*photograph of smiling woman; unfamiliar other question*)

Subjects

Subjects were 12 depressed patients, 10 schizophrenic patients and 12 normal controls. The clinical samples were drawn from the Maudsley, Bethlem, St Giles and St Francis Hospitals, London. All but two of the depressives were inpatients. All depressed subjects had received a primary diagnosis of non-psychotic depression and scored more than 20 on the Beck Depression Inventory (BDI) (Beck, 1967). They had an average age of 41.9 years, and a mean BDI score of 31.6. The schizophrenic subjects had a primary diagnosis of schizophrenia, had exhibited first-rank symptoms and had to score below 15 on the BDI. They had an average age of 33.8 years and a mean BDI score of 8.0. The normal controls had an average age of 28.2 years and a mean BDI score of 2.9. No subject had received ECT, showed any signs of neurological abnormality or had a history of alcohol abuse. All subjects completed the Schonell Graded Word Reading Test, and were excluded if scoring less than 58. This test correlates with intelligence (Nelson & McKenna, 1975). Group characteristics, including IQ scores were derived from the tables provided by Nelson & McKenna. The groups did not differ significantly on IQ.

Procedure

Subjects were told that the author was carrying out research on "the way people understand the meanings of words". If they agreed to participate in the study, the Schonell reading test was then administered, it being explained that the subject was to be required to read some words, and that this was a preliminary to ensure that they would be able to do so. Subjects were then presented with the list of words for the recall task. They were given the following instructions: "In front of you is a list of words. I shall be asking you one question about each word, and I would like you to circle a yes or no answer for each word. You may find it hard to decide upon your answer in some cases, but I would still like you to make a definite choice. After I have asked you each question, I would like you to wait until I signal before circling your answer, and then do so promptly. After completing the questions I shall be asking you some more questions about the words on the list." One question was then asked aloud about each word, the experimenter pausing for 3 s after finishing the question before signalling to the subject to circle their response. Once the list was completed, the experimenter turned it over and said, "Now the next thing I would like you to do is to write down as many of the words on that list as you can remember. You have plenty of time. Don't worry about spelling." Subjects were then given 4 min to think of words. The answer sheet was then turned over to its original position, and subjects told: "The final thing I'd like you to do with the list of words is to tick those words that you think definitely describe you. If you are uncertain then miss the word out." This completed the recall task.

Subjects were then presented with the recognition task word list. They were told: "Here is another list of words. We shall be doing the same kind of thing as with the previous list, except that some of the questions will be slightly different, and so will the questions at the end". The photograph was then put next to the word list, the experimenter explaining that "some of the questions will be about the person in this photograph".

Once the coding questions were finished, the list was taken away and the experimenter continued: "This time, rather than asking you to remember words directly, I've four lists of words here. Each list contains some words from the last list and some new words that weren't on either that list or the first list. Indicate those that were on the last list." Subjects were then presented with the four distractor lists in varying orders across subjects, in case later lists tended to have fewer words selected than earlier ones. Finally, subjects were re-presented with their original answer sheet, and asked to tick those words that described them, as in the previous task.

Subjects then completed the Beck Depression Inventory.

Results

Recall task

Results from the two-way analysis of variance are presented graphically in Fig. 1. Overall, these showed significantly

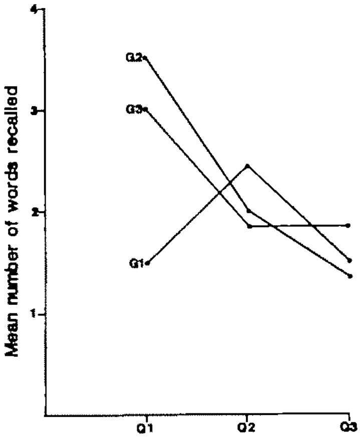

FIG. 1 Group × question interaction (recall task).

TABLE I
Recall task: Group × variable one-way ANOVA

Variable	*G1 Mean*	*G2 Mean*	*G3 Mean*
Self-descriptive positive words recalled	1.7	1.9	2.3
Self-descriptive negative words recalled	0.9	0.9	0.6
Total self-descriptive words recalled	2.6	2.8	2.9
No. of positive self-descriptive words	5.5	6.7	8.3
No. of negative self-descriptive words	5.0	2.7	1.4
Total self-descriptive words	10.5	9.4	9.7
Positive words recalled on self-referential question	0.7	1.8	1.7
Negative words recalled on self-referential question	0.8	1.6	1.3
Total positive words recalled	2.7	2.9	3.2
Total negative words recalled	2.6	3.3	3.4

enhanced recall for the self-referential condition ($P<0.01$, $F=6.639$). Depressives recalled fewer words of which they had been asked the self-referential question than did the other groups ($P<0.01$, $F=5.1$), but showed equivalent recall on the semantic and structural questions.

Analysis of the data of Table I revealed the following:

(a) Depressives selected as self-descriptive fewer positive words ($P<0.06$, $F=3.1$) and more negative words than did controls ($P<0.002$, $F=11.4$).

(b) Depressives recalled as many self-descriptive words as did the control groups, and there was no tendency for any group to recall more self-descriptive words than non-self-descriptive.

(c) Depressives showed no tendency to recall proportionately more negative self-descriptive words, nor did controls show a disproportionate bias towards positive self-descriptive words.

(d) Depressives showed no tendency to recall negative rather than positive words for the task as a whole.

(e) Depressives showed no tendency to recall negative rather than positive words in relation to the self-referential question.

(f) Depressives recalled fewer positive words on the self-referential question than controls ($P<0.001$, $F=7.2$). There was also a (non-significant) tendency to recall fewer negative words ($P<0.2$, $F=1.8$).

There was no tendency for any group to remember more words to which 'Yes' replies were given.

Recognition task

The results of the two-way analysis of variance are illustrated in Fig. 2. The self-referential question resulted in higher recognition scores for all groups ($P<0.01$, $F=8.5$). This is in marked contrast to the depressives' deficit on the recall task under this coding condition.

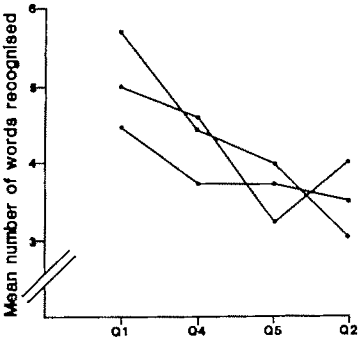

FIG. 2 Group × question interaction (recognition task).

The univariate analyses produced a similar pattern of results as on the recall task and hence will be mentioned only briefly. Depressives recognised as many self-descriptive words as did the control groups. There was no tendency for depressives to recognise only negative words on the self-referential question. Although there was no difference between groups in the number of positive or negative words recognised, depressives did make more negative 'false alarms' than the other groups made ($F=9.6$; d.f. 2, 31; $P<0.001$). The groups did not differ significantly on the number of positive false alarms. Although a signal detection theory analysis of these last finds was considered, it was felt that the assumptions necessary for its application were not necessarily justified for the present task.

Discussion

As expected, the control subjects recalled significantly more words related to the self-referential question than to the semantic or structural question. This is consistent with the original findings of Rogers *et al* (1977). Similar findings for the control subjects are apparent on the recognition task.

The pattern of results for the depressed subjects is somewhat different. As in Davis's (1979) study, on the recall task depressives recalled significantly fewer words of which they had been asked the self-referential question, compared to both control groups, but showed equivalent performance on the semantic and structural questions. Although this group difference was somewhat greater for positive words on the self-referential question, the trend was also apparent for negative words. This is in contrast with the findings of Derry & Kuiper (1981), who claimed that the expected enhanced recall under the self-referential condition was apparent for depressives if depressed content adjectives were employed.

On the recognition task, depressives showed no deficit on the self-referential question. However, while positive 'false alarms' were made equally often by the depressives and normals, negative 'false alarms' were more common in the depressed group. Dunbar & Lishman (1984) found no such difference but did report a higher 'hit rate' for negative words in their depressed group relative to controls. The latter authors carried out a signal detection analysis in an attempt to clarify their findings. As noted above, this was not done here, as it was felt to depend on assumptions whose validity for the present experimental task remains to be demonstrated.

It could be argued that depressives' differential pattern of performance on recall and recognition tasks is because only the recall task required the use of the self-schema as a retrieval cue. However, it is clear that there was no tendency for any group to recall or recognise proportionally more words that were regarded as definitely self-descriptive as opposed to those words that were not. This is hard to reconcile with the theory of Rogers *et al* (1977) that it is the self-schema which is active in producing enhanced recall for the self-referential question. By the same token, Davis's theory that depressives' deficit on recall is due to disorganised self-schema relative to that of non-depressed subjects fails to receive confirmation, since it also depends on the assumption of an observable self-schema effect on non-depressed recall of self-descriptive words. Thus, although the self may indeed be functioning in initial processing, it does not seem to be implicated in retrieval in the way proposed.

Nor are the data clearly supportive of the hypothesis suggested by Beck's theory, that depressives' recall deficit may be due to a negative response bias. His general theory receives some support from the depressives' self-image being significantly more negative, and their tendency on the recognition task incorrectly to select negative words as having been on the original list more often than controls. However, its application to the effect under consideration is questioned by the lack of a tendency for depressives to recall more negative words, their failure to recall proportionately more negative self-descriptive words than positive self-descriptive words, and the lack of a tendency for them to recall more negative words than did the controls in relation to the self-referential question.

Therefore, the data do not clearly support either Davis's or Beck's account of the replicated depressive deficit on the self-referential recall task. The absence of this effect and the greater number of false alarms on the recognition task also have to be explained. Rather than using the self-schema as a cue in retrieval, as suggested by Rogers *et al*, it is possible that in order to recall the self-referential question words, the subject uses the self-referential *question* as a cue. This effectively requires 'thinking about himself'—for a depressed person a process that may act simultaneously as a cue for negative affect and/or a distraction from the recall task on to a stream of automatic, negative thoughts, as suggested in Beck's theory. In other words, the subject may be diverted from the goal of retrieval because the retrieval cue also cues other processes. This could also explain the tendency to perform worse on recall of positive words, since it is plausible that negative affect is more likely to interfere with the recall of positive attributes than negative ones. The lack of differential performance on the recognition task is explained because there is not the same need for a retrieval cue as the words are already present. Also explained is the greater number of negative false alarms for depressives than for controls, since negative affect is likely to make negative items seem more congruent with the subject's experience than positive ones, and therefore seem more familiar.

The problem remains of reconciling the lack of self-schema effect for all groups with the enhanced recall of controls on the self-referential coding condition. One answer is to make the intuitive distinction between the self and the self-schema, the latter being defined as a representation of the former. In accordance with the depth of processing model, one could then agree with Rogers' claim that involvement of the *self* imparts greater richness to the input, and therefore that memory trace strength as measured by recall would ordinarily be enhanced, but dispense with the view that the *self-schema* is used as a retrieval cue. This would explain the key finding that recall performance and self-descriptiveness appear to act independently.

References

Beck, A. T. (1967) *Depression: Clinical, Experimental and Theoretical Aspects*. New York: Hoeber.

——, Rush, A. J., Shaw, B. F. & Emery, G. (1979) *Cognitive Therapy of Depression*. New York: John Wiley.

Bower, G. H. & Gilligan, S. G. (1979) *Remembering information related to one's self. Journal of Research on Personality*, 13, 420.

Bradley, B. & Mathews, A. (1983) Negative self-schemata in depression. *British Journal of Clinical Psychology*, 22, 173.

Craik, F. I. M. & Lockhart, R. S. (1972) Levels of processing: a framework for memory research. *Journal of Verbal Learning and Verbal Behaviour*, 11, 671–684.

—— & Tulving, W. S. (1975) Depth of processing and the retention of words in episodic memory. *Journal of Experimental Psychology: General*, 104, 268–294.

Davis, H. (1979) The self-schema and subjective organisation of personal information in depression. *Cognitive Therapy and Research*, 3, 415–425.

Derry, P. A. & Kuiper, N. A. (1981) Schematic processing and self reference in clinical depression. *Journal of Abnormal Psychology*, 90, 286–297.

Dunbar, G. C. & Lishman, W. A. (1984) Depression, recognition memory and hedonic tone: a signal detection analysis. *British Journal of Psychiatry*, 144, 376–382.

Hemsley, D. R. (1982) Cognitive impairment in schizophrenia. In *The Pathology and Psychology of Cognition* (ed. A. Burton). London: Methuen.

Krishnan, N., Soodiri, N., Carter, L. F. & Baldock, H. M., Jr (1975) *Applied Multivariate Analysis and Experimental Designs*. New York: McGraw-Hill.

Laing, R. P. (1960) *The Divided Self*. London: Tavistock.

Lockhart, R. S., Craik, F. I. M. & Jacoby, L. (1976) Depth of processing: recognition and recall. In *Recognition and Recall* (ed. J. Brown). New York: John Wiley.

Markus, H. (1977) Self-schemata and processing information about the self. *Journal of Personality and Social Psychology*, 35, 63–78.

Nelson, H. E. & McKenna, P. (1975) The use of current reading ability in estimation of premorbid intelligence. *British Journal of Social Psychology*, 14, 259–267.

Rogers, T. B., Kuper, N. A. & Kirker, W. S. (1977) Self-reference and the encoding of personal information. *Journal of Personality and Social Psychology*, 35, 677–688.

——, Rogers, P. J. & Kuiper, N. A. (1979) Evidence for the self as a cognitive prototype: the 'false alarms effect'. *Bulletin of Personality and Social Psychology*, 5, (1).

Sutherland, N. S. (1972) Object recognition. In *Handbook of Perception* (eds E. C. Carterette & M. P. Friedman). New York: Academic Press.

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Brief Reports

Neuroleptic Malignant Syndrome Superimposed on Tardive Dyskinesia

A 30-year-old man with a 3-year history of tardive dyskinesia developed a neuroleptic malignant syndrome while having reserpine and lithium; his symptoms worsened following three doses of neuroleptic medication and improved with bromocriptine. The pre-existing dyskinesia made the presentation confused, and delayed proper diagnosis.

Neuroleptic malignant syndrome (NMS) is an acute and sometimes fatal complication of neuroleptic medication, characterised by muscle rigidity, hyperpyrexia, and mental confusion; it appears to be caused by alterations in dopamine neurotransmission in the basal ganglia and the hypothalamus. Its relationship to other neuroleptic-induced movement disorders such as dystonia, parkinsonism, and tardive dyskinesia (TD) remains unclear. We describe here a case of NMS that developed in a patient with pre-existing TD; to the best of our knowledge, this is the first report of such an association. The concurrence of these two syndromes in this case delayed the recognition of NMS, and led to treatment strategies that may have exacerbated it.

The Case

Mr A, a 30-year-old white male with a history of recurrent paranoid psychosis, was referred to our inpatient service for treatment of severe TD of approximately 3 years' duration. He had taken neuroleptic medication, including thioridazine, oral and depot fluphenazine, and trifluoperazine continuously for 7 years. His abnormal movements began in his tongue, and slowly progressed to involve his head, neck, trunk, and lower extremities. His family noted episodes of severe diaphoresis and hyperpyrexia, but was unable to measure his temperature. Approximately 1 month prior to admission, neuroleptics were discontinued, and lithium and diazepam initiated; psychotic symptoms were well controlled on this regimen.

On admission, the patient was observed to have severe choreoathetoid movements involving all four extremities, spasms of his neck and back muscles, twisting movements of his neck and trunk, grimacing, and occasional grunting; EEG, CT scan, and ceruloplasm were normal. Serum LDH was slightly elevated. There was no family history of movement disorder. Lithium (900 mg/day; serum level 0.6 meq/litre) and diazepam (30 mg/day) were continued.

Reserpine was begun at 0.25 mg/day and was slowly increased over a 2-week interval to 1.25 mg/day. Initially he appeared to benefit, exhibiting a relative decrease in trunkal movements and having improved sleep. However, at doses of 1–1.25 mg of reserpine, his movements became more pronounced, and he developed transient blood pressure elevations to 170/110 mmHg, intermittent diaphoresis, tachycardia, and pallor. Lithium and reserpine were discontinued.

The patient developed intermittent stridor, dysphagia, and auditory hallucinations 5 days after the discontinuation of reserpine, and 3 days after discontinuation of lithium. He was considered to have acutely worsening TD, and was given two 50 mg doses of thioridazine and one 5 mg dose of haloperidol over 24 h. Following each dose, he fell asleep, and became free of movements for several hours. Within 4 hours after receiving haloperidol, he became extremely rigid, and his rectal temperature rose to 104°F. White blood count increased to 24 000, and marked elevations were noted in CPK, LDH, and SGOT. Bromocriptine (5 mg) was administered, producing a rapid (1.5 h) return to normal temperature and a significant reduction in rigidity.

The subsequent course was complicated by the development of acute renal failure secondary to rhabdomyolysis (CPK>99 999U), which required renal haemodialysis. Bromocriptine was continued at a dose of 15 mg/day. Unexplained temperature fluctuation, diaphoresis, and leucocytosis persisted intermittently for 7 weeks, and seemed to worsen whenever bromocriptine doses were withheld. Dyskinetic movements continued at the same level of severity as had been seen on admission, and were unaffected by a trial of lecithin and later increases in the bromocriptine dose to 27.5 mg/day. Three months after the onset of NMS, he was successfully weaned from bromocriptine and was discharged home.

Discussion

Rapid onset of fever, diaphoresis, muscle rigidity, and hallucinosis shortly after neuroleptic administration have been described here in a patient with TD. These symptoms, with the accompanying findings of elevated blood pressure, pallor, leucocytosis, liver function abnormalities, and elevated CPK, are consistent with NMS as described by Cope & Gregg (1983). Rhabdomyolysis and renal failure are not uncommon complications of this disorder (Eiser *et al* 1982). Rapid response to dopamine agonists is also characteristic of NMS (Mueller *et al*, 1983), and further supports the diagnosis in this case.

Our patient's initial symptoms were consistent with TD, and were severe enough to warrant an attempt at pharmacological intervention: reserpine may ameliorate some cases of TD (Klawans *et al*, 1980). Thioridazine and haloperidol were given on a trial basis when the movements continued to worsen, despite the withdrawal of reserpine; this was based on the assumption that we were dealing with worsening TD, and that more complete blockade of dopamine receptors would provide at least temporary palliation. Both of these interventions made sense for TD, but are likely to worsen NMS, which appears to be a syndrome of acute dopamine blockade (Mueller *et al*, 1983). Our assumption that we were dealing with TD alone kept us from pursuing the diametrically opposed, but appropriate strategy of administering a dopamine agonist instead of a dopamine antagonist. When NMS was finally recognised, bromocriptine was administered, with rapid improvement in temperature and muscle rigidity.

Neuroleptic malignant syndrome is usually precipitated by neuroleptics, but can also be induced by other treatments that cause diminished dopaminergic neurotransmission. In our patient, the full syndrome did not become manifest until after a dose of haloperidol, but premonitory signs of autonomic instability and increased muscle tone had actually begun a week earlier, when reserpine dose was increased to 1 mg/day. Thus, we believe that haloperidol simply hastened the process already set into motion by reserpine. Although reserpine itself has not previously been implicated in NMS, there has been a report of NMS associated with tetrabenazine (Burke *et al*, 1981), which has dopamine-depleting effects similar to those of reserpine. Dopamine depletion, which should take longer to reverse than dopamine receptor blockade, may have been the reason why vestiges of NMS persisted considerably longer in this patient than in cases induced by neuroleptics alone. Lithium may interact with haloperidol to produce a syndrome similar to NMS (Spring & Frankel, 1981), but it is less clear that lithium can be implicated in our case, since it had been discontinued 3 days prior to the onset of the full-blown syndrome.

Hot weather may contribute to the development of NMS, or to neuroleptic-related heat stroke, which may mimic NMS. Although our patient's symptoms occurred during the summer months, he was confined to an air-conditioned unit, thus diminishing the likelihood that heat was a causative factor. The findings of diaphoresis and marked increase in CPK are inconsistent with the diagnosis of neuroleptic-related heat stroke.

We cannot tell whether the TD and NMS were related or simply occurred together coincidentally. The reports by the family of transient diaphoresis and elevations of temperature in the months prior to admission suggest that his NMS actually had its onset in a milder form, prior to the severe episode we observed, and that its development paralleled the worsening of the TD. Phenomenologically, NMS appears more similar to acute dystonia than to TD. It is of interest that our patient's TD was atypical, not just in terms of its severity, but also in the preponderance of neck, arm, and trunkal dystonia. In this regard, our case bears some resemblance to those of tardive dystonia reported by Burke *et al* (1982). At present, it is not known whether tardive dystonia is truly distinct from TD in general, or whether it is related to NMS. However, it may be possible that our patient and those described by Burke *et al* (1982) suffered not from typical TD, but rather from a similar, but mechanistically distinct, neuroleptic-related disorder that in some instances can affect autonomic and temperature homeostasis as well as muscle tone.

Not all cases of TD may be at risk for development of NMS, but clinicians should be aware of the possibility of this complication. Reserpine should only be used judiciously in the treatment of TD and is perhaps best avoided in atypical cases. A history of unexplained hyperpyrexia or autonomic instability may help in the identification of those who are more vulnerable to full-blown NMS when exposed to sufficient dopamine blockade.

References

BURKE, R. E., FAHN, S., MAYEUX, R., WEINBERG, H. & WILLNER, J. H. (1981) Neuroleptic malignant syndrome caused by dopamine depleting drugs in a patient with Huntington disease. *Neurology*, **31**, 1022–1026.

——, ——, JANKOVIC, J., MARSDEN, C. D., LANG, A. E., GOLLOMP, S. & ILSON, J. (1982) Tardive dystonia: late-onset and persistent dystonia caused by antipsychotic drugs. *Neurology*, **32**, 1335–1346.

COPE, R. V. & GREGG, E. M. (1983) Neuroleptic malignant syndrome. *British Medical Journal*, **286**, 19–38.

EISER, A. R., NEFF, M. S. & SLIFKIN, R. F. (1982) Acute myoglobinuric renal failure. A conseqence of the neuroleptic malignant syndrome. *Archives of Internal Medicine*, **142**, 601–603.

KLAWANS, H. L., GOETZ, C. G. & PERLIK, S. (1980) Tardive Dyskinesia: Review and Update. *American Journal of Psychiatry*, **137**, 900–908.

MUELLER, P. S., VESTER, J. W. & FERMAGLICH, J. (1983) Neuroleptic malignant syndrome: Successful treatment with bromocriptine. *Journal of the American Medical Association*, **249**, 386–388.

SPRING, G. & FRANKEL, M. (1981) New data on lithium and haloperidol incompatibility. *American Journal of Psychiatry*, **138**, 818–821.

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Koro and Schizophrenia in Singapore

A case of Koro is described in an elderly person with associated inguinal hernia and schizophrenia. Its aetiology and origin are traced in Chinese culture, to the Chin Dynasty. A nosology for the classification of Koro Syndrome is proposed.

Koro is a culture-bound syndrome, characterised by a fear of shrinkage of the genitalia into the abdomen. Its cardinal manifestations are: a belief of retraction of the penis and impending death; a panic syndrome with fear, feeling of collapse, palpitations, sweating, breathlessness, and bodily spasms; complications arising as a result of remedial measures.

In Singapore, Gwee (1968; Koro Study Team, 1969) has traced the fascinating origin of Koro in Chinese culture. The disease was first described in the Yellow Emperor's Book of Medicine in 3000 BC. The cases described had a chronic course, but by the time of the Chin Dynasty, the illness was described as having acute onset, and was associated with sexual activity. In the New Collection of Remedies of 1834, Pao indicated that Koro could be precipitated after sexual intercourse or following exposure to 'cold' or raw food.

In 1969, the Koro Study Team and Ngui reported on an "epidemic" in Singapore. Cases appeared after rumours had spread about some men having Koro after eating pork from pigs innoculated against swine fever. The number of cases rapidly increased, and it became a common sight to see men appearing at admission rooms, with chop-sticks and other mechanical aids tied to their sex organs. The Koro Study Team reported on 450 cases, 95% being young Chinese males; most were of acute onset and brief duration, but 17% had recurrent attacks. Simple treatment measures, such as reassurance and suggestion, were all that was required.

An epidemic of such magnitude is unlikely to occur in Singapore again, due to the rapid modernisation since 1969; isolated cases are probably seen, but not reported. However, in Assam (Dutta *et al*, 1982), and Thailand, epidemics have been reported more recently. It is interesting that very few cases are associated with schizophrenia.

Classification

With a greater number of reports of Koro and Koro-like disorders in both Chinese and non-Chinese subjects, it may now be possible to attempt a classification of the syndrome.

Classical Koro

Koro is common in Chinese people, and has an indisputable cultural origin. It is short-lived, is not related to underlying psychiatric illness, but can be recurrent, and is treatable by simple measures such as reassurance. Epidemics can occur, if enough fear is generated by the spreading of rumours.

Koro grafted upon an underlying psychiatric illness

Koro has a natural history which is different from any underlying illness. It is most likely to be associated with anxiety states and Yap (1965) reported 14 out of 19 cases studied to be suffering from anxiety, but it has been reported also in depression, schizophrenia, and brief psychotic states. It has a cultural basis. Most cases of Koro fall into this category.

Koro as a symptom of mental illness

The Koro symptom is based on a delusion, or is the result of depersonalisation with possible accompanying psychotic states like schizophrenia, and is not culturally determined. It resolves when the underlying illness abates. This is more likely to occur in non-Chinese samples. When associated with mental illness simple measures such as reassurance and education may not be sufficient.

Koro associated with a schizophrenic illness is, however, rare. Yap (1965) described six cases, while a number of Koro-like states have been reported in non-Chinese schizophrenic subjects (Edwards, 1970; Cremona, 1981; Ang & Weller, 1984). We describe here a case of Koro superimposed on an underlying schizophrenic illness.

Case report

Mr X, a 75-year-old widower, emigrated from China to Singapore many years ago; he has one son, who is married with a child. Mr X is an introverted man, with few friends and associates, but fairly independent. At the age of 43, he lost his wife, soon after the birth of a daughter; having no means to care for this child, he had to agree to adoption. He retired from his job as a shop assistant in 1969; the following year, he became depressed and attempted suicide, but did not seek psychiatric treatment.

In 1972 (age 63), he developed a schizophrenic illness, characterised by delusions of persecution, directed at neighbours, and the media (he thought the radio was communicating his thoughts to the neighbourhood). He responded well to trifluoperazine as an inpatient, but relapsed in 1984, with persecutory ideas directed at the police and neighbours. He was treated with promazine as an outpatient. A week later, he appeared at the admission room at midnight, complaining that his genitalia were shrinking into his abdomen, after he had eaten bananas. He was acutely anxious, and feared that the condition could be lethal. As a result, he tied a piece of cloth to his penis to prevent further shrinkage. Physical examination was essentially normal, except for a reducible inguinal hernia; there had been no trauma to his sex organs. He was reassured and counselled about his Koro, and advised to continue with the antipsychotic medication. Despite this advice, however, he stopped the medication after a short period. Mr X's son and daughter-in-law were advised not to reinforce his symptoms, which ceased within a few weeks of onset. He was transferred to a home for the elderly, run by a Chinese Temple, in March 1985. After 6 months he was still deluded about his persecutors, but denied his Koro symptoms with embarrassment.

Over the years, he had become isolated, due to the death of his wife and separation from close neighbours. His vision had also deteriorated. With the appearance of a psychotic relapse, he became house-bound because of his paranoid fears. It was in this context that his Koro appeared. Relevant factors included both cultural beliefs and his physical and psychiatric health.

Discussion

This case had an associated inguinal hernia of a year's duration; its presence probably directed Mr X's attention to the genital area. The Koro Study Team (1969) reported also a case associated with a hernia. This patient also believed that bananas ('cold' food) precipitated the attack, which is consistent with Chinese cultural beliefs as during the Chin Dynasty, it was widely believed that Koro was precipitated either by sexual intercourse of by ingestion of raw or 'cold' food (Gwee, 1970). Though the patient had an underlying schizophrenic illness, many of the features of culture-bound Koro were evident; his psychosis continued after the Koro symptoms resolved, with a high level of anxiety when the psychosis reappeared. It is possible that the presence of anxiety associated with depersonalisation could have further facilitated the appearance of the Koro.

In younger samples, sexual conflicts and personality factors are aetiologically significant, in addition to cultural beliefs (Yap, 1965). The useful analysis by Berrios & Morley (1984) of reports of cases in non-Chinese patients showed that psychosexual conflicts were prominent, but in the elderly, these are unlikely to be relevant.

Though Koro is primarily a disorder of the young, it can nevertheless occur in the elderly: the oldest patient in the Koro Study Team was aged 70. In such cases other diagnostic possibilities such as local or systemic disease, CNS disorders or drug-induced states should be looked for, in addition to psychiatric illness.

References

ANG, P. C. & WELLER, M. P. I. (1984) Koro and psychosis. *British Journal of Psychiatry*, **145**, 335.

BERRIOS, G. E. & MORLEY, S. J. (1984) Koro-like symptom in a non-Chinese subject. *British Journal of Psychiatry*, **145**, 331–334.

CREMONA, A. (1981) Another case of Koro in a Britain. *British Journal of Psychiatry*, **138**, 180–181.

DUTTA, D., POOKAN, H. R. & DAS, P. D. (1982) The Koro epidemic in Lower Assam. *The Indian Journal of Psychiatry*, **24**, 370–374.

EDWARDS, J. G. (1970) The Koro pattern of depersonalisation in an American schizophrenic patient. *American Journal of Psychiatry*, **126**, 1171–1173.

GWEE, A. L. (1968) Koro — its origin and nature as a disease entity. *Singapore Medical Journal*, **9**, 3.

—— (1970) *Koro*, Singapore: Singapore Medical Association Publications.

HARRINGTON, J. A. (1982) Epidemic psychosis (Correspondence). *British Journal of Psychiatry*, **141**, 98–99.

KORO STUDY TEAM (1969) The Koro epidemic in Singapore. *Singapore Medical Journal*, 10(4), 234–242.

NGUI, P. W. (1969) The Koro epidemic in Singapore. *Australian & New Zealand Journal of Psychiatry*, **3**, 263.

YAP, P. M. (1965) Koro, a culture-bound depersonalisation syndrome. *British Journal of Psychiatry*, **111**, 43–50.

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A Standardised Assessment of Personality Disorder in Mental Handicap

Forty patients with mild or moderate mental handicap were assessed by two psychiatrists using the scale devised by Mann *et al*, 1981 which describes abnormalities of personality. Analysis by weighted Kappa showed excellent agreement for some items (introversion, hysterical explosive, sociopathic, anankastic and affective). This questionnaire may be useful in the evaluation of personality in the mentally handicapped.

The concept of personality disorder has always presented problems but most psychiatrists find it helpful to use it in their clinical practice. In the field of mental handicap abnormalities of personality can be very important and may be a crucial factor in determining whether an individual can survive independently in the community. There have been various clinical descriptions of personality disorders in mental handicap but few systematic studies, although Corbett (1979) estimated that 25.4% of adults in the Camberwell survey manifested personality disorder.

In general psychiatry there have been several reports describing methods of systematically estimating abnormal personality (Presly & Walton, 1973; Tyrer & Alexander, 1979; Mann *et al*, 1981). Mann *et al* reported on a standardised assessment of abnormal personality using an observer report and showed this to be reliable in a preliminary study in individuals of normal intelligence. The scale contains the personality types in Section 301 of the ICD (1977) with the addition of 'anxious' and 'self conscious'; the personality characteristics 'introversion' and 'extraversion' are also included. The aim of the present study was to evaluate the use of this scale in the assessment of personality disorder in the mentally handicapped.

Method

Forty mentally handicapped patients in Strathmartine Hospital, Dundee were included. The patients were selected randomly from 144 patients with mild or moderate mental handicap in eight mainly long-stay wards of the hospital: 18 males and 22 females were included and the mean age was 48.5 years (range 20–80 years).

Only individuals with mild (14) and moderate (26) mental handicap were studied as it seemed likely that this questionnaire would be less applicable to more handicapped individuals. The diagnosis was taken from the case records and based on the 9th Revision of the International Classification of Disease categories of mild (IQ 50–70) and moderate mental retardation (IQ 35–49). Patients with acute psychotic illness were also excluded as personality may be distorted, particularly by schizophrenia.

The patients were assessed by interviewing the trained nurses in charge of the ward who knew them well. The nurse involved was usually the Charge Nurse with an RNMD qualification and sometimes also an RNM. The two raters were together when the nurses were interviewed: one of the raters (hereafter described in the text as the principal rater) conducted the interview, whilst the other rater acted as an observer. The two raters then assessed the interview independently using the scale described by Mann *et al* (1981).

The schedule of Mann *et al* was designed to reflect the clinical approach of a psychiatrist interviewing a patient's informant. After a general introduction there is an exploratory section where the description of personality is ascertained, followed by seven standard questions if necessary. If certain 'key words' have been used, a series of questions appropriate to one or more personality types is used. The interviewer then records the type and grading of personality. A grade 1 indicates that the personality can be matched by one of the types and a grade 2 that the description indicates that the patient is very unusual or handicapped by reason of this personality. Features of more than one type may be present but the interviewer should establish which is the more dominant.

The results were analysed by the weighted Kappa method (Cohen, 1968) which provides a measure of agreement between raters. The advantages of this method are that it takes into account chance levels of agreement by the judges and allows credit for partial rater agreement.

Results

Table I describes the personality disorders and characteristics scored by the principal rater, together with the Kappa results using the scores of both raters. Agreement is recorded as being excellent (Kappa values over 0.75) for some items (introversion, hysterical, explosive, sociopathic, anankastic, affective) and fair (Kappa values 0.40–0.59) for others (self-conscious, extraversion) (Cicchetti & Sparrow, 1981). The types schizoid, paranoid, anxious and asthenic were only rated once or not at all.

Ten of the 40 patients were not rated by the principal rater as showing any particular personality types. Of the remainder 17 were rated as having one personality type, 11 as showing two and two as showing three personality types. Mann *et al* (1981) recommended that where this occurs in clinical practice, the dominant personality type should be identified and recorded. Since the present study

TABLE I

Personality type 40 patients	*Grade I*	*Grade II*	*Total grade I and II*	*Weighted kappa*
	(principal rater)			
Extraversion	4	0	4	0.5460
Introversion	11	1	12	0.7829
Self-conscious	2	0	2	0.4748
Schizoid	1	0	1	*
Paranoid	0	0	0	*
Affective	3	1	4	0.7780
Anankastic	2	0	2	1.00
Anxious	0	0	0	*
Asthenic	0	0	0	*
Sociopathic	0	2	2	1.00
Explosive	10	2	12	0.9030
Hysterical	4	2	6	0.9115

*Insufficient ratings for analysis
The principal rater is the rater conducting the interview although results recorded by both raters are used in calculating the weight Kappa.

was aimed at assessing reliability, however, we used all the available data to provide the broadest possible base from which to draw conclusions.

Discussion

The present study only assessed inter-rater reliability and it is not possible to comment on test–retest reliability or validity. The fact that the same interview was used by both raters for practical reasons is also a possible source of criticism, although the ratings were made independently.

The standardised assessment of personality described by Mann *et al* (1981) would seem to be useful for the assessment of patients with mild and moderate mental handicap. We did not attempt to use it for individuals with severe or profound handicap and have doubts as to how well the usual personality types could be defined in this group. The method involved interviewing somebody who knew the patient well, (a trained nurse), and provided suitable data. It is very doubtful if these patients would be able to give any useful description of their own personality characteristics and an observer-based assessment procedure is therefore particularly appropriate in this population.

Many of the usual personality types were encountered although they did not always have quite the characteristic form expected in adult psychiatry. Some types did not occur at all (paranoid, anxious, asthenic) in our sample and this is in contrast with the psychiatric hospital group reported by Mann *et al* (1981). However, the explosive and hysterical types were relatively common in our hospital sample, and this may relate to the reasons for admission of these particular patients. It is not possible to draw firm conclusions about personality disorders in mentally handicapped individuals in the community from the present study.

We conclude that Mann's standardised assessment of personality questionnaire is likely to be a useful and substantially reliable instrument for rating personality in mentally handicapped patients and that it could be used in further investigations.

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References

COHEN, J. (1968) Weighted Kappa: nominal scale agreement with provision for scaled disagreement or partial credit. *Psychological Bulletin*, **70**, 213–220.

CICCHETTI, D. V. & SPARROW, S. A. (1981) Developing criteria for establishing interrater reliability of specific items: Application to assessment of adaptive behaviour. *American Journal of Mental Deficiency*, **86**, 127–137.

CORBETT, J. A. (1979) Psychiatric morbidity and mental retardation. In *Psychiatric Illness and Mental Handicap* (eds F. E. James & R. P. Snaith). London: Gaskell.

INTERNATIONAL CLASSIFICATION OF DISEASES (1977) HMSO London.

MANN, A. H., JENKINS, R., CUTTING, J. C. & COWEN, P. J. (1981) The development and use of a standardised assessment of abnormal personality. *Psychological Medicine*, **11**, 839–847.

PRESLY, A. S. & WALTON, H. J. (1973) Dimensions of abnormal personality. *British Journal of Psychiatry*, **122**, 269–276.

TYRER, P. & ALEXANDER, J. (1979) Classification of personality disorder. *British Journal of Psychiatry*, **135**, 163–167.

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A Case of 'Lethal Catatonia' in a 14-year-old Girl

A case of apparent 'lethal catatonia' in a 14-year-old girl is described; similarities with earlier descriptions are identified, although the precise cause of death in this case remains somewhat obscure.

In spite of the fact that catatonia is generally regarded as having a better prognosis than other forms of schizophrenia, attention has been drawn for many years to the existence of a severe form, proceeding to death in some cases.

In 1832, Calmeil first described a stormy course in some catatonic patients, with catatonic excitement passing into profound stupor, hyperthermia, and sudden death, for which no cause was found at autopsy. In 1934 Stauder described 27 cases of fatal catatonia, half of whom were young people with a positive family history of schizophrenia, normal premorbid personalities, an acute onset, and short history of mutism, followed by 4 or 5 days of motor excitement, exhaustion, and death. He also reported skin bleeding and extreme acrocyanosis, and speculated about disorders in circulation and haemopoeisis. Subsequent reports (Billig & Freeman, 1944; Shulack, 1946; Aronson & Thompson, 1950; Laskowska *et al* 1965) also described the period of hyperpyrexia and non-specific autopsy findings. Shulack described successful treatment and recovery in nine cases where diagnosis was made early, using sedation and intravenous rehydration, with correction of electrolyte imbalance. Fish & Greiner (1960) reported a single case successfully treated by hypothermia with autonomic blockade.

Herman *et al* (1942) reviewed experimental studies on catatonia, and analysed 12 cases of catatonia in non-schizophrenic subjects. They considered that catatonia is a neurological phenomenon, the mechanism for which resides in the posterior part of the hypothalamus, with the neocortex having a controlling influence. This cortical influence was believed to be disturbed by the overwhelming toxic factors of organic cortical brain disease and also by psychological influences. In their series the catatonic symptoms were identical with those found in schizophrenia, but they suggested that the clouded sensorium of organic brain disease differentiated them.

Other writers (Weinstein *et al*, 1955; Himmelhoch *et al*, 1970; Raskin & Frank 1974) have reported cases of viral encephalitis presenting as acute schizophrenia, with unremarkable previous and family histories, and a short history of 'odd' or bizarre behaviour, sometimes proceeding to the development of catatonic stupor. They also reported the subsequent development of positive, although non-specific neurological signs and abnormal electroencephalographic recordings. Raskin, whose single case was finally diagnosed as herpes encephalitis, listed five features distinguishing between functional catatonic stupor and organic conditions: different responses of the pupils and eyelids, absence of roving eye movements in functional stupors, normal respiration, absence of abnormal motor movements such as seizures, asterixis, myoclonic movements, and the production of normal, 'schizophrenic' or depressive speech by amobarbital interview. Himmelhoch *et al* also commented on the rapid fluctuations in mental state of their eight cases and the importance of noting and correctly interpreting positive neurological signs. Most reported cases of encephalitis show pyrexia at some stage, but examination of the cerebrospinal fluid is often normal.

A single case of catatonia culminating in sudden death in a 14 year-old girl is described below; it demonstrated some of the features in the previous literature.

Case report

A. R. was admitted to a regional adolescent psychiatric unit as an emergency, at the age of 14 years 9 months, with a 9-day history of acutely disturbed behaviour. A longer history of more subtle changes emerged later.

Family background

She was the eldest of three clever daughters of professional parents. Her father was from a Jewish family, had suffered from 'idiopathic hepatitis' for some 12 years, and was also said to be cyclothymic, having received treatment for depression in the past. The mother was not Jewish and she was healthy. The only other family history of psychiatric disorder was a sister of the maternal grandfather, who was known to have spent some two years in a psychiatric hospital in her early twenties, with an illness characterised by mutism.

Past history and premorbid personality

A's birth and development was entirely normal. Her only previous illnesses were appenditicis with peritonitis at the age of 7 years and a mild episode of glandular fever at 12 years. The menarche was at 13 years, and periods had been regular, but she had tended to be overweight since then, and dieted intermittently. She had always been successful at school, had a small group of close friends, and was generally regarded by family and teachers as a mature, considerate, self-sufficient, and well-behaved girl.

History of present illness

A's behaviour had begun to change about 9 days before admission, with a series of inexplicable actions, including leaving for school half-dressed, ransacking drawers, and rowing with a neighbour. Her mood varied from elation to anger. For 6 days, she became increasingly withdrawn, with less and less speech, and an increasing tendency to rocking and other self-stimulating behaviour. She was restless, slept poorly, and ate ravenously.

On admission, physical examination was normal, except that she was overweight and her tongue a little dry. Her mental state was noted to be drowsy, passively uncooperative, rocking at times, negativistic, but with delayed response to some requests, e.g. to put out her tongue. She was perseverative with some movements, but there was no convincing waxy flexibility. She was mainly mute, but once echoed the two last words of a question. She stared vacantly, occasionally looking frightened or perplexed. It was thought most likely that the diagnosis was catatonic schizophrenia, but an organic psychosyndrome had to be excluded. The latter possibility was reinforced by her initial deterioration, which included incontinence of urine, attempting to feed herself with her hands, and several incidents of banging her legs or head hard enough to produce extensive bruising. She remained predominantly mute, but with occasional echoing or bouts of screaming. A paediatric neurologist who examined her 2 days after admission noted flexor spasms like a form of primitive seizure, and found all her reflexes to be brisk but no focal neurological signs.

Investigations and results were as follows: EEG—generalised dysrhythmia of non-specific type; CT scan—normal CSF—protein 0.6 g/litre glucose 4.0 mmol/litre, 4 RBC, 2 WBC, no bacterial growth; serum and CSF treponemal HA, cardiolipin WR and WDRL all negative; white cell and plasma enzymes—no deficiency; immunology—ANF negative; smooth muscle, mitchondria and reticulin positive; Hb 14.9 g—WBC 6.1 × 10 with normal differential, ESR 5 mm; blood chemistry—normal except alkaline phosphatase 100 *IU*/litre; LD 515 *IU*/litre, ALT 69 *IU*/litre; virus serology: evidence of past measles only; CSF antibody tests (later report) excluded any of the viruses for which tests are currently available.

For most of the 4 weeks between admission and death, she remained very disturbed, restless, sleeping only for short periods, frequently incontinent of urine and sometimes of faeces, and often removing some or most of her clothing. She frequently banged and bruised her limbs, and continued to stimulate herself sexually. Most of the time, she was mute but occasionally uttered single words or short phrases, or screamed. Her apparent mood varied from apathy to fear or elation. The facial grimacing described as *Schnauzkrampf* was noticed on several occasions, as were other mannerisms such as pointing her index finger at others. There was an interlude of an apparent brief return to normality, when she was sedated with 10 mg diazepam intravenously for the CT scan and, after awakening, for 15 min, appeared normally orientated and rational, before reverting to her former mute state.

At this stage, a presumptive diagnosis of catatonic schizophrenia was made, and treatment with chlorpromazine in increasing doses instituted; at the time of her death, she had been receiving 100 mg *t.d.s.* for one week. During the fourth week after admission, there was slight improvement with less restlessness, a better sleep pattern and more copious speech, with occasional coherence.

Four weeks after admission, she got out of bed at about midnight, having previously slept for 2 hours; as she tried to get back in, she was observed to fall to the floor. Nursing staff noticed she was cyanosed, and found her to be pulseless. Attempts to resuscitate her were continued for 40 min, during which vomit was extracted from the larynx by suction; there was no response, and death was confirmed at 1.00 am. A post mortem next day by a Home Office pathologist revealed bronchi and bronchioles full of vomit, with congestion of the lungs, a small subdural haematoma in the middle fossa, and blood on the surface of both occipital lobes. He concluded that death was due to inhalation of vomit, probably secondary to intracranial haematoma produced by repeated acts of self-injury. At a subsequent inquest, the coroner accepted the pathologist's opinion, and recorded a verdict of accidental death. Nine months later, a neuropathologist reported on further studies on the brain as follows: "the only changes that were seen were those of perivascular lymphocytic cuffing and cell changes compatible with a viral encephalitis."

Discussion

The normal family background, relatively normal family history, and rapid onset of severe symptoms and deterioration in this case seemed to indicate an organic psychosyndrome. This diagnosis was supported by the development of primitive seizures and the non-specifically dysrhythmic EEG, but not by any other investigations, including examination of the cerebrospinal fluid. The brief return to apparent normality after intravenous diazepam for the CT scan also made an organic disorder less probable. However, the eventual neuropathological suggestion of a viral encephalitis seemed an acceptable explanation of the history, clinical course, and investigation results. The cause of death seems to be a separate issue: to those immediately present her death appeared to be a very sudden one, suggesting a cardiac or overwhelming cerebrovascular cause. Staff involved in the attempts to resuscitate her found vomit in the upper respiratory passages but could not judge how long it had been there. Although the pathologist's findings adequately accounted for the death, the clinical team were unsatisfied. Neither had there been any evidence of the hyperpyrexia and extreme restlessness prior to death described by early writers. Her temperature was never appreciably raised, and if anything, she had seemed quieter and more responsive in the day or two before she died.

One feature reminiscent of Stauder's descriptions was the extensive bruising of her legs and arms. On the whole, this was thought to be consonant with her repetitive self-injury, and although it was planned to investigate for any bleeding or clotting disorder, she died before this could be done.

There remained several unanswered questions: (1) Although the later suggestion of a viral encephalitis seemed to explain most of the clinical features, is it rendered less probable by the brief return to apparent normality after intravenous diazepam? (Herman *et al* described a similar response to intravenous sodium amytal in their organic cases). (2) What was the real cause of the sudden death? (3) What might have been the longer-term psychiatric outcome, had she lived?

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References

Aronson, M. J. & Thompson, S. V. (1950) Complications of acute catatonic excitement. *American Journal of Psychiatry*, **107**, 216–220.

Billig, O. & Freeman, W. T. (1944) Fatal catatonia. *American Journal of Psychiatry*, **100**, 633–638.

Calmeil, (1832) cited by Ladame, C. (1919) Psychose aigue idiopathique ou foudroyante schweiz. *Archiv fur Neurologe und Psychiatrie* **5**, 3.

Fish, K. J. & Greiner, A. (1960) Acute lethal catatonia treated by hypothermia. *Canadian Medical Association Journal*, **82**, 630–634.

Herman, M., Harphan, D. & Rosenblum, M. (1942) Nonschizophrenic catatonic states. *New York State Journal of Medicine*, **42**, 624–627.

Himmelhoch, J., Pincus, J., Tucker, G. & Petre, T. (1970) Subacute encephalitis: behavioural and neurological aspects. *British Journal of Psychiatry*, **116**, 531–538.

Laskowska, D., Urbaniak, K. & Jus, A. (1965) The relationship between catatonic delirious states and schizophrenia in the light of a follow up study (Stauder's Lethal Catatonia). *British Journal of Psychiatry*, **111**, 254–257.

Raskin, D. E. & Frank, S. W. (1974) Herpes encephalitis with catatonic stupor. *Archives of General Psychiatry*, **31**, 544–546.

Shulack, N. (1946) Exhaustion syndrome in excited psychotic patients. *American Journal of Psychiatry*, **102**, 466.

Stauder, K. H. (1934) Die todliche Katatonie. *Archiv für Psychiatrie*, **102**, 614–634.

Weinstein, E. A., Lina, L. & Kahn, R. L. (1955) Encephalitis with a clinical picture of schizophrenia. *Journal of Mount Sinai Hospital*, **21**, 341–354.

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Cotard's Syndrome in a Mentally Handicapped Man

A mentally handicapped man with Cotard's syndrome is described, and the usefulness of electroconvulsive therapy in his management discussed. Even the more uncommon psychiatric syndromes such as this can be found in people with lower intellectual functioning, with clinical features little modified.

Delire de negation was first described by Cotard in 1880, in a presentation to the Société Medico-psychologique in Paris and his name was given to the syndrome by Seglas (1897). Its chief characteristic is a denial by the afflicted person of his own existence, that of part of his body, or in extreme cases, his whole environment. It has long been debated whether this is a clinical entity in its own right, or a complex of symptoms which may be associated with any of a number of psychiatric illnesses, most notably depression. A comprehensive review of the literature has been given by Enoch & Trethowan (1979).

Although there has been an increased interest of late in psychiatric illnesses in the mentally handicapped, with some accounts of unusual syndromes, e.g. Gilles de la Tourette syndrome (Burd & Kerbeshian, 1985; Reid, 1984), there has been no description of the phenomenon of Cotard's syndrome in a mentally handicapped person. I here describe such a case.

Case report

The patient is a 67-year-old man who has been resident in a large hospital for the mentally handicapped for the last 27 years; he was admitted after the death of his mother, as it was felt that he could not manage to live an independent existence. At admission, his IQ was assessed as 35. Although he has always been well settled in hospital, he has, for almost his entire time there, been prone to bouts of depression, frequently accompanied by bizarre ideas. During his first recorded episode, he stated that he had "no head" and "eyes only sometimes"; on another occasion, he said that the floor was "soft". His personality was described as hypochondriacal, with a tendency to complain about minor ailments.

His most recent episode began when he was observed by nursing staff to be "confused". After an initial apparent recovery, he once again began to deteriorate, lost interest in his hospital activities, and took to bed, complaining of abdominal pain; when asked why he would not get up, he could not elaborate, and answered "I don't know" to most questions. He began to refuse food, and told nurses that he had no body, no legs, and should be in the hospital mortuary. At this stage, because of enfeeblement due to his poor dietary intake, he was transferred to the sick ward. He continued to express nihilistic ideas, stating that his bowels were missing, and to refuse food and fluids; when asked why he was doing this, he said that he was dead, and that, as he was dead, he should be in the hospital mortuary "where all the dead people go". Asked "What would you do there?", he replied "Go into the fridge". At about this time, it was discovered from discussions with ward staff that a friend of his—a man in another ward—had recently died.

Despite two courses of antidepressants, there was no significant improvement in his condition. (Mianserin was initially administered, up to a maximum dose of 30 mg mane and 60 mg nocte. As this was ineffective, it was replaced after 7 weeks by nomifensine 25 mg *t.d.s.*) It was therefore decided, because of his declining physical health, to commence a course of electroconvulsive therapy (ECT) which, on a previous occasion, had proved effective in resolving a similar episode. Nomefensine was continued during and after ECT. Six treatments were given. The response was slow, but positive; he continued to express delusions, and on a few occasions, was found near the mortuary, again voicing ideas that he was dead and should be in there. At the end of the course, he was eating again, but was still quite taciturn and withdrawn. He then gradually returned to his premorbid state—began talking to staff and other residents, and participating in hospital activities. At this point, he returned to his own ward where he continued to make gains, and in fact became slightly elated for a short period, with mild disinhibition and expansive good humour.

Discussion

The development of a firmly held delusional belief, clearly expressed, in a mentally handicapped man with limited powers of communication, is a phenomenon worthy of note in itself, and the manner of its development bears consideration: an initial stage of poor concentration and apathy was followed by the gradual decay in his sense of personal integrity. His concern with abdominal pain characterised the early phase; giving way to a more profound disintegration, with first a denial of parts of his body and finally a conviction of his own death, which was of sufficient strength and coherence for him to act on it, and ask to be treated as dead. Interwoven with these symptoms were those of a depressive illness, with withdrawal, anorexia, depressed mood, and weight loss.

The aetiology of his illness may be seen in a number of ways. It is well recognised that, in the more severely mentally handicapped, an organic component is likely to be important in the production of the handicap, and Cotard's syndrome has also been associated with organic brain abnormality (Anderson, 1964). An association with hypochondriasis in Cotard's syndrome was also recorded by Anderson (1964). This patient had been described as being of a hypochondriacal personality, but the possibility that his symptomatology was related to a grief reaction is also a strong one. On the two occasions of his most severe episodes of nihilistic delusions, there had been a recent death of a close friend in the hospital. It seems very probable that his premorbid personality structure, his recent experience of death at close quarters, and a more concrete comprehension of events due to his intellectual handicap predisposed him to decompensate in the manner in which he did.

Antidepressants of various types proved ineffective in his earlier management, in spite of their having been tried in adequate doses over appropriate lengths of time. However, ECT produced a more satisfactory response, with an eventual full resolution of his pathological symptoms; it was also noted that there appeared to be a response to the use of minor tranquillisers, in the form of short-acting benzodiazepines, which seemed to bring about a reduction in his agitation, with an associated improvement in mood. The usefulness of ECT in the syndrome was recorded by Enoch & Trethowan (1979), and it is of interest that its effectiveness in a mentally handicapped subject was similar to that in normal subjects.

This case demonstrates once more that it is possible to find presentations of even the more uncommon psychiatric syndromes in mentally handicapped people, with features little modified by their lower intellectual functioning. It also underlines the fact that just as the symptoms can be of a relatively conventional nature, so also can be the treatment: ECT was as useful in this case as it has been in people of normal intelligence.

References

ANDERSON, E. W. (1964) *Psychiatry*, London: Balliere, Tindall.

BURD, L. & KERBESHIAN, J. (1985) Tourette syndrome, atypical pervasive developmental disorder and Ganser syndrome in a 15-year-old visually impaired, mentally retarded boy. *Canadian Journal of Psychiatry*, **30**, 74–76.

ENOCH, M. D. & TRETHOWAN, W. M. (1979) Cotard's syndrome. In *Uncommon Psychiatric Syndromes*. Bristol: John Wright.

REID, A. H. (1984) Gilles de la Tourette syndrome in mental handicap. *Journal of Mental Deficiency Research*, **28**, 81–83.

SEGLAS, J. (1897) *La Delire de Negation*, Vol. 1. Paris: Masson.

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Senile Dementia and Parietal Lobe Dysfunction

Two samples of patients suffering from senile dementia were administered the Kew tests for parietal lobe function and aphasia. No evidence emerged of any clinical heterogeneity; parietal lobe test performance was found to be more a function of degree of impairment rather than of age.

Interest in the clinical differentiation of patients with senile dementia has a long history. Several earlier writers had suggested a distinction between 'simple' or benign senile dementia, and a more clinically impaired or symptomatic type (cf. Bleuler, 1928, Schenk, 1955). As growing evidence emerged of similar underlying neuropathology in presenile Alzheimer's disease and senile dementias, however, a clinical unity has been drawn (cf. Rossor, 1982). Nevertheless, interest has been maintained in possible heterogeneity in both clinical manifestations, aetiology and prognosis within the primary degenerative dementias of Alzheimer type, especially with regard to differences in age and focal cortical signs (cf. Seltzer, 1984).

A much quoted study was reported by McDonald (1969) who distinguished between type A and type B senile dementia patients, on the basis of signs of parietal lobe dysfunction. Type A referred to patients making more than one error on a six-item 'parietal' scale, whom McDonald found to be younger and yet to have a significantly enhanced mortality. In a later paper, Hare (1978) claimed that using McDonald's measures, patients who showed only memory impairment and disorientation but no parietal signs had quite favourable outcomes, unlike the clear dementias who had poor outcomes (and parietal signs). Roth (1980) has argued, however, that "focal features . . . after the age of 70 can rarely be given the same status as the parietal lobe phenomena found . . . in the fifth and sixth decades".

While not employing McDonald's measures, other studies have examined different clinical features in senile dementia in relation to subsequent mortality, and two studies have demonstrated a relationship between 'temporoparietal' signs and enhanced mortality (Kaszniak *et al*, 1978; Naguib & Levy, 1982). At the same time such parietal lobe features as drawing ability and such temporal lobe features as nominal aphasia have been shown to correlate strongly with measures of severity of cognitive impairment within a population of senile dementia patients, supporting Roth's comment that focal features do not represent unique clinical phenomena in these cases, and thus may not be useful bases for a meaningful source of clinical heterogeneity (Skelton-Robinson & Jones, 1984; Moore & Wyke, 1984).

In neither McDonald's nor Hare's papers are there signs to exclude deliberately cases whose clinical features may have suggested a vascular aetiology to the dementia. In attempts to replicate McDonald's findings it would seem necessary (as he recognised at the time) to try and exclude possible cases of vascular (multi-infarct) dementia, and indeed to minimise the effects of physical health and of long-term institutionalisation. The present report describes data from two studies employing McDonald's Kew test, on patients selected to avoid the above complicating factors.

Method

Subjects

The first group of patients were seen and tested by E. S. using the Kew Cognitive Map. They were 20 female patients aged 65 or over attending psychogeriatric day hospitals or recently admitted to a psychogeriatric unit. All had a clinical diagnosis of senile dementia, no psychiatric history before the age of 65 years and none of the usual indices of possible multi-infarct dementia (i.e. no history of stroke, no focal neurological signs, no history of hypertension, no peripheral atherosclerosis). Laboratory investigations had not revealed any evidence of associated physical disorders. Mean age of this sample was 80.1 ± 4.6 years (range 70–87 years).

The second group of patients were seen and tested by R.O'C. using the parietal and aphasia scales of the Kew Map. Eighteen patients were selected on the basis of being in good health, attending a psychogeriatric day hospital while living at home, having a clinical diagnosis of senile dementia, no previous psychiatric history, age over 65 years, and a Hachinski index of 4 or less (Hachinski *et al*, 1975). Mean age of this sample was 78.3 ± 5.5 years (range 66–86 years).

All the patients had at least a 6-month history of mental deterioration and forgetfulness, all could repeat at least three digits forward, and all were capable of some degree of self-care. It is very difficult to date accurately the onset of dementia; however, no subjects with obvious presenile dementia were included in the analysis.

Measures

Both groups were tested on the 'aphasia' and 'parietal' subscales of the Kew Cognitive Map (McDonald, 1969). The Kew Aphasia test requires the subject to (1) name a wristwatch, (2) name the wrist strap or band, (3) name the buckle or clasp, and to state what the following appliances are used for: (4) refrigerator, (5) thermometer, (6) barometer. The Kew Parietal test was not described in detail by McDonald (1969). The version used in the present study required the subject to (1) show left hand on request, (2) touch left ear with right hand, (3) identify an object (key or coin) by touch alone, (4) correctly identify finger or hand when touched, (5) demonstrate normal 2 point discrimination, (6) construct a square with matches.

In both groups, apart from the Kew parietal and aphasia scales, the patients also completed other psychological tests, including a mental status test. The first group completed a 10-item extension of the Kew 'memory scale', the second the 12-item Information/Orientation scale of the C.A.P.E. (Pattie & Gilleard, 1979). Given the equivalence of most mental status test measures, the differences in these two tests seemed unlikely to be of significance (cf. Gurland, 1980).

Results

Table I presents the cross-tabulation of the aphasia and parietal scales. The rank order correlation between the two scales is +0.70, $P<0.001$, indicating a significant association between 'temporal lobe' and 'parietal lobe' signs. However, while patients who do badly on the aphasia test may still make no errors on the parietal test, the reverse is not the case, suggesting either that the appearance of 'parietal' signs is a later, more severe index of deterioration or that the test items in the aphasia scale are simply more difficult to perform.

When the sample is split according to McDonald's 'A' and 'B' types (A = those scoring more than 1 error on the parietal scale, B = those scoring 1 or less), and their mean

TABLE I

Cross-tabulation of Kew Aphasia and Parietal Scale Scores

Kew Parietal Scale	*0–1*	*2–3*	*4–5*	*6*	*Total*
Kew Aphasia Scale					
0–1	2	1	3	1	7
2–3	0	2	9	2	13
4–5	0	1	2	11	14
6	0	0	0	4	4
Total	2	4	14	18	38

ages compared, no differences emerged. Group A X age = 79.0 years, $n = 13$; group B X age = 79.3 years, $n = 25$. In the first group of patients the correlation between age and parietal score was $\rho = -0.077$ and in the second group $\rho = 0.085$, both non-significant. Rank order correlations between the two samples' mental status test scores and aphasia scores were: $\rho = +0.63$ ($n = 18$) and $+0.59$ ($n = 20$); between mental status test scores and parietal scores: $\rho = +0.59$ ($n = 18$) and $+0.16$ ($n = 20$). The latter correlation is not significant: all others are significant at $P < 0.01$.

Discussion

The findings do not support McDonald's qualitative distinction between a younger parietal lobe-impairment group and an older benign parietal lobe-intact group of patients diagnosed as senile dementia. Rather the study suggests a general relationship between existing deterioration in mental status and the accumulation of 'cortical' deficits. (The insignificant correlation between mental status test score and parietal score in Group 1 is an exception.) Since 'parietal' signs seem to occur later than 'temporal lobe' signs, it might be expected that their presence will augur an increased probability of dying in the near future. Of course this assumed 'sequence' of deterioration may reflect the different level of difficulty of the items in the 'parietal' and 'aphasia' tests rather than the true progression of dementia. However, recent evidence (Colgan, 1985) seems to confirm that level of mental deterioration reflected in impaired mental status performance is of equal if not greater prognostic significance than 'parieto-temporal' impairment scores in predicting mortality within a dementing population.

The age differences that were observed in McDonald's study may be explicable when those factors leading elderly patients to be admitted into long-term care are considered. Thus Green & Timbury (1979) have reported that their day hospital patients who were married and subsequently institutionalised were behaviourally more deteriorated than similar patients living alone. Since older patients, especially older women, are more likely to be living alone, this may lead to their being less deteriorated on admission—findings which have indeed been noted from behaviour ratings (Gilleard, 1978).

It is possible that, as Roth (1980) suggests, in presenile dementia such cortical localising signs may be significantly more frequent and distinctive compared with senile dementia patients. Nevertheless, empirical studies such as those reported by Pearce & Pearce (1979) have not reported any greater frequency of 'dysphasia, dyspraxia or agnosia' between pre-senile and senile groups. Similar conclusions to those indicated here of a global progression in the development of cognitive deficits in dementia have been reached by Reisberg and his team (Reisberg, 1984). The weight of empirical evidence does not at present provide much support for any clear-cut clinical heterogeneity in the primary (non-vascular) dementias.

Finally, it may be argued that the failure to replicate McDonald's findings reflects the inadequacies of the Kew Cognitive Map, rather than the unimportance of parietal lobe dysfunction in differentiating subtypes of senile dementia (witness the evidence from CT regional density studies, such as those reported by Colgan, 1985). Whilst this may be so, the burden of proof remains for investigators to show that clinical signs of parietal lobe impairment do indeed contribute unique information concerning the course and nature of senile dementia, rather than merely reflecting the severity of deterioration.

References

Bleuler, E. (1928) *Textbook of Psychiatry*, 2nd ed. London: Longmans.

Colgan, J. (1985) Regional density and survival in senile dementia. *British Journal of Psychiatry*, **147**, 63–66.

Gilleard, C. J. (1978) An investigation into the nature of behavioural change in the dementias of old age. Unpublished Ph.D. thesis, University of Leeds.

Green, J. G. & Timbury, G. C. (1979) A geriatric psychiatry day hospital service; a five year review. *Age and Ageing*, **8**, 49–53.

Gurland, B. J. (1980) The assessment of the mental health status of older adults. In *Handbook of Mental Health and Aging*, (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Hachinski, V. C., Iliff, L. D., Zilhka, E., du Boulay, G. H., Marshall, J., Ross Russell, R. W. & Symon, L. (1975) Cerebral blood flow in dementia. *Archives of Neurology*, **32**, 632–637.

Hare, M. (1978) Clinical checklist for the diagnosis of dementia. *British Medical Journal*, **ii**, 266–267.

Kaszniak, A. W., Fox, J., Gandell, D. L. & Ramsay, R. G. (1978) Predictors of mortality in presenile and senile dementia. *Annals of Neurology*, **3**, 246–252.

McDonald, C. (1969) Clinical heterogeneity in senile dementia. *British Journal of Psychiatry*, **115**, 267–271.

Moore, V. & Wyke, M. A. (1984) Drawing disability in patients with senile dementia. *Psychological Medicine*, **14**, 97–105.

Naguib, M. & Levy, R. (1982) Prediction of outcome in senile dementia; a computed tomography study. *British Journal of Psychiatry*, **140**, 263–267.

Pattie, A. H. & Gilleard, C. J. (1979) *Manual for the Clifton Assessment Procedures for the Elderly (CAPE)* Sevenoaks: Hodder & Stoughton Educational.

Pearce, J. M. S. & Pearce, I. (1979) Nosology of Alzheimer's disease. In *Alzheimer's Disease: Early Recognition of Potentially Reversible Deficits*, (eds A. I. M. Glen & L. J. Whalley. Edinburgh: Churchill Livingstone.

Reisberg, B. (1984) Commentary on "Is Alzheimer's disease inherited?" by Folstein and Powell, *Integrative Psychiatry*, **2**, 171–173.

Rosser, M. N. (1982) Dementia. *Lancet*, **i**, 1200–1203.

ROTH, M. (1980) The diagnosis of dementia in late and middle life. In *The Epidemiology of Dementia*, (eds J. A. Mortimer & L. M. Schuman). Oxford: Oxford University Press.

SCHENK, V. W. D. (1955) *Folia Psychiatrica et Neurologica Neerland*, cited by Corsellis, J. A. N. & Meyer, A. (1959) Aspects of neuropathology in relation to mental disease. In *Recent Progress in Psychiatry*, (eds G. W. T. H. Fleming & A. Walk. Vol. 3, London: Churchill.

SELTNER, B. (1984) Commentary on 'Is Alzheimer's disease inherited?" by Folstein and Powell *Integrative Psychiatry*, **2**, 170–171.

SKELTON-ROBINSON, M. & JONES, S. (1984) Nominal dysphasia and the severity of senile dementia. *British Journal of Psychiatry*, **145**, 168–171.

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Brain Imaging in a Case of Capgras' Syndrome

A patient developed Capgras' syndrome as part of an interictal psychosis of epilepsy; magnetic resonance imaging revealed bilateral subcortical lesions in occipitotemporal and frontal regions. These findings have implications for the postulated association between Capgras' syndrome and neuropsychological deficits, in particular prosopagnosia.

The delusional misidentification of closely familiar persons as being alien imposters, despite the acknowledgement of an extreme resemblance, is known as Capgras' syndrome. This phenomena has attracted a variety of explanations. Capgras & Carette (1924) offered a psychoanalytical interpretation of the second case described, and such explanations still have their adherents (Berson, 1983). However, in their original description, Capgras & Reboul-Lachaux (1923) noted a prodrome of derealisation, and suggested that it was this compelling feeling of strangeness which determined the emergence of the delusion—a view which has found support in similar observations by Christoloudou (1977) and by Todd *et al* (1981). Others have remarked on the frequency of organic brain disease, and have proposed that underlying neuropsychological deficits, such as prosopagnosia (Hayman & Abrams, 1977) or sensory inattention (Stuss & Benson, 1984) are important in its evolution. These concepts are appealing, but remain speculative. The new brain imaging techniques, however, offer an opportunity to test such hypotheses in a more direct way. A case of Capgras' syndrome is described, in association with unusual cerebral lesions whose relevance to observed neuropsychological impairments and to the syndrome itself are of interest.

Case report

Jane is a 19-year-old, left-handed girl, the youngest of five daughters of caring, professional parents. There is no family history of psychiatric disorder or epilepsy. She was born by difficult forceps delivery, 2 weeks post-mature, and it was thought she had suffered perinatal intraventricular haemorrhage, although lumbar punctures were repeatedly negative. After being nursed in an incubator for 8 days, she recovered well, and milestones were within normal limits. At the age of 5 years, she developed complex partial epilepsy: her seizures comprised absence attacks, lasting several minutes, during which she would be unresponsive, clench her fingers, and pluck at her clothes. She often experienced a brief prodrome of flashing coloured lights.

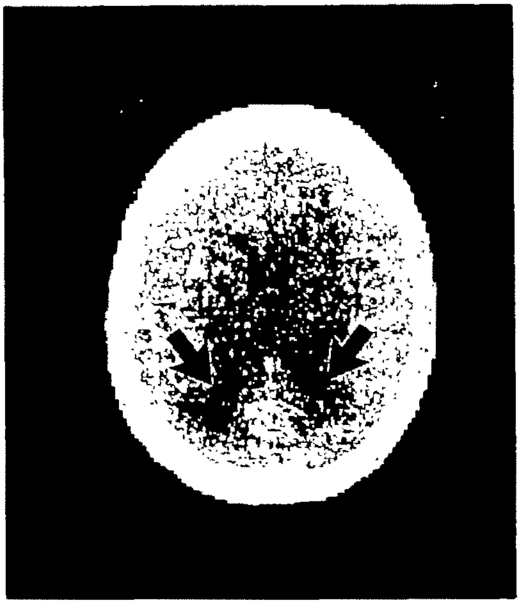

FIG. 1. Computerised tomographic scan showing bilateral occipitotemporal lesions (arrowed).

She had a single convulsive fit at the age of ten and her seizures continued without remission, on an almost daily basis, despite treatment with several different anticonvulsant drugs. Annually repeated EEGs consistently showed bilateral occipitotemporal slow waves, more prominent on the right. Observed seizures were accompanied by high amplitude spike activity in the left posterior temporal area.

As a child, Jane showed problems in discriminating the identities of her familiars; she would often hold the hands of other women, when out shopping with her mother. Psychometric assessment at the age of 18 revealed a verbal IQ of 72, a performance of 61; word-finding difficulties were evident and on tests of learning and memory she was globally weak. She was apraxic, but her most severe deficits were noted to be in visual perceptual processing. Here, although basic shape perception was intact, she showed difficulties in more complex aspects of visual processing, which were most marked on tests of memory for faces and designs. Nonetheless, her social skills were well developed, and she was verbally expressive. Computerised tomography (CT) at this time revealed bilateral occipitotemporal areas of low density, likely to represent infarction (Fig. 1). Neurological examination was normal, apart from a left divergent squint, slight impairment of colour vision, and isolated small scotomata in the absence of gross field defect.

She showed no psychiatric or behavioural symptoms before her admission at the age of 19 to a specialist residential unit for epilepsy; she was then taking carbamazepine and phenytoin, with therapeutic plasma levels. Shortly after her admission, an increased dose of phenytoin was prescribed inadvertently: after 7 days at this higher dosage, her daily fits ceased. Over the next 3 days, she

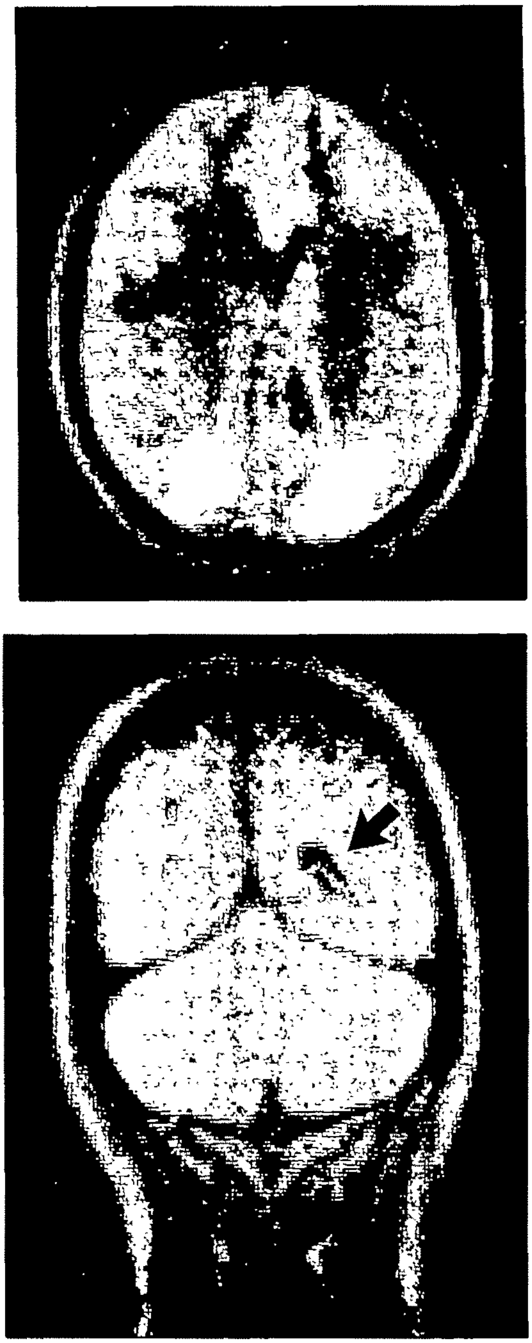

FIG. 2a & b. Magnetic resonance imaging scans using spin echo technique. Axial slice (at same level as Fig. 1) confirming bilateral occipitotemporal lesions (white) and showing smaller bilateral frontal lesions. Coronal slice shows anterior extent of occipitotemporal lesions (arrowed).

became agitated, and described third-person auditory hallucinations of family members, with the secondary delusion that God had suddenly equipped her with supersensitive hearing. She was fully orientated in time and place. Plasma phenytoin at this time was almost twice the upper limit of the therapeutic range. Despite prompt withdrawal of the phenytoin, her psychotic symptoms persisted, and she experienced fleeting complex visual hallucinations, on one occasion seeing the decapitated corpse of her boyfriend. She became suspicious of the staff, and her mood was labile. When her family visited, she failed to recognise her sister, who had a new hairstyle, claiming instead she was a long-dead maternal aunt. The following day, she was convinced that her mother, father, and sisters had been replaced by "dummies", who visited with the intention of kidnapping her; she believed that these imposters lived at an identical house nearby, and also owned a replica of her pet cat. She said that they held her real family as prisoners, and were torturing them. When questioned, she stated that whereas the "dummies" looked exactly like her real family, they possibly dressed differently, and that her mother had given her a tighter hug than usual. This delusion lasted about a week, after which her fits returned at their previous frequency. Magnetic resonance (MR) imaging confirmed the presence of the bilateral occipitotemporal lesions, and also revealed smaller symmetrical lesions in the frontal region (Fig. 2). The appearance and distribution of these lesions suggested them to be old infarctions at the 'watershed' areas between the middle and posterior, and middle and anterior, cerebral arteries respectively. The lesions were not seen to extend to the cerebral cortex.

At 6-month follow-up, she reported experiencing occasional auditory pseudo-hallucinations, but had full insight into her past psychopathology. The diagnosis was of a schizophreniform psychosis, arising as an interictal psychosis of epilepsy, apparently precipitated by phenytoin overdosage. Both the form and content of the psychosis were presumably influenced by the nature and situation of pre-existing cerebral lesions.

Discussion

This case illustrates well the organic epiphenomena often reported in patients with Capgras' Syndrome. Evidence of pre-existing cerebral disease—in this case temporal lobe epilepsy—is commonly found (Cummings, 1985). Electroencephalographic abnormalities are frequently seen, and usually take the form of paroxysmal bitemporal slow wave activity, as here; in some instances, the EEG changed as the psychosis resolved (MacCallum, 1973). Neuropsychological impairments have attracted much attention. Christoloudou (1977) found specific impairments of visuospatial perception and memory in nine out of ten patients tested systematically; these findings have been taken as further circumstantial evidence for a regional deficit in Capgras' syndrome, more specifically in the non-dominant occipitoparietal area, although not all patients tested have shown this pattern. Some perform normally (Merrin & Silberfarb, 1976), or show evidence of frontotemporal impairment (Morrison & Tarter, 1984). Classical prosopagnosia, in the sense of an isolated loss of the ability to recognise previously familiar faces, has never been satisfactorily demonstrated in patients with Capgras' syndrome. However, difficulties in facial recognition—as part of a more general impairment in higher visuospatial function—have been recorded in several patients, and were again observed in the case described here.

Results of CT have been reported in four cases of Capgras' syndrome: appearances were normal in one patient (Thompson *et al*, 1980), while changes suggestive of cortical atrophy were seen in two (Todd *et al*, 1981; Quinn, 1981). In the fourth case, Alexander *et al* (1979) reported marked frontal and right temporal atrophy in a patient who developed Capgras' syndrome following a severe head injury; on this basis, it was suggested that frontal lobe impairment was important in the genesis of the syndrome. However, this patient also showed visuospatial deficits on testing, and his psychopathology was atypical, in that he showed a jovial, apathetic affect in place of the suspiciousness regarded by many as characteristic of Capgras' syndrome (Todd *et al*, 1981).

MR imaging is superior to CT in identifying areas of cerebral infarction, particularly where these lie close to the bone (Bydder, 1983). In the patient described above, roughly symmetrical bilateral lesions were revealed in the occipitotemporal and, to a lesser extent, frontal regions. Previous CT showed that these lesions were present before the onset of Capgras' syndrome, and the clinical history implies that they were lifelong. The possible role of perinatal cerebrovascular damage in some psychoses has been discussed recently (Murray *et al*, 1985). In the light of these findings, can anything further be said about the relationship between neuropsychological deficits, in particular prosopagnosia, and Capgras' syndrome?

Like Capgras' syndrome, prosopagnosia (Bodamer, 1947) is a rare disorder, whose aetiology has provoked much speculation; it is usually accompanied by a visual field defect and a disturbance of colour vision. A primary face-specific perceptual impairment has been the mechanism favoured in the past (Whiteley & Warrington, 1977), and this was thought to reflect damage to the non-dominant occipitoparietal cortex, but more recently, this interpretation has been questioned. Studies with split-brain subjects have demonstrated that both hemispheres are capable of facial recognition, although different strategies may be employed (Levy *et al*, 1972). Prosopagnosic patients recognise and can name the component parts of a face, as well as knowing that what they observe is a face. Indeed, unfamiliar faces can often (but not always) be discriminated well, while accurate perception of other highly complex visual stimuli is possible. What suffers is the recognition of previously familiar faces and, occasionally, other visually similar, once-familiar items: Damasio (1985) cites a prosopagnosic farmer no longer able to recognise individual animals previously well known to him. These observations have led to the alternative hypothesis that the primary defect in prosopagnosia is not of perception but of memory—the perception is correct, but it fails to elicit the appropriate associated contextual memories which usually provide the experience of familiarity. These memories can still be triggered by non-visual stimuli so that a person's voice will prompt recognition, but not his face. Furthermore, instead of showing a non-dominant occipitoparietal lesion, almost all patients with prosopagnosia who have come to post mortem (Meadows, 1974) or computerised tomography or magnetic resonance imaging (Damasio, 1985) have shown bilateral occipitotemporal lesions, highly reminiscent of those found in the patient reported here. Patients with similarly placed unilateral lesions, or bilateral occipitoparietal lesions, do not show prosopagnosia (Damasio *et al*, 1982). The bilateral lesions are functionally symmetrical, and involve the subcortical pathways by which complex visual information is passed forward to the temporal lobe. From this, a disconnection of limbic structures from the visual association cortex has been proposed as the mechanism responsible for prosopagnosia (Damasio, 1985). Could a similar mechanism be operating in Capgras' syndrome?

The idea of a possible relationship between the disorders has been rejected for several reasons. Firstly, they have not been shown to co-exist: patients with prosopagnosia do not show psychiatric symptoms and, conversely, classical, isolated prosopagnosia is not displayed in Capgras' syndrome. However, it is clear that many patients with Capgras' syndrome do show a more generalised visuospatial impairment, of which poor facial recognition is an important part. Secondly, the restriction of Capgras' syndrome to the identity of those most closely familiar to the patients is an observation often used to discount organic explanations in favour of psychodynamic ones (Berson, 1983), yet prosopagnosia shows exactly the same pattern. The third objection is that the natural history of the two disorders differs, the assumption being that Capgras' syndrome is essentially episodic, whereas prosopagnosia is a permanent neuropsychological deficit. Again, this is not necessarily true: Capgras' syndrome may be remarkably chronic, and reports of episodic prosopagnosia exist. Whiteley & Warrington (1977) cite a case of prosopagnosia which resolved during radiotherapy to an occipital tumour and, more pertinent to the present case report, Agnetti *et al* (1978) described a patient with recurrent, short-lived prosopagnosia which proved to be an ictal phenomenon. Lastly, critics point to the lack of an established neuropathology common to the two conditions. This is challenged by the findings of the present report; the detection of such lesions obviously depends to some extent on the sensitivity of the instrument used.

That organic factors operate in Capgras' syndrome is increasingly evident and the demonstration, in such a patient, of lesions apparently characteristic of prosopagnosia strengthens the conceptual link between the two disorders. They remain separate clinically, but in at least some cases of Capgras' syndrome, a neuropsychological mechanism analogous to that postulated for prosopagnosia might be at work. Thus, by reason of a functional disconnection between the central visual system and the temporolimbic lobe, a correct perception fails to evoke the normal affective memories tied to it. The result might best be regarded neither as an illusion (Cutting, 1985), nor a delusion (Berson, 1983), but as a confabulation.

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References

Alexander, M. P., Stuss, D. T. & Benson, D. F. (1979) Capgras syndrome: a reduplicative phenomenon. *Neurology*, **29**, 334–339.

Aonetti, V., Carreras, M., Pinna, L. & Rsati, G. (1978) Ictal prosopagnosia and epileptogenic damage of the dominant hemisphere: a case history. *Cortex*, **14**, 50–57.

Berson, R. J. (1983) Capgras' syndrome. *American Journal of Psychiatry*, **140**, 969–978.

Benton, A. (1980) The neuropsychology of facial recognition. *American Psychology*, **35**, 176–186.

Bodamer, J. (1947) Die Prosop-Agnosie. *Archiv fur Psychiatrie und Nervenkrankheiten*, **179**, 6–53.

Bydder, G. M. (1983) Clinical nuclear magnetic resonance imaging. *British Journal of Hospital Medicine*, **29**, 348–356.

Caporas, J. & Reboul-Lachaux, J. (1923) Illusion des sosies dans un delire systemise chronique. *Bulletin de la Societe Clinique de Medicine Mentale*, **2**, 6–16.

—— & Carette, P. (1924) Illusion des sosies et complexe d'Oedipe. *Annales Medico-psychologiques*, **82**, 48.

Christoloudou, G. N. (1977) The syndrome of Capgras. *British Journal of Psychiatry*, **130**, 556–564.

Cummings, J. L. (1985) Organic delusions: Phenomenology, anatomical correlations and review. *British Journal of Psychiatry*, **146**, 184–197.

Cutting, J. C. (1985) *The Psychology of Schizophrenia*. London: Churchill Livingstone.

Damasio, A. R. (1985) Prosopagnosia. *Trends in Neurosciences*, **8**, 132–135.

——, Damasio, H. & Van Hoesen, G. W. (1982) Prosopagnosia: anatomic basis and behavioural mechanisms. *Neurology*, **32**, 331–341.

Hayman, M. A. & Abrams, R. (1977) Capgras syndrome and cerebral dysfunction. *British Journal of Psychiatry*, **130**, 68–71.

Levy, J., Trevarthen, C. & Sperry, R. W. (1972) Perception of bilateral chimeric figures following hemispheric deconnexion. *Brain*, **95**, 61–78.

MacCallum, W. A. (1973) Capgras symptoms with an organic basis. *British Journal of Psychiatry*, **123**, 639–642.

Meadows, J. C. (1974) The anatomical basis of prosopagnosia. *Journal of Neurology, Neurosurgery and Psychiatry*, **37**, 489–501.

Merrin, E. L. & Silberfarb, P. M. (1976) The Capgras phenomenon. *Archives of General Psychiatry*, **33**, 965–968.

Morrison, R. L. & Tarter, R. E. (1984) Neuropsychological findings relating to Capgras' syndrome. *Biological Psychiatry*, **19**, 1119–1128.

Murray, R. M., Lewis, S. W. & Reveley, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, **i**, 1023–1026.

Quinn, D. (1981) The Capgras syndrome: two case reports and a review. *Canadian Journal of Psychiatry*, **26**, 126–129.

Stuss, D. T. & Benson, D. F. (1984) Neuropsychological studies of the frontal lobe. *Psychological Bulletin*, **95**, 3–28.

Thompson, M. I., Silk, K. R. & Hover, G. L. (1980) Misidentification of a city: delimiting criteria for Capgras Syndrome. *American Journal of Psychiatry*, **137**, 1270–1272.

Todd, J., Dewhurst, K. & Wallis, G. (1981) The syndrome of Capgras. *British Journal of Psychiatry*, **139**, 319–327.

Whiteley, A. M. & Warrington, E. K. (1977) Prosopagnosia: a clinical, psychological and anatomical study of three patients. *Journal of Neurology, Neurosurgery and Psychiatry*, **40**, 395–403.

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Alcohol-related Psychiatric Emergencies: Their Characteristics and Care at a Walk-in Clinic

Fifty cases attending a walk-in psychiatric emergency clinic with alcohol-related problems were studied retrospectively. Results confirm that this group represents a sizeable load on the clinic – about 15% of all assessments. Other psychiatric problems were common, and many patients had recently attempted or threatened suicide.

Alcoholism and alcohol-related problems have long been regarded as a substantial demand on emergency services. In the USA, Rund *et al* (1981) estimated the overall prevalence of alcoholism in patients attending a general emergency department as high as 20%. In the UK, a study of casualty attenders found that of those who presented with psychiatric disorder (2.2%), 10% were for alcoholism (Anstee, 1972). In a psychiatric emergency clinic, Jones (1979) discovered a serious rate of missed diagnoses of alcoholism amongst patients subsequently admitted, though Lim (1983), examining the same clinic, still found 10% of index cases to have a diagnosis of alcoholism or drug dependence.

Clinical practice suggested that there were higher rates of alcohol-related disorder, and in the absence of other relevant work, it was thought appropriate to examine in detail the size and nature of the problem. It was hoped that these data would provide useful information to those intending to improve or design alternative services. The study is based on the Maudsley Hospital Emergency Clinic, which has been well described in other work (Brothwood, 1965; Mindham *et al*, 1973; Lim, 1983). The Bethlem Royal and the Maudsley Hospitals are referred to as the Joint Hospital.

Method

To minimise seasonal bias, cases were collected from two separate 2-week periods, 6 months apart: the middle two weeks of November 1981 and the corresponding weeks in May 1982. Clinic's journal; all such cases were found to have major alcohol problems.

Using a coded schedule, data were abstracted retrospectively from the case notes. Information was coded by two raters: a nursing sister from the Alcohol Treatment Service and the author. The study of Edwards *et al* (1976) indicated that inter-rater reliability could be a cause of concern in abstracting data from case notes with this group of patients, but preliminary checks confirmed that both inter-rater reliability and the standard of case note recording was generally high.

Results

Size of the problem

Over the 4-week study period, 50 patients with alcohol-related problems were identified: this amounted to 66 separate presentations, out of a total of 447. Thus, 14.9% of the emergency clinic assessments had alcohol-related problems.

Social characteristics

The mean age of the sample was 41.9 years, with a range from 23 to 75 years. The sex distribution revealed 33 males and 17 females. 19 were currently married or had been co-habitating with a partner for 6 months or more, and only four were listed as having no next of kin. Social class as defined by the Hall-Jones Scale of Occupations Prestige (Oppenheim, 1966), based on best-ever reported occupation, showed a span between class 1 and class 8. The employment rate was low, with only ten being currently in work. The majority of patients (66%) came from outside the Hospitals' catchment area; seven were recorded as having no fixed abode.

Past treatment histories

Only 15 were classified as new patients to the Joint Hospital; 21 were current attenders (had attended sometime within the last 6 months), while the remaining 14 were categorised as new-old patients. Therefore, over two-thirds of these patients had had previous treatment at the Joint Hospital. However, only a third of these (11) had attended the Alcohol Treatment Service, and only five of them were current attenders. A substantial number of those registered as patients of the Joint Hospital were (or had been) receiving help for their alcohol problems in other departments: 11 as past outpatients, eight as past inpatients, and four currently attending an outpatient clinic.

A history of past treatment of alcohol-related problems outside the Joint Hospital was another common feature. At other hospitals, 19 had been inpatients, 14 past outpatients and one a current outpatient. Six had been through a community-based detoxification unit. Eight at some time had stayed at a rehabilitation hostel, and one was currently resident in such a hostel.

Source of referral

Self-referral was the most common means of introduction to the clinic, accounting for 21 cases. Other sources included: general practitioners (28%), other hospitals (16%), community agencies (4%), and the police (2%). Interestingly, none came from those agencies specifically helping alcoholics. All patients attended on a voluntary basis, none being brought or detained under the Mental Health Act.

Presenting problems

Drinking problems

Just over a quarter (14) requested help for symptoms of alcohol withdrawal. In all, eight asked for medication to stop or reduce their alcohol consumption, while only one case was noted to want help because of a physical problem—alcohol-induced gastritis. Over half (29) cited other problems e.g. an inability to cope leading to demands for admission, general distress and depression associated with alcohol, drinking causing marital problems, black-outs, and a case of alcoholic hallucinosis.

Other psychiatric problems

Nearly half (23) complained of feeling depressed, but only eight were noted to have anxiety. Other symptoms were recorded in 14 cases and included auditory hallucinations in three patients, distress over their persistent antisocial and aggressive behaviour in two, cognitive deficits in two, visual illusions in two; and in single cases, persecutory delusions, hypomanic mood swings, premature ejaculation, and dysmorphophobia.

Social problems

Nearly 90% had evidence of social problems and, for many, in several areas of their life. The most frequently reported were marital difficulties—in 15—i.e. three-quarters of those married. Other common social problems lay in the areas of family relationships (in 24% of all cases), accommodation (22%), difficulties at work (18%), and other relationship problems (18%). Surprisingly, recreational difficulties were recorded in only one case.

Associated deliberate self-harm

Nearly a quarter had committed, within a week of presentation, an act of deliberate self-harm which was noted to relate to their current difficulties: ten cases had taken a tablet overdose and another two had cut their wrists.

Behaviour at attendance

Levels of intoxication from alcohol were categorised as follows: six cases were documented as drunk, intoxicated or ataxic; five as 'smelling of alcohol'; and in nine others, although there was no evidence in the clinical notes, they were recorded as 'alcohol overdoses'. The single case who was described as unco-operative and six more who refused to co-operate with history taking were all in this intoxicated group.

Threatening behaviour to others was noted in five cases: three issued only verbal threats; one was both verbally and physically threatening, and one threatened physical violence by waving a knife about. In this last case, the man was suffering from symptoms of acute alcohol withdrawal, but in the others, two males and two females, there was evidence of ebriation. Three patients caused minor damage to hospital property. Threats of self-harm were more common, being noted in 14 cases: the majority of these (nine) had already made a deliberate attempt to harm themselves, but five had not.

Diagnostic practices

Duty doctors commonly listed two diagnoses, and occasionally three. In descending order of frequency, these were in the following broad categories: alcohol-related disorders, depression, personality disorder, and stress reactions. There were no diagnoses of schizophrenia.

Treatment given

Just over half, 28, received a prescription; the use of medication was unremarkable. Only in 17 cases was there evidence that any advice had been given to patients; this covered: abstaining from drinking, going to a voluntary agency, registering with a GP, or returning when sober. Three referrals were made to Alcholics Anonymous, and in one of these it was not the patient but his family who were advised to go.

Discussion

Virtually 15% of patients assessed at the Emergency Clinic were found to have alcohol-related problems; this indicates the substantial size of the problem, and, moreover, it is likely to be an underestimate. Duty doctors may not recognise all cases of alcohol dependence (Jones, 1979), and other patients may have escaped inclusion through a failure to record their alcohol misuse.

Patients with these problems showed a broad range of social characteristics, with one exception; there were none younger than 23 years, so that it is likely that young persons who are abusing drink are not seeking help at the Clinic. Although the great majority had a recorded next of kin, this may belie the true rate of social isolation, as the study could not appraise how many had actually become alienated from their stated relatives and friends.

Despite the 'emergency' nature of the Clinic, only in one in six could it be stated that they had not received previous help for their drinking problems. Indeed, the impression given is that this is a group of patients suffering from chronic distress, repeatedly presenting at times of crises to any available source of help. The high frequency of previous treatment attempts only highlights the importance of providing long-term therapeutic support.

Psychiatric complaints, suicidal behaviour, and complications of alcohol withdrawal make it mandatory for appropriately skilled doctors to be involved, but the large number of social problems suggests an advantage in also involving other professionals. Busy duty doctors covering the Clinic tend to give precedence to cases with more obvious mental illness; the social aspects of distress may be no less deserving of attention, but could be dealt with by others. The finding of only one recording of recreational difficulties commonly associated with alcohol misuse, suggests that relevant social problems are being neglected.

Alcohol intoxication was associated with many of the problems encountered in psychiatric assessment. Of the minority who showed threatening behaviour or could be regarded as unco-operative, all but one—who was suffering from acute withdrawal—was inebriated. However, from the very nature of this population, it would have been expected that many would have consumed alcohol, but in those who proved troublesome, documenting a state of intoxication might have been considered particularly important as a possible explanation. Such a tendency could serve to exaggerate the connection between intoxication and difficult behaviour.

Curiously, there was an absence of schizophrenia from the diagnoses. This could be due to the small sample size, but is perhaps more likely a result of diagnostic bias at initial assessment, when symptoms of psychotic illness might foreshadow abuse of alcohol.

Whereas little criticism could be made of the Clinic's prescribing habits, this cannot be said about other therapeutic options: only in a third of cases was there written evidence that advice had been given, though it is possible that advice was more often given but not recorded. Indeed, when there is an awareness of the importance of a therapeutic strategy, not only is it more likely to be performed, but its execution will also be documented. Therefore, it must be assumed that deliberate use of 'strong medical advice' to stop, with a warning of the outcome of continued drinking, was largely an absent therapeutic force. In addition, there was very little use made of community agencies helping alcoholics; though the potential value of community-based detoxification services, counselling agencies, and Alcoholics Anonymous can be enormous, these resources went virtually untapped. This lack of interaction seems to be mutual, as there were no referrals to the Clinic from these agencies. Better training of staff from all centres would engender a more active awareness of local facilities, and would also be useful in directing this mostly unemployed group towards potentially rewarding occupational and leisure opportunities.

It has been argued that these patients still require psychiatric assessment, but in view of the limitations on medical time, counselling and referral to community facilities could be undertaken by others. As nursing staff already man these units around the clock, suitable candidates could receive the necessary training to enable them to fulfil this role. The work could be incorporated along with other duties, and should therefore not require any substantial increase in manpower. Furthermore, having nurse counsellors on the regular clinic staff should also promote better liaison with community facilities, which in turn might improve the effectiveness of aftercare, and lead to a decrease in the numbers presenting in crisis. It is difficult to demonstrate the efficacy of providing such an extra service, but this is a group of patients who demand help, are severely distressed by their predicament, and tend to cause concern to family, friends, and all those involved in their welfare. It is a need which surely calls for further therapeutic endeavour.

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References

ANSTEE, B. H. (1972) Psychiatry in the casualty department. *British Journal of Psychiatry*, **140**, 2, 180–184.

BROTHWOOD, J. (1965) The work of a psychiatric emergency clinic. *British Journal of Psychiatry*, **111**, 631–634.

EDWARDS, G., RATHOD, N. H., THOMSON, I. G., KYLE, E. & NICHOLLS, P. (1976) Case-note abstraction: A methodological substudy as a cautionary tale. *International Journal of the Addictions*, **11**, 409–416.

JONES, G. H. (1979) The recognition of alcoholism by psychiatrists in training. *Psychological Medicine*, **9**, 789–791.

LIM, M. H. (1983) A psychiatric emergency clinic: A study of attendance over six months. *British Journal of Psychiatry*, **143**, 460–466.

MINDHAM, R. H. S., KELLEHER, M. J. & BIRLEY, J. L. T. (1973) A psychiatric casualty department. *Lancet*, **ii**, 1169–1171.

OPPENHEIM, A. N. (1966) *Questionnaire Design and Attitude Measurement*, London: Heinemann.

RUND, D. A., SUMMERS, W. K. & LEVIN, M. (1981) Alcohol use and psychiatric illness in emergency patients. *Journal of the American Medical Association*, **245**, 1240–1241.

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Genetic Markers in Late Paraphrenia: A Study of HLA Antigens

Patients with late paraphrenia were typed for HLA-A, -B and -C. Increased frequencies of BW55 ($P=0.036$), B37 ($P=0.008$) and CW6 ($P=0.056$) were found relative to controls. Unlike findings in paranoid schizophrenia, there was no HLA-A9 association, suggesting that paraphrenics may be genetically distinct from schizophrenics. The primary association seems to be with B37, which has the lowest corrected P value and highest relative risk; if this were replicated, it would make possession of this antigen a strong risk factor for developing the disorder.

Most studies of paraphrenia have concerned themselves with descriptive aspects, so that very little is known about possible aetiological factors. Why does a 'normal' individual become overtly psychotic for the first time late in life? Abnormal personality of paranoid or schizoid type, prolonged social isolation, and long-standing sensory defects—deafness and/or visual impairment—have all been implicated in the genesis of late paraphrenia. (Kay & Roth, 1961; Post, 1966; Herbert & Jacobson, 1967; Cooper *et al*, 1974).

The genetic contribution to paraphrenia (or rather, to the liability to develop the disorder) is still undetermined. Various studies have estimated the risk of developing schizophrenia among relatives of probands with late-onset schizophrenia and of patients with more chronic forms of the illness; the general conclusion has been that relatives of late-onset patients have an appreciably lower risk of developing schizophrenic disorders (Funding, 1961; Kay, 1963, 1972). Although this might be interpreted as showing that the two conditions are distinct, the findings are also consistent with the notion of a multifactorial genetic-environmental continuum of liability (Reich *et al*, 1972), with the disorders differing quantitatively, but not qualitatively, with respect to genetic loading, Farmer *et al* (1984). Another obvious demographic difference is that late paraphrenia shows a female predominance, while schizophrenia affects both sexes equally, but the age of onset of schizophrenia in females is later than that in males, so that late paraphrenia may still be part of the same continuum. As the relationship between paraphrenia and schizophrenia remains in dispute, an investigation into potential genetic markers of paraphrenia might shed some light on the aetiology of the disorder, and contribute to our knowledge of its nosology.

Certain antigens of the HLA (Human Leucocyte Antigen) Complex have been shown to be associated with susceptibility to disorders such as ankylosing spondylitis (B27), rheumatoid arthritis (DR4) and diabetes mellitus (DR3 and DR4). There has also been considerable interest in the possible association between HLA antigens and the major functional psychoses (Goldin & Gershon, 1983). Although for schizophrenia as a whole the results of studies have been conflicting, there has been virtually complete consistency in the finding of an association between HLA-A9 and paranoid schizophrenia (McGuffin *et al*, 1981; Ivanyi *et al*, 1983). Moreover, HLA-A9 was also found to be markedly increased among older schizophrenics (Eberhard *et al*, 1975), but no studies, to our knowledge, had attempted to examine HLA antigen associations in late paraphrenia.

Method

Thirty-one unrelated British patients, 29 females and two males, were HLA typed; this formed part of a larger study on the possible role of organic factors in the genesis of the syndrome. The ages of the patients ranged from 61 to 88 years, with a mean of 74.96. HLA-ABC typing was done by a modified two-stage NIH technique (Festenstein *et al*, 1973); HLA types were assigned on the basis of reactions with a panel of at least 180 local and workshop sera. The frequencies of the HLA antigens of the patients were compared with those of an unrelated British control panel of 187 individuals (average age 35).

Patients were first diagnosed as paraphrenic by the consultant psychiatrist in charge of their case, but before inclusion in the study, all were assessed using the Geriatric Mental State schedule (GMS) (Copeland *et al*, 1976) and a detailed psychometric procedure. Subjects were required to fulfil the following criteria: (1) presence of fantastic, persecutory, grandiose delusions; delusions of reference and hallucinations; (2) absence of a primary affective disorder; (3) intellectual capacity in keeping with that of normal ageing; (4) phenomena occurring in a setting of clear consciousness; (5) first onset of paraphrenia at the age of 60 or over; (6) absence of a history of cerebrovascular accident, neurological disorder, or alcoholism.

The software used to analyse the HLA data was developed by Dr J. D'Amaro, Department of Immunohaematology, Leiden, The Netherlands. The significance of differences in antigen frequencies between patients and controls was assessed by the method of Woolf (1955), with the modification of Haldane (1955). The aetiological fraction (Green, 1982), which estimated the degree of contribution of an antigen to the development of a disease, was also calculated.

Results

The antigen frequencies are shown in Table I. When compared with the controls, the paraphrenic patients showed increased frequencies of BW55, B37, and CW6, significant at the 0.05 level before correction for the number of comparisons. These differences became non-significant both after correction for the number of comparisons (43) (Svejgaard *et al*, 1975) and after Edwards' (1974) correction, which is slightly less conservative, was applied.

Neither A9 nor its splits (A23 and A24) were significantly increased in frequency in the patients.

Discussion

The question whether the positive HLA antigen associations found in this study are in fact due to the disparity in age between the controls and the patients can probably be answered in the negative, as no changes in HLA profile with ageing have been reported (Blackwelder *et al*, 1982).

The lack of an association with A9 antigen suggests that patients with late paraphrenia may be biologically different or genetically distinct from schizophrenics. Funding (1961), reviewing his data on 148 patients in whom paranoia developed after the age of 50, concluded that paraphrenia, if it is inherited, is transmitted in a manner different from that of schizophrenia. A similar opinion was also expressed by Retterstol (1968), who concluded in his consideration of data on the subtyping of monozygotic twins and schizophrenia that later-onset paranoid forms of schizophrenia may be inherited in a manner distinct from that of other subtypes of schizophrenia.

In this study, the sample size was too small to refute confidently an association with A9. We have calculated that if the relative incidence of the disorder in A9-positive versus A9-negative individuals is approximately two, and if about 15% of the controls carry the marker trait, the sample size required to detect an association significant at the 5% level would be 105. Nevertheless, there was not even a trend towards A9 associations appeared in our data. It is therefore unlikely that we are commiting a type II error. Of the three HLA antigens with which positive associations were found in this study (BW55, CW6

TABLE I
Phenotype frequencies of HLA-A, -B and -C antigens in late paraphrenia patients compared with controls.

Phenotype Frequency			
Antigen		*Patients (n=31)*	*Controls (n=187)*
	A1	38.70	32.09
	A2	48.38	47.06
	A3	41.93	27.81
(A9)	A23	6.45	3.76
(A9)	A24	9.67	12.37
(A10)	A25	6.45	4.81
(A10)	A26	0.00	5.88
	A11	22.58	13.90
	A28	6.45	6.41
(AW19)	A29	3.22	9.67
(AW19)	A30	0.00	4.30
(AW19)	A31	0.00	6.45
(AW19)	A32	3.22	8.60
(AW19)	AW3		
3		3.22	2.68
(B5)	B51	6.45	8.02
	B7	22.58	24.06
	B8	32.26	25.67
(B12)	B44	29.03	35.83
	B13	6.45	3.21
	B14	12.90	10.16
(B15)	BW62	6.45	9.19
(BW16)	B38	0.00	1.60
(BW16)	B39	3.23	2.67
(B17)	BW57	6.45	4.32
(B17)	BW58	0.00	1.08
	B18	6.45	9.10
(BW21)	B49	0.00	1.07
(BW22)	BW55	9.67	2.69**
(BW22)	BW56	0.00	0.54
	B27	6.45	12.83
	B35	12.90	14.44
	B37	16.13	4.28***
	BW41	0.00	1.60
	BW47	0.00	1.60
(B40)	BW60	16.13	10.33
(B40)	BW61	0.00	1.09
	CW1	6.45	7.82
	CW2	3.23	10.61
	CW3	29.03	24.02
	CW4	19.35	19.55
	CW5	16.13	23.46
	CW6	29.03	15.64*
	CW7	51.61	36.69

*P=0.056, EF=15.9%, RR=2.24
**P=0.036, EF=7.2%, RR=4.05
***P=0.008, EF=12.4%, RR=4.38
EF = etiologic fraction
RR = relative risk

and B37), the most likely to be relevant is B37. HLA B37 has the lowest (uncorrected) P value for the χ^2. Also, as CW6 is in linkage disequilibrium with B37 in caucasoids, its increased frequency is likely to be secondary to that of B37. If the primary association of paraphrenia were with CW6, one would have expected an increase in other HLA-B locus antigens in linkage disequilibrium with it (i.e. B13, BW47, BW57, BW50), as is the case in the only CW6-associated disorder so far described—psoriasis (Tiilikainen *et al*, 1980).

We can conclude that since B37 and CW6 are in positive linkage disequilibrium in European caucasians, our results probably represent an increased frequency of these two antigens simultaneously in the patients investigated. This finding merits further investigation. The best way to proceed would be to repeat the study in an independent sample. The B37 association, if confirmed, would make possession of this antigen the strongest known risk factor for developing the disorder.

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References

BLACKWELDER, W. C., MITTAL, K. K., MCNAMARA, P. M. & PAYNE, F. J. (1982) Lack of association between HLA and age in an ageing population. *Tissue Antigens*, **20**, 188–192.

COOPER, A. F., KAY, D. W. K., CURRY, A. R., GARSIDE, R. F. & ROTH, M. (1974) Hearing loss in paranoid and affective psychoses of the elderly. *Lancet*, **ii**, 851–861.

COPELAND, J. R. M., KELLETT, J. M., GOURLAY, A. J., GURLAND, B. J., FLEISS, J. L. & SHARPE, L. (1976) A semistructured clinical interview for the assessment of diagnosis and mental state in the elderly: The Geriatric Mental State Schedule: 1. Development and reality. *Psychological Medicine*, **6**, 439–449.

EBERHARD, G., FRANZEN, G. & LOW, B. (1975) Schizophrenia susceptibility and HLA antigens. *Neuropsychobiology*, **1**, 211.

EDWARDS, J. H. (1974) HLA and disease. The detection of associations. *Journal of Immunogenetics*, **1**, 249–258.

FARMER, A. E., MCGUFFIN, P. & GOTTESMAN, I. L. (1984) Searching for the split in schizophrenia, a twin study perspective. *Psychiatry Research*, **13**, 109–118.

FESTENSTEIN, H., ADAMS, E., BURKE, J., OLIVER, B. T. D., SACHS, J. A. & WOLF, E. (1973) The distribution of HLA antigens in expatriates from East Bengal living in London. In *Histocompatibility Testing 1972* (ed. Dausset). Copenhagen: J. Munskgaard.

FUNDING, T. (1961) Genetics of paranoid psychoses in later life. *Acta Psychiatrica Scandinavica*, *37*, 267–282.

GOLDIN, L. R., & GERSHON, E. S. (1983) Association and linkage studies of genetic marker loci in major psychiatric disorders. *Psychiatric Developments*, **4**, 387–418.

GREEN, A. (1982) The epidemiologic approach to studies of association between HLA and disease. II Estimation of absolute risks, etiologic and preventive fraction. *Tissue Antigens*, **19**, 259–268.

HALDANE, J. B. S. (1955) The estimation and significance of the logarithm of a ratio of frequencies. *Annals of Human Genetics*, **20**, 309–311.

HERBERT, M. E. & JACOBSON, S. (1967) Late paraphrenia. *British Journal of Psychiatry*, **113**, 461–469.

IVANYI, P., DROES, J., SCHREUDER, G. M. T. H., D'AMARO, J. & VAN ROOD, J. J. (1983) A search for association of HLA antigens with paranoid schizophrenia. *Tissue Antigens*, **22**, 186–193.

KAY, D. W. K. (1963) Late paraphrenia and its bearing on the aetiology of schizophrenia. *Acta Psychiatrica Scandanavica*, **39**, 159–169.

—— (1972) Schizophrenia and schizophrenia like states in the elderly. *British Journal of Hospital Medicine*, **7**, 369–376.

——, ROTH, M. (1961) Environmental and hereditary factors in the schizophrenia of old age (late paraphrenia) and their bearing on the general problem of causation in schizophrenia. *Journal of Mental Science*, **107**, 649–686.

MCGUFFIN, P., FARMER, A. E. & YONACE, A. H. (1981) HLA antigens and subtypes of schizophrenia. *Psychiatry Research*, **5**, 115–122.

POST, F. (1966) *Persistent Persecutory States of the Elderly*, Oxford: Pergamon Press.

REICH, T., JAMES, J. W. & MORRIS, C. A. (1972) The use of multiple thresholds in determining the mode of transmission of semi-continuous traits. *Annals of Human Genetics*, **36**, 163–184.

RETTERSTOL (1968) Paranoid Psychoses. *British Journal of Psychiatry*, **114**, 533.

SVEJGAARD, A., PLATZ, P., RYDER, L. P., NEILSON, L. S. & THOMSON, M. (1975) HLA and disease association: a survey. *Transplant Review*, **22**, 3–39.

TIILIKAINEN, A., LASSUS, A., ARVONEN, J., VARTIAINEN, P. & JULIN, M. (1980). Psoriasis and HLA-CW6. *British Journal of Dermatology*, **102**, 179.

WOOLF, B. (1955) On estimating the relation between blood groups and disease. *Annals of Human Genetics*, **19**, 251–253.

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Prolonged Oculogyric Crisis on Addition of Nifedipine to Neuroleptic Medication Regime

Extrapyramidal symptoms, including oculogyric crisis, are well recognised side-effects of clopenthixol medication, but precipitation of prolonged oculogyric crisis on addition of nifedipine to an ongoing clopenthixol regime has not so far been reported.

Case Report

The patient, a 26-year-old-single person, is a long-stay resident at Leavesden Hospital. He suffers from disintegrative psychosis with infantile autism, probably following infantile convulsion when aged 18 months; he is extremely anxious. His principal problems are self-injurious behaviour, in the form of eye poking, aggressive outbursts directed towards others and stereotyped ritualistic behaviour. In addition to behaviour modification, he had been treated with chlorpromazine 50 mg *q.d.s.* and clopenthixol 400 mg i.m. fortnightly for 2 years. In 1983 he was found to have idiopathic hypertension, BP 160/130 mmHg; he was investigated but no cause was found for his hypertension. He was treated with atenolol 100 mg daily, which had to be stopped on 17 June 1984, as he developed an allergic reaction in the form of wheezing and eye irritation. He was prescribed nifedipine 10 mg *t.d.s.* 2 days later, but after 1 week he became extremely drowsy and nifedipine was stopped and chlorpromazine reduced to 50 mg *b.d.* Within 3–4 days he was alert again and able to attend school. He had his last injection of clopenthixol on 28 July 1984; one week later nifedipine was recommenced and the very next day he developed oculogyric crisis (eyes were permanently fixed in upturned position), which failed to respond to i.m. injections of procyclidine up to 60 mg daily. One week later nifedipine and neuroleptic medication were stopped, and 3 days later oculogyric crisis again subsided. Neurological examination did not reveal anything significantly abnormal. During this episode his blood pressure remained between 150/110 mmHg–130/100 mmHg. To the time of writing (6 months) he has been taking chlorpromazine 600 mg daily and captopril 12.5 mg *b.d.* without any untoward effects.

Discussion

Nifedipine is a potent calcium antagonist. Its most important effect is to protect the heart against excessive oxygen utilisation. It also causes peripheral vasodilatation. Its known side-effects are throbbing headache, flushing and dizziness, tachycardia and peripheral oedema. It is known to interact with digoxin and β-adrenergic receptor blocking agents, but there are no reports of the type of adverse reaction seen in our patient. He has not been given nifedipine or clopenthixol again for fear of precipitating another oculogyric crisis. As we did not use nifedipine on its own, it cannot be assumed that the prolonged oculogyric crisis was due to nifedipine but it appears probable that nifedipine potentiated the extrapyramidal symptoms of neuroleptic medication. It may be that the interaction has something to do with the calcium antagonist properties of nifedipine. It is perhaps of interest that diltiazem, another calcium antagonist vasodilator, has been reported to produce akathisia (Jacobs, 1983).

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I thank Dr Isweran, Consultant Psychiatrist, for permitting me to send this case for publication.

Reference

Jacobs, M. B. (1983) Diltiazem and akathisia. *Annals of Internal Medicine*, 99, 794.

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The Continuum of Psychosis and the Gene

SIR: Crow (*Journal*, October 1986, **149**, 419–429) appears to fall into the trap, common to most discussions of unitary psychosis, of regarding psychosis or psychoses as disease(s) rather than syndrome(s). As a result, he is led to postulate the existence of a peculiarly unstable gene or limited set of genes whose abnormalities express themselves as psychosis of varying symptomatology.

That schizophrenia is a syndrome has been recognised ever since Eugen Bleuler wrote about "the schizophrenias", and the evidence that they have multiple aetiologies is now fairly compelling. Of particular relevance to Crow's argument is the finding that process schizophrenias may have a rather low heritability relative to some other types. Severe depression, also, seems to have multiple causes and it is probable that mania has more than one cause (it appears that, in a small number of families, its inheritance is X-linked while in most families it is not; some patients may have abnormal vanadium metabolism; some patients respond well to lithium while others do not, etc.). In situations like this, the observation of continua between syndromes is to be expected and has no great aetiological significance. After all, high dose steroids can produce depression in one patient, mania in another and paranoid schizophrenia in a third. It would be most interesting to know the reason for these idiosyncrasies, but they would hardly justify postulating the existence of a special 'steroid psychosis' gene with unique properties.

It would not be difficult to construct an argument similar to Crow's, centering around bellyache. There is considerable overlap in the symptoms and other characteristics of people suffering abdominal pain, and it may well be the case that children of women with gallstones are prone to develop diverticulitis (as a result of common nutritional habits). Equally, there are probably seasonality effects in bellyache since it is likely to be more common at Christmas time and over the summer holiday period. Perhaps, therefore, there is a bellyache gene similar to the proposed psychosis gene. There are no doubt genes controlling cerebral organisation and dominance effects which may one day be of great interest to psychiatrists especially, I suspect, those studying personality disorder, but existing knowledge is insufficient to enable one to say anything of heuristic value about them.

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SIR: Crow is to be congratulated on his imaginative theory of psychosis (*Journal*, October 1986, **149**, 419–429). One thing puzzles me about his use of the season of birth data to support a viral explanation. His Figure 2 certainly shows, consistent with the hypothesis, that there is an excess of births in patients with psychotic illnesses during the winter months. However, it also shows an equally large *reduction* during the summer months, a phenomenon that seems to be difficult to reconcile with the presumed pathogenic effects of a virus.

There are several equally convincing explanations of the season of birth effect, one being that it reflects the cycles of sexual activity among the parents of future psychotic patients. Might this not be a more credible and parsimonious explanation of the data, given that there is no other evidence for the viral theory?

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Chronic Schizophrenia and Long-term Hospitalisation

SIR: It was good of Abrahamson (*Journal*, September 1986, **149**, 382) to notice *Institutionalism and Schizophrenia* (Wing & Brown, 1970), but I wonder if I could persuade him to re-read it? He will find that we did not describe one pattern of relationship between negative aspects of behaviour in schizophrenia and length of stay in hospital. There were three, a different pattern for each of our hospitals. More important, our conclusions about the influence of social environment were not derived from these results. On the contrary, we stated (p. 12) that there was overwhelming evidence against the "startling assumption that length of stay is in any way significant as a measure of severity of illness".

What we did find was that when age and length of stay were standardised, patients in the hospital with the poorest social conditions still had the most severe deficit. Moreover, when twenty patients from each hospital, matched for age, length of stay, clinical condition, negative score and attitude to discharge, were compared, major differences remained between the social environments provided for them by the hospitals.

Among much other evidence provided, Abrahamson may be surprised to read that this was not just a cross-sectional study but involved repeated observations over a period of eight years. The social withdrawal scores rated at the beginning and end of that time were highly correlated and showed a mild average improvement in those who remained in hospital. His observation that schizophrenic patients do not necessarily deteriorate thus replicates ours. However, we did measure an increase in 'social poverty' in two of the hospitals during certain periods of observation, with a concomitant increase in 'clinical poverty'.

We pointed out that surveys of this kind could not provide definitive tests of the three kinds of theory stated in chapter 1, but this unit has provided a series of other studies, some of them experimental, that strongly support an interactive socio-medical model. Abrahamson might be particularly interested in an experiment involving very severely impaired patients (Wing & Freudenberg, 1961). They were not impervious to further social stimulation, the problem was to maintain improvement once it was withdrawn.

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References

WING, J. K. & BROWN, G. (1970) *Institutionalism and Schizophrenia.* London: Cambridge University Press.

—— & FREUDENBERG, R. K. (1961) The response of severely ill chronic schizophrenic patients to social stimulation. *American Journal of Psychiatry,* **118,** 311–321.

Gluten Sensitivity in Schizophrenia

SIR: Vlissides, Venulet & Jenner (*Journal,* April 1986, **148,** 447–452) have concluded that gluten sensitivity is of little importance for schizophrenia in general. This far-reaching conclusion is based on a study which is methodologically unsound and, at best, inconclusive.

The difficulty in interpreting the data on quite remarkable patient improvement noted after the switch from pre-trial hospital diet to gluten-free diet is a case in point. Five parameters showed statistically significant improvement: psychotic disorganisation, retardation, hostile belligerence, anxious depression, and depressive mood. No significant worsening was noted on any parameter. Judging from Figure 1, several of the remaining seven PIP dimensions may well have registered significant improvement under gluten-free diet had one-tailed (directional) statistical tests been performed, which is the appropriate statistical technique since a specific hypothesis was under investigation. However, regardless of the statistical method, one is unable to determine whether the data reflect treatment effect, observer bias or a placebo effect because the period before gluten-free diet was non-blind and there was no parallel control group. The subsequent period of gluten challenge did not do much to resolve the issue because possible observer bias arising from knowledge of the sequence of treatments and the time series effects were not controlled. More importantly, the research design did not permit a consideration of the possibility that the six week gluten challenge, while not worsening psychopathology to base-line values, may well have had enough adverse effect in the time period to arrest therapeutic progress. In the AB research design used, this would show up as 'no change', whereas in the ABA design used in the Singh & Kay (1976) study, such an adverse effect would very likely have been detected.

The authors attributed the clinical improvement in patients to the attention they received during the study, or what would generally be described as a placebo effect. Within the framework of their study design, such a conclusion would be no more valid than that suggesting an active treatment effect of gluten exclusion. To the contrary, sustained improvement of a highly chronic and treatment-resistant group of schizophrenics lasting over 3 months due to mere attention or placebo effect would be highly implausible indeed.

The study is also open to criticism because of the patient sample used. While the hypothesis being tested relates to schizophrenia, 7 of the 24 patients in the study had other diagnoses. Of the 17 schizophrenics, only one had a diagnosis of non-paranoid schizophrenia, which is the classification suggested by previous work to be at greater risk for gluten sensitivity (Singh, 1979). On top of that, the population was highly chronic and one in which likelihood of response to any form of treatment would be small.

We conclude, therefore, that the study is not a valid test of the gluten hypothesis of schizophrenia and that the conclusions reached by the authors with regard to the hypothesis are not warranted. In an area of such importance as schizophrenia, this hypothesis represents one of the few viable notions of aetiology and a new treatment approach. It deserves to be investigated thoroughly and with carefully designed studies in which adequate provisions are made for the heterogeneity of this condition (Singh & Kay, 1976; 1983).

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References

SINGH, M. M. (1979) Celiac-type diets in schizophrenia. *American Journal of Psychiatry*, **136**, 733.

—— & KAY, S. R. (1976) Wheat gluten as a pathogenic factor in schizophrenia. *Science*, **191**, 401–402.

—— & —— (1976) Wheat gluten-schizophrenia findings. *Science*, **194**, 449–450.

—— & —— (1983) Study of gluten effect in schizophrenia. *Archives of General Psychiatry*, **40**, 345.

Psychiatric Disorder in the General Hospital

SIR: Mayou & Hawton (*Journal*, August 1986, **149**, 172–190) are right in asserting that there have been few systematic studies about differences in the prevalence of psychiatric disorder in the many types of in-patient and out-patient units within the general hospital. The paucity of data is most apparent in the setting of emergency departments. In a descriptive study of psychiatric emergencies in a general hospital setting, we studied 352 patients presenting psychiatric emergencies over a four-month period (1.86% of all attenders). Only 26 (7.4%) of these patients were already registered with the outpatient services of the psychiatry department, the rest being new patients. The case detection increased by 550% with the continuous presence of a psychiatrist in the emergency room—in contrast to "on-call' cover. Inaccuracies of identification were made by non-psychiatric physicians in approximately 14% of cases. Despite detection, physicians had the tendency not to refer patients to the psychiatrists-on-call. In only 34% of the patients screened, were the non-psychiatric physicians able to make a correct diagnosis of the psychiatric illness. In two-thirds of all patients, non-psychiatric physicians were unable to suggest any management for the psychiatric emergency.

Males outnumbered females in a ratio of 2:1. The majority of the patients (77%) were referred to emergency services by relatives and friends or patients themselves. Two-thirds of the patients were brought owing to the severity of the clinical condition and the rest, one-third, for medico-legal and social reasons. Approximately 80% of our patients sought consultation within one month of the onset of the illness episode. About 40% of those using psychiatric emergency services had long-standing problems of more than one year's duration. Only 10% had a history of hospitalisation for psychiatric illness in the past; and only 20% of the patients had visited emergency services more than once in the past one year. Thirty-one per cent had neurotic disorders, 26% had functional psychotic illnesses and 18% had alcohol-related problems.

There is considerable psychiatric morbidity in the emergency-rooms of general hospitals, much of which is unrecognised by non-psychiatric physicians. There is a need for improved research designs in studies of the epidemiology of psychiatric emergencies in general hospital settings, as psychiatric emergency services represent one of the chief entry points into the network of mental health services.

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Hydroxylated Metabolites of Tricyclic Antidepressant in the Elderly

SIR: We read with interest the report by Kutcher *et al* (*Journal*, June 1986, **148**, 676–679). These findings are consistent with our own experience with nortriptyline in a similar population.

Like 2-hydroxydesipramine, the 10-hydroxylated metabolite of nortriptyline is pharmacologically active (Bertilsson *et al*, 1979). In elderly depressed patients, average unconjugated plasma concentrations of 10-hydroxynortriptyline are higher than in younger patients taking equivalent doses, despite comparable concentrations of plasma nortriptyline (Young *et al*, 1984). There are also marked inter-individual differences in plasma 10-hydroxynortriptyline/nortriptyline ratio in this population. We reported development of symptoms and signs of congestive heart failure in an elderly patient with moderate plasma nortriptyline concentrations but high plasma 10-hydroxynortriptyline (Young *et al*, 1984). We also noted that, in 18 geriatric depressed in-patients, plasma 10-hydroxynortriptyline concentrations or combined plasma 10-hydroxynortriptyline and nortriptyline concentrations, but not plasma nortriptyline concentrations alone, differentiated those patients who developed quinidine-like effects during treatment (Young *et al*, 1985).

Taken together, our own preliminary findings and those of Kutcher *et al* suggest that further study of hydroxylated metabolites of tricyclic antidepressants is warranted in geriatric patients. Their contribution to toxic and therapeutic effects, and to changes in neurobiological measures during treatment, are open to further investigation; these issues might be studied to advantage in elderly patients.

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References

BERTILSSON, L., MELLSTROM, B. & SJOQVIST F. (1979) Pronounced inhibition of noradrenaline uptake by 10-hydroxymetabolites of nortriptyline. *Life Sciences*, **25**, 1285–1292.

YOUNG, R. C., ALEXOPOULOS, G. S., SHAMOIAN, C. A. & KUTT, H. (1984) Heart failure associated with high plasma 10-hydroxynortriptyline levels. *American Journal of Psychiatry*, **141**, 432–433.

——, ——, —— & —— (1984) Plasma 10-hydroxynortriptyline in elderly depressed patients. *Clinical Pharmacology and Therapeutics*, **35**, 540–544.

——, ——, —— & —— (1985) Plasma 10-hydroxynortriptyline and ECG changes in elderly depressed patients. *American Journal of Psychiatry*, **142**, 866–868.

Diagnostic Value of Schneider's First Rank Symptoms in Schizophrenia

SIR: Kurt Schneider (1959) claimed that certain "first rank symptoms" (FRS) were pathognomonic of schizophrenia in the absence of organic brain disorder. Recent investigations have argued against this assertion, but most of the studies have considered FRS collectively and the value of individual symptoms has not been explored much. The frequency of FRS in schizophrenics ranges from 28–72% (Mellor, 1982). This variation is probably due to differences in the diagnostic criteria used and methods employed in eliciting FRS.

We conducted a pilot study based on fifty consecutive admissions to evaluate the prevalence and diagnostic implications of FRS. All the patients, irrespective of their diagnosis, were interviewed by one of the authors (MG) for the presence of FRS as early as possible after admission. The findings were recorded on a questionnaire specially prepared for the study and based on Mellor's definitions of the FRS. The patients were later seen by the first author (HDC) to confirm the findings and diagnosis. Diagnostic labels were given according to DSM-III (1980).

The distribution of 50 patients (23 males, 27 females; age range: 21–86 years) according to the diagnostic categories was as follows: schizophrenic disorders 24; psychotic disorders not elsewhere classified 6 (schizophreniform disorder 2, brief reactive psychosis 1, schizoaffective disorder 1, atypical psychosis-schizophrenia onset after age 45, 2); affective disorders 9 (mania 2, major depression recurrent 4, atypical depression 1, dysthymic disorder 1); organic mental disorders 3; anxiety disorders 2; adjustment disorders 5; and personality disorders 2. FRS were present in 26 patients distributed as follows: schizophrenic disorders 17; psychiatric disorders not elsewhere classified 4; affective disorders 2; organic mental disorders 2; and personality disorders 1.

The analysis of data on schizophrenics with FRS produced the following results. (a) The majority of patients with schizophrenia had 2 or more symptoms, maximum 9 symptoms; only 3 had 1 symptom each. (b) The symptoms more commonly reported (in order of frequency) were: 'made' affect (11), thought insertion (10), thought broadcasting (9) and voices arguing (7). (c) Auditory hallucinations were reported by half the patients, the commonest being voices arguing, but always accompanied by a symptom of passivity. (d) 16 patients with schizophrenia had one or more passivity experiences. (e) Delusional perception was reported only by 3 patients. (f) The commonest association between any two symptoms was between thought insertion and thought broadcasting.

The findings suggested that FRS, though observed in other disorders at times, were much more common among schizophrenics. Passivity phenomena were the commonest. Auditory hallucinations alone were of no diagnostic value. Similar observations were made in patients with schizophreniform disorder and schizophrenia after the age of 45.

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References

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders, 3rd Ed.* (DSM-III). Washington, D.C.: American Psychiatric Association.

MELLOR, C. S. (1982) The present status of first-rank symptoms. *British Journal of Psychiatry*, **140**, 423–424.

SCHNEIDER, K. (1959) *Clinical Psychopathology*. Engl. Transl. New York: Grune & Stratton.

Folate and Vitamin B_{12} in Eating Disorders

SIR: We investigated serum folate and vitamin B_{12} in a series of patients with eating disorders in comparison with an age-matched group of normal subjects, and examined the relationship between their deficiency and the presence of depressive symptoms and weight loss. Forty-five female anorectic outpatients (cross-section study), nine of whom were in-patients (longitudinal study) were studied. Folate and vitamin B_{12} levels in this group were compared with their levels in 7 normal weight bulimic patients and 14 normal controls. Anorectic patients showed severe folate deficiency (less than 1.0 ng/ml) in 37% of cases, a significantly greater rate than that of bulimic patients (14%) and normal controls (11%) ($P<0.01$). There were no significant differences between rates of vitamin B_{12} deficiency (less than 150 pg/ml) between anorectics (22%), bulimic patients (14%) and normal controls (11%). Fifteen anorectic patients who were assessed to have mild depression (Hamilton Rating Scale score of 10 or above) showed a trend for lower folate concentrations than those with HRS less than 10 ($P<0.07$). There was no association between folate levels and weight in the anorectic group. Patients studied in series showed an increase in folate levels in association with their increased weight and fewer depressive symptoms. In one patient who had 10 folate estimations there was a significant correlation between folate levels and HRS scores ($r=-0.7$, $P<0.05$). Anorectic patients who were receiving psychotropic medication had a significantly greater rate of folate deficiency than those who were drug-free, an association that was mediated by greater affective morbidity exhibited by the former group. These findings further confirm the association of folate deficiency and depressive symptoms in a group of patients with anorexia nervosa, highlighting the role of folate in the regulation of mood (Reynolds & Stramentinoli, 1983; Abou-Saleh & Coppen, 1986).

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ABOU-SALEH, M. T. & COPPEN, A. J. (1986) Psychiatric Progress: the biology of folate in depression, implications for nutritional hypotheses of the psychoses. *Journal of Psychiatric Research*, **20**, 91–101.

REYNOLDS, E. H. & STRAMENTINOLI, G. (1983) Folic acid, S-adenosylmethionine and affective disorders. *Psychological Medicine*, **13**, 705–710.

Koro in an Israeli Man

SIR: I read with interest the Modai *et al* Case Report (*Journal*, October 1986, **149**, 503–506), adding another patient to the literature of Koro among non-Chinese subjects. These authors stated that there are only eight case reports of Koro from the Western hemisphere. I wonder what is the demarcation of their western hemisphere, because in 1984 features of Koro-like symptoms among 16 subjects culturally unrelated to China or Indonesia were discussed by Berrios & Morley (1984). Since then five more patients have been reported: (Ang & Weller, 1984; Emsley 1985; Moodley, 1985; Oyebode *et al*, 1986) making a total of 21. Even though Modai *et al* presented a primary Koro patient, I am not convinced of the typicalness of their patient: because of 22 non-Chinese subjects (including the authors' case) only Barrett's (1978) patient presented with all Koro symptoms i.e., (1) shrinking of the penis; (2) into the abdomen; (3) associated with fear of death. Lapierre's (1972) patient presented with shrinking of penis associated with fear of death and the remaining 20 patients presented with only shrinking of the penis.

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References

ANG, P. C. & WELLER, M. P. I. (1984) Koro and psychosis. *British Journal of Psychiatry*, **145**, 335.

BERRIOS, G. E. & MORLEY, S. J. (1984) Koro-like symptoms in a non-Chinese subject. *British Journal of Psychiatry*, **145**, 331–334.

EMSLEY, R. A. (1985) Koro in non-Chinese subjects. *British Journal of Psychiatry*, **146**, 102.

MOODLEY, P. (1985) Koro in non-Chinese subjects. *British Journal of Psychiatry*, **146**, 102–103.

OYEBODE, F., JAMIESON, R., MULLANEY, J. & DAVISON, K. (1986) Koro: A psychophysiological dysfunction? *British Journal of Psychiatry*, **148**, 212–214.

British Journal of Psychiatry (1987), **150**, 134–143

Book Reviews

Editor: Sidney Crown

Psychological Medicine: An Introduction to Psychiatry (10th edition). By PETER STOREY. Edinburgh: Churchill Livingstone. 1986. Pp. 484. £12.95.

This is the tenth edition of the well known textbook for students, founded in 1943 and for many years known as "Curran and Partridge". Partridge died in 1984, and Sir Desmond Curran has died since this edition went to press, so the enterprise is now Storey's, and has been brought up to date.

The introduction has the flavour of civilised commonsense, plus great experience. The author is excellent at explaining how and why psychiatry is part of medicine, and other personal themes include the great importance of work in people's lives and the limiting effect of constitution, as opposed to physical and psychological causes of disorder, in damping enthusiastic claims of cures. The general chapter on aetiology is an excellent essay on the subject. It is followed by a chapter on symptoms and the author's classification (clear and reasonable, but there is no mention of ICD-9 or DSM–III) and then by one on case taking.

The chapters on illnesses are in general as up-to-date and thorough as they should be at this quite extensive length for a students' book (although trazodone is still in use and involutional melancholia being diagnosed). There is a separate chapter for general aspects of treatment, and it is noticeable that physical methods (ECT is allocated 6 pages, and psychosurgery 5 pages) are well covered, perhaps more enthusiastically than psychotherapy, which nevertheless gets 20 pages. Behaviour therapy and cognitive therapy are covered very briefly.

There is a new chapter on psychosomatic conditions, and extensive updating on anxiety, hysteria and mental handicap. There is no coverage of child and adolescent psychiatry. The chapter on the law covers thoroughly the Act of 1983 in England and Wales, and 1984 in Scotland.

The style is conversational and readable, the text well printed, the reading list poor and unrevised, and the index excellent. Overall, the standard of earlier editions is well maintained, as befits one of the best general textbooks.

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Family Approaches to Major Psychiatric Disorders. Edited by MELVIN R. LANSKY. Washington: American Psychiatric Press. 1985. Pp. 147. $12.00.

This book is a collection of papers from a symposium entitled "Family Therapy and Major Psychopathology". The central themes for the papers arose from an earlier book in which warnings were given about the dangers of splitting off family therapy from other therapeutic interventions. The main theme is therefore about integration of various therapeutic approaches which take account of the patient, the family and the wider systems. A second and related theme is to highlight for each major disorder the special problems presented.

The editor in his excellent introduction traces the historical and psychological development of family therapy. He sees family therapy as having recently gone through its rebellious adolescence, thumbing its nose at boring old psychoanalysis and conservative psychiatry. Now in its maturity, family therapy is ready to manage its own identity and allow the identity of others and therefore be one of several integrated treatments.

It is a short collection of only six papers, each emphasising the need to serve the interests of the patient and his family in whatever way seems best. In the first paper on the Narcissistically Vulnerable Marriage, Lansky gives a clear account of the use of psychoanalytic understanding in family therapy. The chapter on alcoholism highlights the special problem of abstinence and the multifaceted nature of the disorder with its genetic background and relationship to depression and the social, cultural and family factors. Each paper has something special to offer. The one on bipolar marriages emphasises the crucial role of medication, especially since it makes the patient and his family more amenable to psychotherapy and multiple family group therapy. The now familiar psycho-educational approach in schizophrenia is reviewed and described verbatim in an appendix. The role of education is again seen as part of an ongoing family treatment plan.

The last two papers are on organic brain syndrome and cancer. Both describe not only the traumatic effects of these conditions on families and patients but how they may emphasise pre-existent pathological patterns. Family treatment is seen as vital in both cases but needs to be combined with that of the health care systems. The paper on cancer determinedly and touchingly emphasises life as much as death.

I found this an interesting and sensitive book. The authors emphasise the stress and burden of chronic illness as well as the need to be extremely careful about inducing guilt in families. They also pay particular attention to their own countertransference.

This book is well worth reading and is to be recommended to all those who work with these extremely difficult problems.

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Self Injurious Behaviour: A Collection of Published Papers on Prevalence, Causes, and Treatment in People who are Mentally Handicapped or Autistic. Edited by GLYNIS MURPHY and BARBARA WILSON. Kidderminster: BIMH Publications. 1985. Pp. 455. £19.95 (hb), £9.95 (pb).

This is a valuable book that brings together a collection of 54 previously published papers extensively covering the topic of self-injurious behaviour in mentally handicapped and/or autistic individuals. It is divided into three sections, dealing with the areas of prevalence, development and maintenance of self-injurious behaviour and treatment. Each section is subdivided and covers such areas as animal studies, self-injury in non-handicapped individuals, and various hypotheses that have been proposed to explain such behaviour.

Why people who are mentally handicapped or autistic injure themselves is not known, but between 5 and 15% exhibit this form of behaviour at some time in their lives. The papers in this book examine a series of hypotheses including developmental aspects, the role of infections (e.g. otitis media), sensory stimulation as reinforcement, learned behaviour and psychodynamic views. The main message of this collection of papers is that proper evaluation and observation is essential before, during and after intervention whether this be behavioural, medical or the use of protection. What initiates the behaviour may well be different from what perpetuates it, and what has been shown to diminish behaviour in one circumstance may well increase it in others. There are also clearly ethical issues to be considered, particularly with adversive therapies, and the danger that the emotional impact of the self-injurious behaviour on others (care staff and family) may precipitate them into ill-informed and inappropriate treatments. This book is an excellent reference work but is not a guide to management. In the introductory sections there is discussion concerning management and many of the papers deal with treatment issues, but there can be no substitute for experience and careful thought when it comes to the treatment of such difficult behaviour problems.

Self-injurious behaviour can occur in a variety of handicapping conditions but its association with the Lesch Nyhan syndrome is particularly perplexing. The papers by Nyhan and others illustrate how varied the degree of handicap and how severe the behaviour can be. The largest number of treatment papers deal with the use of psychological techniques. Properly applied these clearly can be effective, but some papers report single cases and how well such treatment can be generalised to other circumstances is questionable.

Readers of this book have to be critical and selective but this is undoubtedly a valuable reference work in an area of study neglected by many professionals.

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Job's Illness: Loss, Grief and Integration. A Psychological Interpretation. By J. H. KAHN. London: Royal College of Psychiatrists. 1986. Pp. 232. £10.00.

The *Book of Job*, and in particular its titular hero, Job himself, have for ever been the subjects of heated debate among Talmudists and theologians. The manifest problem posed is: how is it possible for the life of a man like Job, a man of unsurpassed generosity and piety, to be suddenly shot through with the heavy artillery of disaster, transforming it into a personal hell? There are those scholars, Talmudists included, who would maintain that Job never existed, and that the narratives in the drama are allegorical and devised to answer the burning question: "why is it the righteous who suffer? Why are the innocent slaughtered?".

The pages of history are repeatedly stained with soul-searing illustrations. Take as recent examples the victims of the chemical disaster at Bhopal, and, even worse, those who died or will inevitably die as a result of the Chernobyl nuclear catastrophe; or the countless thousands dying of famine in Ethiopia and elsewhere. Go back forty years or so and ponder the obscene maltreatment of Jews and other innocents in Hitler's Germany and Stalin's Russia. All these helpless victims – the list is never-ending – are entitled to share the agony of Job; like him they are lost in a meaningless universe and abandoned by their God or gods. The answer to the problem, persuasive in its simplicity, is to be found in the allegory, namely, that man must rest in faith and resign himself to the mysterious and, offentimes, incomprehensible ways of God.

The allegorical explanation is, however, rejected by some scholars, including Dr Kahn, the author of this book. He, a practising psychoanalyst whose cultural roots are steeped in the Judeo-Christian tradition, is eminently qualified to explore an alternative explanation. He insists that there is a valid psychological interpretation.

Dr Kahn argues that Job was a real person who, apart from his somatic complaints, suffered from a combination of three psychiatric syndromes; obsessional neurosis, paranoia, and, most importantly, depression. The book, he infers, is in effect a case history of Job's complex illness offering to its readers an insight into its aetiology and treatment, the latter, incidentally, including something akin to what today would be called group therapy.

For myself, I have to confess that I am unconvinced by Dr Kahn's argument; but, then, I am an unbeliever in both the biblical and psychoanalytic sense. Nevertheless, I would be the first to acknowledge Dr Kahn's profound scholarship and would salute the courage of his undertaking.

It should be added that his book was first published in 1975 by Pergamon, and the College is to be congratulated for being the means whereby a second edition of such an intensely stimulating piece of research is again made available.

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Study Guide and Self-examination Review for Understanding Human Behaviour in Health and Illness. 3rd edition. By MARITA J. KEELING and RICHARD C. SIMONS. London: Williams & Wilkins. 1986. Pp. 348. £13.00.

This is an attractively laid out book consisting of questions (mostly multiple choice), answers and explanations. Although the questions are generally of a high standard, unfortunately none of the multiple choice type follows the multiple true-false format used predominantly in medical examinations in this country.

Clearly, the book will be of most value when used in conjunction with its parent text (Simons, 1985), a book aimed primarily at medical students. Herein lies another possible limiting factor, because I suspect that the parent book is not widely used or available in this country. This is in some ways a pity because, having had a look at it, I find it impressive in its attempt to integrate clinical aspects of behaviour and the basic sciences and so make the former more comprehensible. However, it does have a strong transatlantic flavour as evidenced by, for example, the choice of references and, perhaps as a result of so much psychology being included, the psychiatry sections are rather scanty.

On the positive side, however, the question book has a strong appeal which stems from the philosophy of the parent text and lies in its coverage of topics often neglected. For instance, subjects covered include the nature of the doctor-patient relationship, the 'difficult' patient, psychological reactions to pain and physical illness, pregnancy, grief and mourning, sexuality, the dying patient, work and creativity.

Alas, I think the limitations I have mentioned will outweigh the positive features for most medical students and trainee psychiatrists in this country, although I think it and the parent textbook are reasonably priced at £13 and £26 respectively. Libraries, however, could usefully stock both books.

ROGER FARMER, *Consultant Psychiatrist, University College Hospital, London*

Alcohol, Drugs, and School-Leavers. By MARTIN A. PLANT, DAVID F. PECK and ELAINE SAMUEL. London: Tavistock. 1985. Pp. 190. £16.95.

This book is about a group of fifteen to sixteen year-old Scottish school children, whose use of alcohol and other psycho-active drugs was followed up for four years. The research project was originally conceived as being solely an alcohol study, but during the course of the study illicit drug use and tobacco smoking were also investigated. The aims of the original research project were, firstly, to determine whether knowledge, attitude and behaviour concerning alcohol among school leavers predict subsequent alcohol-related behaviour, and secondly, to find out whether educational programmes meet their objectives. With regard to the second aim, since none of the schools provided alcohol education in a systematic or elaborate way, it could not be adequately measured, and therefore the study cannot be regarded as a very useful evaluation of alcohol education. With regard to the first aim, the study was more interesting. It found that future alcohol use was not predicted by the student's knowledge, attitudes or behaviour concerning alcohol. However, both high alcohol consumption and alcohol-related consequences were highly correlated with *not* being brought up by the natural father and mother. I found much of interest in this book, both as a Consultant Psychiatrist specialising in alcohol problems, and also as a School Governor. In my particular school, I have used my psychiatric skills not in direct alcohol education, but indirectly by devising courses in human relationships to help the young people to prepare for marriage, in the belief that helping young people to turn to other people will make them less likely to turn to alcohol. If in addition these courses succeed in helping young people to form stable marriages, then alcohol problems in the next generation should be reduced. This book confirmed my view that in the present state of knowledge, alcohol prevention by such indirect methods is likely to be more fruitful than direct alcohol education in schools.

T. A. Spratley, *Consultant Psychiatrist, St Martins Hospital, Canterbury*

Advocacy and Empowerment: Mental Health Care in the Community. By Stephen M. Rose and Bruce L. Black. London: Routledge & Kegan Paul. 1985. Pp. 231. £29.95.

This book documents the problems of discharged, institutionalised patients from psychiatric hospitals in New York State. These problems, for example 'the revolving door', will be familiar to those working in the British context, though the volume and intensity of the difficulties may be less than in the American situation. The authors indict the change from hospital to community care as being merely 'a drift in the locus as opposed to the focus of the mental health delivery system'. It is argued that there was a failure to transfer funding from the State Hospitals to community care as the former were being run down. Of more importance to the authors is the carry over of the medical model of care from the hospitals to the community. There follows a fairly standard diatribe against medical hegemony during which almost every noun with negative connotations is used.

The alternative approach, proposed and described by Rose and Black, for ex-patients in the community is a somewhat confrontational one known as 'advocacy and empowerment'. This is based on the theories of a number of sociologists and educators. In essence, it involves an understanding by the worker of the client's behaviour and view of the world in the light of experiences within the psychiatric system. The relationship built up between worker and client is used to help the client to become active and creative in gaining the knowledge and skills for life in the community. Confidentiality criteria for workers are strict but the relationship with the client usually gives rise to authorisation for the worker to advocate on the client's behalf. Since this approach starts from the assumption that clients are oppressed persons as a result of their experiences, advocacy often leads to conflict with other more conventional agencies.

The book is strong on theoretical perspectives, principles, frameworks, processes and programmes, yet examples from practice are few and, when given, tend to be 'snapshots' relating to uncoordinated issues. The book lacks case histories which might over a period of time in the life of a client illustrate the benefits of the advocacy/empowerment approach. This seems particularly important since, although objective evaluation strategies are described and criticised, no results are forthcoming. A lingering impression remains that the principal driving force is the commitment to the ideology.

Donald P. Forster, *Senior Lecturer in Community Medicine, University of Newcastle upon Tyne*

Pharmacological and Behavioural Treatment: An Integrative Approach. Edited by Michel Hersen. New York: John Wiley. 1986. Pp. 400. £45.50.

This book is one in a large series on Personality Processes, the emphasis of which is principally on psychological aspects of assessment and management of various types of personal problems and psychiatric disorders. This volume seems to emphasise psychological aspects more than pharmacological ones in many chapters. In fact, pharmacological and psychological aspects of management are often discussed separately for a large section of a chapter with only a relatively short section on their combined use, as in the sections on Obsessive/Compulsive Disorder and Neuropsychologic Disorders.

The book is divided into three sections: General Issues, Child Disorders and Adult Disorders. The first section provides a brief review of general issues related to drug treatment, more appropriate for non-medical graduates, a general description of various behavioural techniques and discussion of methods for evaluation of treatment effects. In Child Disorders the value of drug treatment is perhaps overemphasised, particularly with regard to Conduct Disorders and Learning Disabilities. Under Adult Disorders several psychiatric illnesses are discussed in separate chapters: Unipolar Depression, Obsessive/Compulsive Disorder, Phobia and Anxiety, Substance Abuse, Schizophrenia, Psychophysiological and Neurophysiological Disorders. A few of the chapters provide a reasonably critical review of the available literature, such as those on Autism and Obsessive/Compulsive Disorder, but many do not. The chapter on Unipolar Depression, for example, tends to quote references with little critical examination of them and, like many of the other chapters, relies rather heavily on results from single case studies. The chapter on Schizophrenia examines the effects of neuroleptic drugs on psychological functioning and the effects of psychosocial interventions rather than the combination of drugs with behaviour therapy.

Overall, I felt the book was rather weak on living up to its title. Perhaps this is because there is not a wide literature on the combination of treatments? I doubt if this book would be of great value to those with a fairly eclectic training as exists in psychiatry in Britain.

STEPHEN J. COOPER, *Senior Lecturer,*
Department of Mental Health,
The Queen's University of Belfast

Psychotherapy and the Grieving Patient. Edited by E. MARK STERN. New York: The Haworth Press. 1985. Pp. 138. $19.95.

This book was also published as *The Psychotherapy Patient*, Volume 2, Number I, February 1986. It is a collection of essays on diverse aspects of bereavement. It seems quite possible, with tighter editing, to bring the whole volume down to a single article. There is little new inside these pages, and that which is there is perhaps less rigorously presented than we are used to on this side of the Atlantic. Most of the individual 'illustrative' case reports are simply not detailed enough for any objective analysis to be made by the reader of therapeutic method and outcome. Dr Stern, a Professor of Pastoral Counseling, describes the case of Erin, a lady who unfortunately killed herself. One doesn't need to read between the lines to come away with the strong impression that her "bursting with joy" and her inability to recall "a day of sunshine" in her past, together with her excessive drinking, "self-imposed isolation", and her search for "tidbits of hope", not to mention such items as her coffin-buying escapades, just might have responded to lithium or some such alternative medication.

Dr T. L. Brink, co-author of the 'Geriatric Depression Scale', tells us that the ideal therapist is he who is sought out by the client, the ideal therapy depends on factors such as education and degree of dementia, and so on. One cannot help but feel that he has modified his normal way of thinking in order to keep step with the general tenor of the book.

One is fascinated by Edward Tick's catharsis when it comes to his discussion of the "psychohistoric factors affecting the United States people", terminating in a cry for grieving for the Vietnam experience. Undoubtedly Vietnam was a terrible experience for many millions of Americans. My point is that there seems to be some little lack throughout the book of standing back, of not becoming enmeshed in the subjective rationalisations of the patient. The chapter on adult incest survivors, one of the best in the book, is marred by liberal use of snippets of comments made by clients, used to illustrate dynamics that the author wishes to prove, whereas, I feel, fuller case histories would have been more credible.

This volume is a product of American private practice. It is written by authors of many persuasions. There is much modern knowledge about grief which is not contained between its covers, and its references are not reflective of the wider world of thinking. Those on a tight library budget could do worse than look elsewhere.

BRIAN O'SHEA, *Consultant Psychiatrist,*
Newcastle Hospital, County Wicklow, Ireland

The Integration of Neuroscience and Psychiatry. Edited by HAROLD ALAN PINCUS and HERBERT PARDES. Washington: American Psychiatric Press. 1985. Pp. 94. $12.00.

This short monograph is based on the Proceedings of the 1984 Annual Meeting of the American Psychiatric Association, which was devoted to the topic of Neuroscience and Psychiatry.

The chapters provide a graduated sequence from the general to the particular. In Chapter 1, Pardes and Pincus review the new techniques now available for the investigation of brain function, including positron emission tomography (PET), nuclear magnetic resonance (NMR) and brain electrical activity mapping (BEAM). In Chapter 2, Post cites a variety of examples of the application of neuroscience to clinical diagnosis and treatment, including the investigation of affective and schizophrenic psychoses by PET, NMR and BEAM and the introduction of clonidine for opiate-withdrawal and carbamazepine in the treatment of bipolar psychosis. For good measure he adds a brief review of neuropeptides and behaviour. In Chapter 3, Paul reviews recent advances in neurochemistry with particular reference to dopamine and schizophrenia and the role of cholecystokinin as a co-transmitter. In Chapter 4, Wexler, Gusella, Conneally and Housman describe their recent successful application of molecular genetic techniques to the study of Huntington's chorea and face up to the difficult ethical issues involved in its pre-symptomatic diagnosis.

In the final chapter Pincus and Rubinow explore the possibilities for research into the relationship between medical and psychiatric disorders, employing the new data and techniques. A noteworthy contribution is their series of models depicting possible modes of interaction between psychiatric and medical conditions, reminding us that correlation does not necessarily indicate causation.

This small volume provides a brief yet stimulating review of some of the important growing points in biological psychiatry. It will be of particular value to trainee psychiatrists and anyone wishing to keep abreast of this rapidly developing subject.

K. DAVISON, *Consultant Psychiatrist,*
Newcastle General Hospital, Newcastle upon Tyne

Hyperkinetic Children: A Neuro-Psychosocial Approach. By KEITH C. CONNERS and KAREN C. WELLS. London: Sage Publications. 1986. Pp. 160. £19.75 (hb). £10.00 (pb).

This is the seventh volume in the Sage series in Developmental Clinical Psychology and Psychiatry. The authors are two of the best-known American authorities on hyperkinetic children. Publication coincides with the issue of a book covering similar ground edited by one of the leading United Kingdom researchers in the field (*The Overactive Child.* Edited by E. A. Taylor. Oxford: Spastic International). Comparisons are inevitable, in particular because there are such differing views about diagnosis, aetiology and treatment in the USA and Britain. There are many similarities, but importance differences.

Both books are intended to be up-to-date presentations of theory, research and practice for professionals working in the field, whatever their discipline. Both consider concepts of hyperactivity and hyperkinesis, and both review the historical development of the concepts, although this is fuller in the Taylor book. Both consider epidemiology and both draw heavily on research done by the authors or their associates, while reviewing relevant literature. However, the group at the Institute of Psychiatry is engaged in more recent or current research on the topic; their coverage of epidemiology is more extensive, and their review of the work of others more thorough and far-ranging. Both books look at aetiology and examine specific behaviour such as overactivity, attention and impulsiveness, and report on treatment. Conners and Wells are more detailed in their discussion of electroencephalographic research, but for all other aspects the Taylor book is to be preferred. The conclusions drawn by Conners and Wells are usually balanced, and often not vastly different from those of Taylor and his colleagues, but the approach of the latter group is altogether more carefully considered. Furthermore, it is more thorough and enlightening in the elucidation of relevant concepts such as attention and impulsiveness; social aspects receive greater weight, and treatment is reviewed at more length.

The paperback version of Conners' and Wells' book is cheaper and handier but in hardback Taylor's book is less expensive and almost twice as long. Although *Hyperkinetic Children* is in general a readable and sensible account, most professionals would be advised to choose *The Overactive Child.*

ANTHONY D. COX, *Consultant Child Psychiatrist/*
Senior Lecturer, Institute of Psychiatry, London

The Selected Melanie Klein. Edited by JULIET MITCHELL. Penguin: London. 1986. Pp. 256. £4.95.

This is a small but useful book. Juliet Mitchell uses a compact historical approach to demonstrate the development of Klein's work through a collection of key papers, each one prefaced by a short commentary. She is herself a psychoanalyst as well as a feminist. She writes well and in her short introduction she gives some biographical facts and explains certain basic psychoanalytic ideas both from Freud and Klein and highlights the difference between the two. This is an interesting section of the book but it is not entirely satisfactory. She seems uncertain about her readers' understanding of psychoanalytic issues so that on the one hand she carefully explains that dreams and symbols cannot be understood on an *ad hoc* basis and yet on the other speaks about the resolution of the oedipus complex as a result of castration anxiety without any explanation whatsoever of any of these concepts as if everyone should understand immediately what Freud was talking about. She gives some sloppy definitions of concepts such as splitting and projective identification which could spoil a deeper grasp of the issues from the text.

Klein, of course, differs from Freud in many important ways and Juliet Mitchell highlights the difference between Freud's view of the dynamic unconscious as a reservoir of historical repressions contrasted with Klein's view of the internal world as constantly present structuring the individual's internal and external experiences in a much more direct and immediate way, hence unconscious processes including symbols, mental mechanisms and fantasies are far less disguised when compared with the classical viewpoint. The very short introduction, however, did not allow the refinement of these differences, so misconceptions could very easily arise. Although Klein did not see patients as suffering from reminiscences in the same sense as Freud, nevertheless she strongly believed in the memory of pre-verbal experiences which she called 'memories in feeling' and when splitting and projective identification lessen they can be re-experienced in a most extraordinarily detailed way.

In spite of these reservations the book is a valuable contribution which should be enjoyed by psychiatrists interested in psychoanalytic ideas and will be a very useful addition to postgraduate libraries wanting to be representative but not overloaded with large quantities of psychoanalytic works.

P. L. G. GALLWEY, *Consultant Psychiatrist,*
St George's Hospital, London

American Psychiatric Association Annual Review Vol. 5 (Psychiatry Update Series). Edited by ALLAN J. FRANCIS and ROBERT E. HALES. Washington: American Psychiatric Press. 1986. Pp 791. $36.95 (pb), $49.95 (hb).

The fifth Annual Review contains six sections, dealing with (i) *schizophrenia* (section editor, Nancy C. Andreasen): "This section is uncommonly well-balanced and of uniformly high quality. Each chapter is scholarly and careful, yet clinically rich and pleasing to read" (Frederick K. Goodwin); (ii) *drug abuse and drug dependence* (section editor, Robert B. Millman): "presents the latest research from the field's most knowledgeable and experienced psychiatrists" (Lawrence G. Kolb); (iii) *personality disorders* (section editor, Robert M. A. Hirschfeld): "In masterfully written chapters, this section . . . could well stand on its own as a definitive treatise on human personality and its disorders" (John C. Nemiah); (iv) *adolescent psychiatry* (section editors Carolyn B. Robinowitz & Jeanne Spurlock): "The editors selected timely and critical topics . . . The chapters represent essential information in a highly readable style" (George Tarjan); (v) *psychiatric contributions to medical care* (section editors David Spiegal & W. Steward Agras): "an extraordinary collection of essays. They have great depth in the review of diagnostic issues and treatment approaches to the entire gamut of psychosomatic problems" (Paul J. Fink); and (vi) *group psychotherapy:* "without a doubt the best review of the present state of the art of group therapy that I have come across to date. Each of the six chapters is highly informative, sophisticated, and lucidly written. Anyone who practices group psychotherapy or hopes to, should read this section" (Judd Marmor).

The material is less outstanding on international and cross-cultural perspectives and shows a singular regard for DSM-III.

GREG WILKINSON, *Honorary Lecturer,*
Institute of Psychiatry, London

Young Children in Family Therapy. By JOAN J. ZILBACK. New York: Brunner/Mazel. 1969. Pp. 177. $25.00.

This book, a welcome addition to the growing literature of family therapy, is the first dedicated to the specifics of including younger children in family therapy. From the outset, Zilbach draws our attention to the curious position children occupy in family therapy. She contends that younger children are often excluded from therapy sessions and this is reflected in the paucity of literature dealing with children. This book is her attempt to redress the balance.

Zilbach has drawn upon her considerable experience to produce this book. The style is clear and concise. While primarily a practical manual, Zilback has not neglected theory. She presents a selection of quotations from the pioneers of family therapy, such as Ackermann, Satir, Minichin and Whitaker, emphasising the value and necessity of working with the entire family. Zilbach demonstrates with examples how children can promote understanding of family interaction by revealing family secrets in drawing and in play as well as acting, at times, as an ally or co-therapist.

The book is beautifully illustrated, with photographs of sessions and of children's drawings. These are well intergrated into the text of the case vignettes and the therapeutic use of the drawings is made very clear. The author acknowledges her psychoanalytic perspective, but in no case is an interpretation made which is not straightforward and readily understood by the family.

Overall, this is a very good book. It should be recommended reading for those professionals dealing with families with younger children. The author ought to be congratulated not only for informing us of the benefits of working with younger children, but alterting us to the dangers of excluding them.

A. C. JAMES, *Senior Registrar in Child and Adolescent*
Psychiatry, Maudsley Hospital, London

Roots and Shoots: A Guide to Counselling and Psychotherapy. By R. F. HURDING. Hodder & Stoughton, London. 1986. Pp. 464.

The author traces the rise of secular psychologies and examines Christian responses to them. The first half of the book examines in some detail the psychological theories from which and on which the practical counselling and psychotherapy is based. It does so in some detail ranging from Behaviourism, through the analytical schools, to the New Therapies. It is well documented and gives the reader an overall view of the broad spectrum covered by the generic concept of "psychology".

In the second half, the author faces the awesome task of presenting the Christian reaction and response. On the whole this might better be described as the Christian Biblical response. The problem which he faces can be seen if we were to turn the process round and give a treatise on the variety of Christian denominations and then give the psychological response to each.

The inherent danger in such a work is that it may appear to conclude that only a Christian can be a therapist or benefit from therapy. While some of the Christian responses might be taken in this light I would perfer to consider the relationship in two ways. Psychology has to presume a philosophy, which may well be Biblical, and the quality of the therapist may well spring from his or her spiritual strength.

This book provides an important source of understanding both for the Christian of psychotherapeutic theory and for the psychotherapist of the Christian response.

LOUIS MARTEAU, *The Dympna Centre, London*

Child Development and Psychopathology. By DONNA M. GELFAND and LIZETTE PETERSON. London: Sage Publications. 1985. Pp. 156. £19.75 (pb).

This book, the first of a series devoted to developmental clinical psychology and psychiatry, reads more as a collection of five essays than as a coherent account of the relationship between child development and psychopathology. The use, or abuse, of the English language throughout the book was also irritating. Phrases such as "the relationship between developmental and clinical child psychology as a well known 'smouldering civil war', in which those who are naturally close to each other have engaged in combat" and "Fortunately, ideological strife is not ubiquitous" reminded me more of Rambo than of Piaget. There is little recognition that empirical research has significantly altered views about child development. I cannot recommend this book.

D. P. HOARE, *Senior Lecturer in Child and Adolescent Psychiatry, University of Edinburgh, Royal Hospital for Sick Children, Edinburgh*

Animal Models for Psychiatry. By J. D. KEEHN. London: Routledge & Kegan Paul. 1985. Pp. 251. £20.00.

Professor Keehn draws on case material and experimentation to illustrate some of the ways in which animal models can have psychiatric value. Early chapters discuss the status of animals and describe many examples of abnormal animal behaviour. Particularly welcome in a work ultimately concerned with the alleviation of suffering is a chapter devoted to the welfare of animals and the ethics of research. However, an initial section clarifying basic concepts (definitions of abnormality, models in psychology, reductionism) would have been helpful to newcomers to the subject. Later chapters describe attempts to increase our understanding of human abnormal behaviour by the use of animal models. The topics covered include neuroses, substance abuse, psychotic behaviour, and depression. Many of the models discussed are from the "Skinnerian" school of experimental psychology and from ethology.

The strength of this book is that it sets out some of the contributions of animal models in an interesting and positive way. Its weakness is a lack of critical appraisal. The usefulness of animal models is more frequently asserted than justified; and the models are not convincingly assessed in the context of alternatives. The claim in the concluding chapter that "Animal models may be all that we have for finding new ways to treat the mentally ill" (p. 207) requires more justification than this book provides, and would, I am sure, be firmly disputed by many practitioners working with cognitive and humanistic approaches.

GLYN V. THOMAS, *Lecturer in Psychology, University of Birmingham*

Social Work, Psychiatry and the Law. By NORMAN N. PRINGLE and PAUL J. THOMPSON. London: William Heinemann Medical Books. 1986. Pp. 241. £8.95.

The authors have tried to bring together an outline of psychiatry, social work and the current law relating to the mentally disordered. Part I gives a superficial account of the main psychiatric disorders (including the disturbances of childhood and adolescence). Psychopathic disorder is inadequately dealt with and the M. H. Act definition is given twice. Morbid jealousy is dealt with in the section dealing with aggressive psychopathy. Mental Impairment is dealt with in 4 pages, and the chapter on Methods of Treatments is a curious assemblage of poorly connected facts. Part II is concerned with the social work practice and legislation. The sections on the role of the A.S.W. in assessment are quite helpful but the authors' appraisal of the law is patchy. The wrong date is given for the 1890 Lunacy Act; Section 117 of the 1983 Act is concerned with the after-care of patients *who have been detained*; the fact that the Code of Practice is a *Draft* is not made clear and the authors imply that it has been promulgated by the MHAC whereas it is in fact a DHSS document. The very brief material dealing with admissions from the courts could mislead the ill-informed, and the references to further reading at the end of each chapter are not clearly related to the matters discussed in the text. As a representative of social work I am distressed that I cannot give this social work contribution a positive recommendation.

HERSCHEL PRINS, *Former Director, School of Social Work, University of Leicester*

Brain Neurotransmitters and Psychiatry. By A. VILLENEUVE. Obtainable free of charge from Astra Pharmaceuticals Canada Ltd, 1004 Middlegate Road Mississuaga, Ontario, Canada L4Y 1M4. 1985. Pp. 108.

This book is a small treatise in which distinguished research workers, mainly from Canada, but also from France and the United States, contribute a series of articles bearing strictly on neurotransmission and its relationship to events both inside and outside the brain. The short crisp chapters include the "Effects of Hormones on Brain Neurotransmitters", "Behavioural Approaches of the Biochemical Mechanism of Action of Psychoactive Drugs", "Indolamines in Affective Disorder and Suicide", and "Psychiatric Aspects of Neuroendocrine Disorders". Dopamine and dopaminergic systems, norepinephrine, neuromodulators, and the role of neurotransmitters in anxiety are carefully considered. Sources are fully documented throughout, but there is no general index. This slender volume is easy to read, and moderately easy to understand. It should be particularly useful for junior staff already familiar with basic neuroendocrine physiology and biochemistry.

JOHN POLLITT, *Medical Director, Hayes Grove Priory Hospital, Kent*

Marital Therapy in Britain. Edited by WINDY DRYDEN. **Vol. 1. Context and Therapeutic Approaches.** Pp. 349. $8.50. **Vol. 2. Special Areas.** Pp. 350. £8.50. London: Harper & Row. 1985.

I cannot say that I can strongly recommend either of these volumes. There are some excellent chapters, particularly those of Treacher and Street, who are recognised teachers and practitioners of family therapy. The latter includes a quite proper discussion on the relevance of marital therapy, particularly when there are children. I believe Street agrees with me in suggesting that such a course of therapy would be irrelevant and that family therapy would be the treatment of choice. This, of course, does not apply where there are clear and discrete sexual difficulties and there are a number of good chapters addressing this particular aspect of therapy, which included a stimulating and controversial chapter by Martin Cole on the use of surrogate therapists, which leaves many questions tantalisingly unanswered. The Institute of Marital Studies have contributed excellently to this book and maintain the high standard of their publication since their inception. The section on research inevitably leans heavily on sources outside Britain since, with the exception of Michael Crowe, there is not much to write about within the United Kingdom.

JUSTIN SCHLICHT, *Consultant Psychotherapist, The Maudsley Hospital, London*

The Death of Desire. A Study in Psychopathology. M. GUY THOMPSON. New York: New York University Press. 1985. Pp. 215. $35.00.

The author attempts to bridge the gap between phenomenology and psychoanalysis. Phenomenologists do not accept Freud's concept of the unconscious. They are concerned with "appearances", and "for Freud, the unconscious *does* appear – as 'desire' ". This desire is not Freud's libido. Thompson defines it (with Hegel) in terms of the subject's existential appeal for recognition by another one, rooted in "a lack at the heart of being".

The "death of desire" is its elimination by denial or the illusion of fulfillment. For Thompson, all psychopathology springs from that death. The "murderer" is the ego, here not seen as the carrier of identity but (echoing Lacan) as the sum of identifications, and the seat of all resistance to therapy. Neurotic and psychotic symptoms are in the service of the flight from the pain of unfulfilled desire. It is the therapist's task to help the patient to face this pain. Therefore, the therapist needs to avoid collusion with the patient's ego.

There is an important chapter on the place of time in psychopathology and treatment, influenced by Heidegger's concept of temporality. There is an interesting redefinition of perversion as *any* sexual activity that does not "desire" the recognition of a partner, so that also "normal" heterosexual acts could at times be called perverse. A concluding chapter surveys the role of language in psychoanalysis.

The book is lucidly written but not easy to read, as it raises very complex issues. I recommend it warmly to anyone interested in the philosophical aspects of psychoanalysis.

H. COHN, *Psychologist, Fabye House, Kew, Surrey*

Behavioural Case Formulation. Edited by IRA DANIEL TURKAT. New York: Plenum Press. 1985. Pp. 321. $35.00.

The uncompromising objective of this book is to guide its behaviourally-trained reader to formulate complex clinical cases. After a basic introduction to behavioural interviewing and formulation by Wolpe and Turkat, the format adopted is that of illustrative chapters. Each begins with a clinical case summary followed by discussion of the condition described in relation to DSM III or psychiatric practice, then from a psychometric or behavioural perspective. The case vignette is then re-examined and formulated in behavioural terms.

Malatesta's excellent chapter on Geriatric Organic Syndromes alone fulfils the aims of the book. For the rest, the chapters on Paranoid Personality Disorder, Antisocial Personality Disorder and Histrionic Personality serve better as demonstrations of the limitations of a strictly behavioural approach to complex problems. The absence of behavioural techniques to follow on from the often laboriously derived formulations is frankly admitted at the end of the chapter on Alcohol Abuse and implied by the absence of treatment outcome in the other cases discussed. One's mounting incredulity is compounded by the acknowledged dearth of a relevant research literature.

Only tutors of trainee clinical psychologists may be tempted to order this book, as an object lesson on the hazards of too narrow a conceptual base.

CHARLES LUND, *Consultant Psychotherapist, Royal Victoria Infirmary, Newcastle upon Tyne*

Tics and Related Disorders. By A. J. LEES. Edinburgh: Churchill Livingstone. 1985. Pp. 276. £30.00.

A comprehensive account of the idiopathic tic is followed by a description of the more exotic Gilles de la Tourette syndrome, the essential features of which are multiple body and vocal tics beginning in childhood or adolescence. Coprolalia is commonly associated. Haloperidol, and other neuroleptics such as pimozide, produce marked improvements in 75% of patients. Also of interest to psychiatrists is the uncommon but distressing Meige Syndrome characterised by blepharospasm, grimacing, and oromandibular torsion. When idiopathic, this is often associated with manic-depressive psychosis, but it can occur as a potentially irreversible complication of neuroleptic treatment. There is a useful chapter on the drug-induced dyskinesias, although the relationship of these to tics is not very clear. This readable and well-presented book should find a place in most psychiatric libraries.

D. J. S. FERNANDO, *Clinical Assistant in Psychiatry, Glenside Hospital, Bristol*

Misuse of Drugs. By PATRICK BUCKNELL and HAMID GHODSE. London: Waterlow Publishers. 1986. Pp. 410. £35.00.

This book is the second in a series of volumes, each of which deals with a specialised aspect of the criminal law.

The authors are a practising barrister and a psychiatrist; their primary readership is the legal profession, rather than psychiatrists or other health care professionals. In keeping with this aim, the bulk of the book is devoted to legal matters and the law. The sections on the nature of drug addiction, drugs of dependence and the treatment of drug dependence are comparatively brief and deal with issues at a level appropriate for ensuring that lawyers are informed about this topic, but which is at the same time insufficiently detailed for persons directly concerned with the study and management of drug dependence.

If psychiatrists buy this book, therefore, it will be because of its legal content. For anyone who requires an authoritative exposition of the law relating to drug misuse, together with many of the relevant statutes themselves (reprinted as appendices, which constitute more than half of the book), this is an essential book.

There is a useful directory of national and regional organisations and clinics concerned with drug dependence.

PAUL WILLIAMS, *Senior Lecturer, Institute of Psychiatry, London*

An Introduction to the Psychotherapies (2nd edition). Edited by SIDNEY BLOCH. Oxford: Oxford University Press. 1986. Pp. 285. £8.95 (pb), £15.00 (hb).

The first edition of this introduction had a releasing effect on students who had previously found themselves dazed by the subject's complexity. It distinguished in simple and unambiguous language the essential differences of major forms of psychotherapy, and by its unifying title established the term 'the psychotherapies' in general nomenclature. Two new chapters have been added to the second edition. The first, by Rosen, on brief focal psychotherapy shows skilful handling of an important field and is a model of pithiness and clarity. Wolff, on client centred psychotherapy, surveys the field whilst focusing primarily on the individual treatment of children by dynamic and non-directive psychotherapies. Both chapters follow the format of earlier contributions, commenting on the goals, theory and techniques of therapy as well as on the training needed. Essential references are quoted together with recommended reading. This book is a "best buy".

BRIAN LAKE, *Consultant Psychotherapist, St James University Hospital, Leeds*

Neurotransmitter Actions on the Vertebrate Nervous System. Edited by MICHAEL A. ROGAWSKI and JEFFERY L. BARKER. New York: Plenum. 1985. Pp. 511. $69.50.

Neurotransmission is at the heart of many disciplines and is particularly relevant for neurochemists and psychopharmacologists. This book is a valuable source of information for all aspects of neurotransmission in vertebrates, and includes sections on amino acids, acetylcholine, biogenic amines, neuropeptides, adenosine and ATP. Although the multi-authored text leads to inevitable repetition, the chapters by the editors on GABA and noradrenaline are models of clarity and well worth reading by research workers in biological psychiatry.

PETER TYRER, *Consultant Psychiatrist, Mapperley Hospital, Nottingham*

Human Relationships: An Introduction to Social Psychology. By STEVE DUCK. London: Sage Publications. 1986. £5.95 (pb), £17.50 (hb).

Many mental health professionals experience difficulty in understanding adequately the relevance of the 'frontier' discipline of social psychology to their day-to-day practice. This short book is intended primarily for undergraduates. Duck's central concern is with relationships, which 'run through the fabric of our lives ... but are regarded as too important or too obvious to write about ...' His chosen topics include Social Emotions, Communication Skills, Long-Term Relationships, and aspects of relationships with families, friends, acquaintances and strangers. The chapter on socio-psychological aspects of health is the most valuable and there is an interesting survey on stressors in relation to psychosomatic disorders. The author's style is easy and refreshingly humorous. This book should be useful for those coming to psychiatry with little knowledge of psychology; they will find that this is psychology with a 'human face'.

HERSCHEL PRINS, *Former Director, School of Social Work, University of Leicester*

My Self, My Many Selves. By J. W. T. REDFEARN. Orlando: Academic Press. 1986. Pp. 142. $40.00 (hb), £24.00 (pb).

This book, by one of the most experienced British Jungian Analysts, is an account of the notion of self in Jungian psychology. This is a highly complex and subtle concept, difficult to grasp especially in the precise language of academia. This Redfearn has happily eschewed. In its place he has adumbrated the self, or more aptly the selves, experientially, almost poetically, drawing on his own experiences and feelings and those of his analysands. I must say that I found this an illuminating and sensitive description, which anyone concerned with psychotherapy should find valuable to read.

Redfearn's thesis is that "... we are not one person but many, although some people are more one person than others. Our feeling of "I", as well as our behaviour, is being competed for and dominated by our various selves, one of which is in charge at one time, another at another." What is good about this book is that although distinctively and authoratively Jungian, Redfearn is not dogmatic. He admits that these phenomena, of archetype and complex, can be encapsulated in orthodox Freudian and object-relation theory. It is this concentration of the phenomena of selves and how they may be best understood that makes the book of interest to a wider readership than Jungians.

This beautifully produced volume can be warmly recommended.

PAUL KLINE, *Professor of Psychometrics, Department of Psychology, University of Exeter*

THE AMERICAN JOURNAL OF PSYCHIATRY

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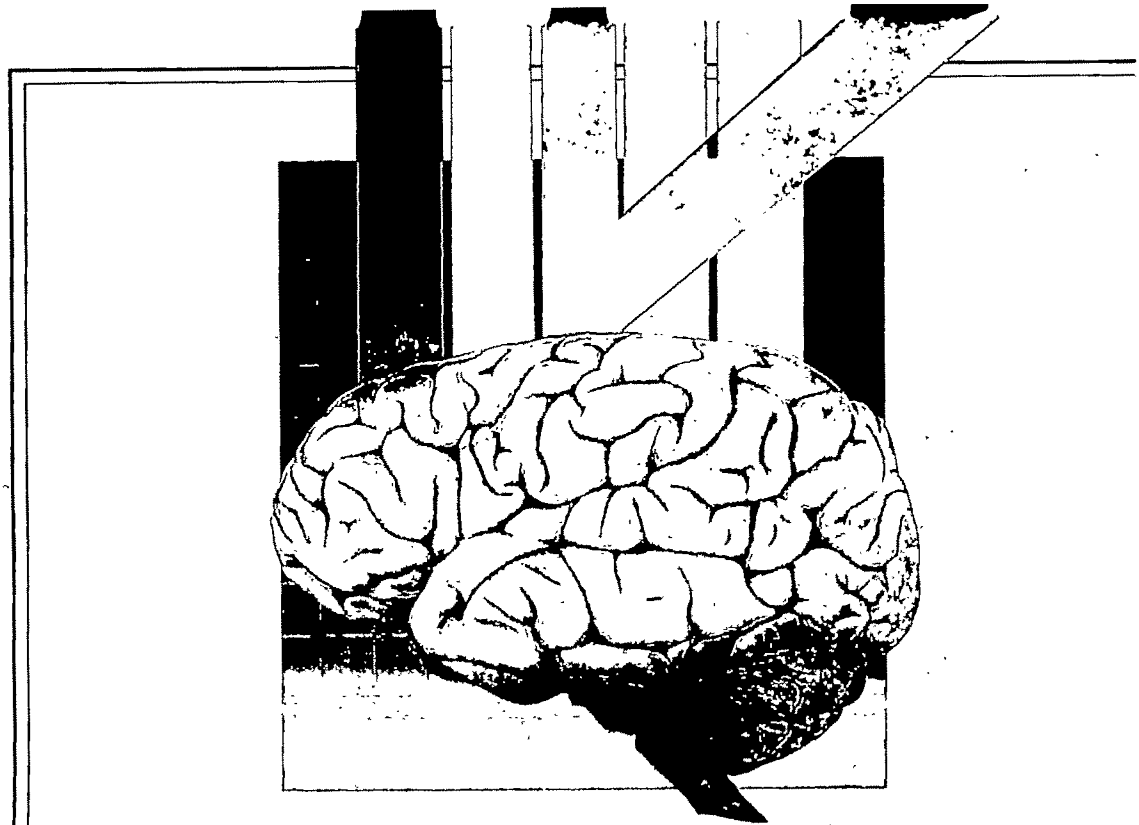

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Lecture

Assessment of Suicide Risk[1]

K. HAWTON

Among the many clinical skills that psychiatrists must acquire, the ability to assess the risk of patients killing themselves is probably the most important and demanding. It is often a crucial factor when making clinical decisions, such as in the choice of treatments, when deciding whether admission to a psychiatric hospital is necessary and when implementing the Mental Health Act. This review first explores the problems in assessing suicide risk. Subsequently, the risks of suicide for patients with major psychiatric disorders and for particular clinical populations, including hospitalised patients and suicide attempters, are examined together with the findings from research investigations which can assist psychiatrists when making decisions about the risk of suicide.

Problems in assessing suicide risk

The psychiatrist's task in assessing suicide risk is confounded by serious theoretical and practical difficulties.

Base-rate problem

The main difficulty is that of predicting what is fortunately a relatively rare event, especially as the available criteria are necessarily crude. This is illustrated by the fact that if one examines a high risk group, such as suicide attempters, and makes the assumption that the risk of suicide during the year after an attempt is of the order of 1% (see below), among 1000 suicide attempters there may be ten deaths by suicide during the following year. If our ability to predict those who will kill themselves is 80% efficient (this is as good a figure as is claimed by most workers), we should be able to identify correctly eight out of ten of the eventual suicides. However, in doing so a further 192 individuals are likely to be identified incorrectly as possible suicides. This rate of false-positive prediction is far too great if, for example, a decision were made to concentrate therapeutic efforts on only those most at risk.

Difficulties in applying general risk factors to individuals

While suicide risk factors may be useful in identifying high risk *groups* of individuals, such criteria are far less useful when it comes to predicting risk in the *individual* patient. To illustrate this in an extreme fashion, while both sex and age may be of some assistance in predicting suicide risk in a group of people, such criteria are of only limited value when considering an individual. Even when large numbers of criteria are employed, including some which are far better predictors than age and sex, this problem still pertains.

Means of identifying risk factors

Our knowledge of risk factors for suicide is based largely on the findings of studies that have examined the distinguishing characteristics of those people who, out of a group of individuals, kill themselves during a fixed time period, irrespective of what treatment may have been provided. However, some additional suicides are likely to have been prevented by the use of appropriate and effective treatment. Therefore, such studies actually identify factors which predict the risk of suicide *in spite of* treatment, and thus there must be some doubt about the extent to which the findings assist the psychiatrist in everyday clinical practice.

Short-term vs long-term risk

A major concern in clinical work is usually the assessment of whether or not a patient is likely to commit suicide during the next few hours, days or weeks, yet most of our knowledge concerning risk factors for suicide is based on examining the risk in the long term (and indeed this will be the main focus of this review). However, long-term risk factors are unlikely to be *identical* to those that predict short-term risk. Again, therefore, doubts exist concerning the extent to which findings from long-term follow-up studies can be used to assist the clinician in the assessment of short-term risk.

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Changeability of risk factors

A further problem with long-term risk factors is that we cannot predict what events or changes an individual will face in future which might alter his or her risk status. For example, a happily married man with a good job might, in a couple of years time, have become divorced, unemployed and have started drinking heavily. Thus he will have moved from being a member of a low risk group to one of much higher risk. Ideally, risk factors used for predictive purposes should be stable, but clearly most are not. Therefore, as Kreitman (1982) has noted, long-term prediction of suicide risk on the basis of current characteristics may be a somewhat illogical exercise.

Risk factors are not universal

It is important to acknowledge that the risk factors for suicide differ according to the group of individuals under consideration. For example, there are important differences between risk factors in psychiatric hospital in-patients and those identified for suicide attempters.

These problems should be borne in mind in assessing the value of research findings concerning suicide risk in various clinical disorders and populations. Before considering specific psychiatric disorders, there are a few general factors relevant to suicide risk, whether or not a person is suffering from mental illness. First, suicide is more common in men than women. Secondly, the risk increases with age until late middle or early old age, although recently there has been an increase in suicide rates among young men (McClure, 1984). Thirdly, most people who kill themselves have recently told, or intimated to others, what they were thinking of doing. Such communication may be straightforwardly direct, or it may be more subtle (Robins *et al*, 1959*a*).

Suicide risk and psychiatric disorder

The prevalence of various psychiatric disorders, based largely on retrospective diagnoses, among individuals in three major studies of suicides is summarised in Table I. In all three studies, the suicides were consecutive series identified through local coroners. The methods used in each study were similar, and included interviews with relatives and other informants, as well as scrutiny of official documents. However, since the studies included only cases in which an official verdict of suicide had been reached, and not probable suicides without an official suicide verdict, the findings may not be entirely representative of *all* people who kill themselves. For example, a verdict of suicide might be more likely in someone with a known psychiatric history, which might result in an overestimate of the prevalence of psychiatric disorder among suicides. Furthermore, the reports of informants could have been biased, with a tendency to over-report psychiatric symptoms in an attempt to explain or justify the act.

The disorders indicated in the table were the major or primary diagnoses—in many cases more than one diagnosis was made. Although amalgamating the findings from the three studies is scientifically unsound, particularly in view of the different diagnostic procedures that were employed, it provides an overall impression of the extent to which people who kill themselves are likely to suffer from various psychiatric conditions.

TABLE I
Psychiatric disorders among completed suicides (%)

	Affective disorders	*Alcoholism*	*Schizophrenia*	*Other psychiatric disorders*	*Total with psychiatric disorders*
Robins *et al* (1959*a*) ($n = 134$)	47	25	2	20	94
Dorpat & Ripley (1960) ($n = 108$)	30	27	12	31	100
Barraclough *et al* (1974) ($n = 100$)	70	15	3	5	93
($n = 342$)	48	23	6	19	96

The first conclusion to be drawn from these studies is that virtually all of the individuals who killed themselves were thought to have suffered from psychiatric disorders. Secondly, approximately half had affective disorders, almost a quarter alcoholism and approximately 5% schizophrenia. Factors that can assist in the assessment of suicide risk in these three disorders are discussed below, together with some comments on the risk of suicide in individuals with neuroses and those with major personality disorders.

Affective disorders

On the basis of 17 follow-up studies of patients with affective disorders, Guze & Robins (1970) calculated that approximately 15% ended their lives by suicide, which is 30 times the risk in the general population. They also noted that the risk of suicide is highest relatively early in the course of the disorder and that, unsurprisingly, suicides usually occur during episodes of depression rather than during remission. However, since depression is the most common condition for which patients are referred to psychiatrists, and a minority appear to be at risk of suicide, considerable efforts have been made to identify the characteristics of those patients most at risk.

Barraclough & Pallis (1975) compared 64 suicides diagnosed retrospectively as having depression with 128 living depressed individuals. The following characteristics were more common among the suicides: male sex, older age (in females), single status, living alone (six times more frequent than in controls) and having a history of suicide attempts (seven times more frequent). Symptoms which distinguished the suicides were insomnia, impaired memory and self-neglect. Furthermore, almost half the suicide showed retardation, and although this did not distinguish them significantly from the controls, this finding questions the dictum that retardation protects depressed patients against suicide. Depressed patients with delusions may be at greater risk than similar patients without delusions (Roose *et al*, 1983). A study by Roy (1983) of 21 depressed suicides and 21 depressed controls, matched for sex and age, confirmed that suicides are more likely to be single, living alone and have a history of previous suicidal behaviour.

Hopelessness, or negative expectations about the future, has long been emphasized as one of the most important factors leading to suicide in depressed individuals. Beck proposed that hopelessness might be the main symptom preceding suicide and could be of greater importance than depression itself (Beck *et al*, 1975*b*). He and his colleagues have recently examined the predictive value, in terms of suicide risk, of hopelessness, depression and suicidal ideation in 207 patients admitted to psychiatric hospital in-patient care with suicidal ideas, but not a recent suicide attempt (Beck *et al*, 1985). Measures of depression (Beck Depression Inventory [Beck *et al*, 1961]), hopelessness (Hopelessness Scale [Beck *et al*, 1974*b*]) and suicidal ideation (Scale for Suicidal Ideation [Beck *et al*, 1979]), all of which were completed during the 72 h following hospital admission, were compared between the 14 patients who ended their lives by suicide during the 5–10 years follow-up and those who were alive at the end of the follow-up period. While the suicides did not differ significantly from the survivors in terms of the original levels of depression and suicidal ideation, they did differ in terms of their scores on the measure of hopelessness, individuals who died by suicide having described themselves are more pessimistic about the future than those who had survived. Further support for this finding was provided by the fact that the pessimism item within the depression scale also correlated with risk of suicide. The results of this study have been replicated in a large group of psychiatric out-patients (Herold *et al*, in preparation).

Beck has elaborated on his view of hopelessness and depression in relation to suicide risk, saying that a sense of hopelessness in depression may arise out of activation of particular underlying cognitive patterns (probably established in response to early experiences), and that people differ in the extent to which they are prone to developing pessimistic attitudes towards the future, even when suffering from similar degrees of depression (Beck *et al*, 1985). Thus a high degree of hopelessness during one episode of depression may not just increase the current risk of suicide, but may also predict a high degree of hopelessness, and therefore greater risk of suicide, in a subsequent episode. This accords with clinical experience. Beck's work emphasises the care with which one should assess every depressed patient's attitude towards his or her future, as well as enquiring about severity of depression and specific suicidal ideas and plans. Furthermore, in patients thought to be of high risk, special care should be paid to the prevention and early detection and treatment of subsequent episodes of depression.

Alcoholism and other addictions

On the basis of several follow-up studies, Miles (1977) estimated that approximately 15% of alcoholics eventually commit suicide. In contrast to the affective disorders, alcoholic suicides tend to occur relatively late after the development of the disorder (Robins *et al*, 1959*b*).

The majority of alcoholics who kill themselves are also depressed (Pitts & Winokur, 1966; Barraclough *et al*, 1974). Motto (1980) identified three further factors which distinguished alcoholic suicides from surviving alcoholics—poor physical health, poor work record in the previous 4 years and a previous history of suicidal behaviour, especially if this had involved serious suicidal intent. Murphy and colleagues (Murphy & Robins, 1967; Murphy *et al*, 1979) demonstrated that the recent loss of a close relationship through separation or death was especially common among alcoholic as compared with depressed suicides. During the 6 weeks prior to suicide, such an event occurred in 32% of alcoholics compared with only 3% of depressives in their first study, and 26% compared with none in the second. Although there was no comparison group of surviving alcoholics to determine if the frequency of loss was especially high for people with this disorder, intuitively it appears to be so.

The risk of suicide among *drug addicts* is also known to be very high. James (1967) suggested that the risk of suicide among male heroin addicts is approximately 20 times that of the general population. Unfortunately there appears to be a paucity of empirical evidence which can assist in the detection of those drug addicts who are most at risk of suicide. However, the risk factors identified for other populations, especially alcoholics, are likely to be relevant.

Schizophrenia

Because such varying diagnostic criteria have been used for schizophrenia, at least until very recently, the risk of suicide among individuals with schizophrenia is uncertain. Miles (1977) reviewed a series of follow-up studies, most of which would have involved different diagnostic criteria, and estimated that 10% of schizophrenics died by suicide, the risk being particularly high during the early years of the illness.

On the basis of a comparison of 30 chronic schizophrenics who had killed themselves, with 30 surviving chronic schizophrenics, Roy (1982*a*) reported the following distinguishing characteristics of the suicides: male sex, relatively young age, chronic illness marked by relapses and remissions, depression noted at the last contact; expression of suicidal ideas, unemployment, and recent discharge from in-patient psychiatric care. Clearly, these characteristics apply to a large number of schizophrenic patients. A recent study has identified other and more subtle risk factors (Drake *et al*, 1984). One important finding was that all of a series of schizophrenic suicides occurred during a relatively non-psychotic phase of the illness. Furthermore, most of the suicides had attained a relatively high level of educational status prior to the illness, were usually living alone and had previously indicated suicidal intent. They tended to have high, non-delusional expectations of themselves, were largely aware of the effects of their illness and its consequences for their future functioning, and tended to be depressed, particularly with feelings of inadequacy, hopelessness and suicidal ideas, rather than biological features of depression. In keeping with Beck's postulate of a strong association between negative expectations about the future and suicide, three variables were especially important in predicting suicide: namely, fears of mental disintegration, suicide threats and hopelessness. Virtually all of the suicides and very few surviving controls had shown some or all of these symptoms. Akathisia (Drake & Ehrlich, 1985) and abrupt stopping of medication (Cohen *et al*, 1964) may also increase the risk of suicide. These findings emphasise the need for patients with schizophrenia to receive long-term support and also careful surveillance during remission as well as during relapse.

Neuroses

Sims & Prior (1978) demonstrated that patients with a variety of neurotic conditions had an increased risk of suicide. However, since their sample consisted of patients who had all been admitted to psychiatric in-patient care they would have had relatively severe disorders. The risk was especially high during the year after discharge. Many patients had a diagnosis of depressive neurosis. It is unclear what proportion of these might have now received a diagnosis of major depression. However, the suicide risk was high even when patients with depressive neurosis were omitted (Sims, 1984). Coryell has demonstrated that the risk is high in patients with panic disorder (Coryell *et al*, 1982) but not in those with obsessive-compulsive disorders (Coryell, 1981). Unfortunately, further information is lacking about the specific characteristics of patients with neuroses who are at particular risk.

Personality disorders

Little is known about the risk of suicide in people with abnormal personalities, except that they are relatively common among suicides (Barraclough *et al*, 1974), and that as many as 10% of suicides suffer from sociopathic disorders (Ovenstone & Kreitman, 1974). Individuals most at risk are probably those who show marked lability of mood, aggressiveness, impulsivity and those who have become alienated from their peers, especially if this picture is complicated by alcohol or drug abuse.

Other factors relevant to the assessment of suicide

Physical illness

Physical disorders, especially those of a chronic nature, are common among suicides. For example, in Dorpat & Ripley's (1960) study, at least half of the suicides had relatively serious physical disorders and in many cases the illness appeared to have contributed to the suicide. Physical illness was especially common among elderly male suicides. Specific conditions that often occur in suicides include peptic ulcer, cardiovascular disease and malignancies (Sainsbury, 1955; Dorpat & Ripley, 1960). Epilepsy is another important risk factor. In a review of several follow-up studies, Barraclough (1981) estimated that there is a five-fold increased risk of suicide in this condition, and that the risk may be very much higher in people with temporal lobe epilepsy.

Bereavement

Death of a parent during childhood may increase the risk of suicide in adulthood (Dorpat *et al*, 1965), probably because of increased vulnerability to depression (Brown & Harris, 1978). Recent bereavement, particularly loss of a spouse (McMahon & Pugh, 1965) or parent, is also a risk factor. Bunch (1972) found that the risk was increased for at least 4 or 5 years following bereavement, and was particularly high during the first 2 years. Those most at risk had a history of psychiatric disorder and/or suicidal behaviour before the bereavement, and lack of family support following the loss.

Family history of suicide

A family history of suicide appears to increase the risk of suicide in other family members who develop psychiatric disorders (Roy, 1982a,b), although whether this is related to family predisposition to affective disorder and other conditions, to having a family model of suicidal behaviour, or both, is uncertain.

Unemployment

Although there is substantial evidence that the risk of suicide is increased among unemployed men, and to a lesser extent among unemployed women (Platt, 1984), it is unclear to what extent unemployment itself contributes to suicide risk, or whether the association is due to the effects of unemployment on mental health, or whether people with poor mental health are at increased risk of becoming unemployed (Smith, 1985). Thus, while Shepherd & Barraclough (1980) found a markedly increased proportion of unemployed men in a series of suicides compared with matched surviving controls, they argued that the association was most likely to be due to a third factor, namely psychiatric disorder, since psychiatric illness seemed to be responsible for both unemployment and suicide in most cases. However, in some individuals, unemployment may also have exacerbated a pre-existing psychiatric condition and thereby increased the risk of suicide. Nevertheless, unemployment is a risk factor for suicide among individuals with psychiatric disorders (Roy, 1982*a*,*b*), irrespective of the nature of this association.

Biological factors

Several recent studies have suggested that abnormally low cerebral serotonin (5-HT) levels may exist in suicides (Asberg *et al*, 1976; Traskman *et al*, 1981; Stanley & Mann, 1983; van Praag, 1983). Whether this relates directly to suicidal behaviour, aggressiveness or severity of depression (Stanley *et al*, 1982) is unclear. However, if in the future cerebral seretonin activity could be evaluated easily by a blood test, it raises the remote possibility of a biological marker of suicide risk being developed that could be used in conjunction with other risk factors.

Psychiatric hospital patients

In spite of all our therapeutic efforts, suicides are relatively common among patients in psychiatric hospitals (Hawton, 1978). The risk on acute units has been estimated at over 50 times that of the general population (Fernando & Storm, 1984), although the risk among *all* psychiatric hospital patients, taking account of the elderly and long-stay, is considerably lower (Copas & Robin, 1982).

Patients with the highest risk are young or middle-aged males with affective disorders or schizophrenia and young females with affective disorders or personality disorders (Pokorny, 1964; Copas & Robin, 1982), especially if there is a history of previous suicidal behaviour (Fernando & Storm, 1984). Risk appears to be increased during the early stages of recovery from illness, when staff vigilance may have lessened (Sletten *et al*, 1972; Gale *et al*, 1980). Morgan & Priest (1984) have suggested that alienation from others on the ward, including staff, often precedes suicide. Some hospital environments may be conducive to certain forms of suicide, (e.g. a high building [Salmons, 1984] or adjacent railway track [Langley & Bayatti, 1984]). The week following admission to hospital (Copas & Robin, 1982) and the month immediately following discharge are the periods of greatest risk (Pokorny, 1964; Roy, 1982*b*). The risk following discharge may be related to the clinical team being misled by spurious improvement in a patient's condition in hospital while stresses outside hospital remain unresolved (Morgan & Priest, 1984).

Suicide attempters

The risk of suicide during the year following attempted suicide is approximately 1% (Kessell & McCulloch, 1966; Buglass & Horton, 1974; Hawton & Fagg, in preparation), which is about 100 times that of the general population. The risk declines somewhat in subsequent years but continues to remain relatively high until at least 8 years after an attempt (Hawton & Fagg, in preparation).

A considerable amount of research has been devoted to identifying those at particularly high risk within this group. One of the first investigations was carried out by Tuckman & Youngman (1963, 1968). This was based on cases identified by the police rather than through hospital referral alone. They demonstrated that the closer an individual's profile, in terms of demographic and clinical characteristics, approaches that typical of completed suicides, the greater the risk of eventual suicide. They developed a list of risk factors on the basis of two major follow-up studies: (i) age 45 years or more; (ii) male sex; (iii) unemployed or retired; (iv) separated, divorced or widowed; (v) living alone; (vi) poor physical heath; (vii) medical treatment within previous 6 months; (viii) psychiatric disorder, including alcoholism; (ix) violent method used in attempt (e.g. hanging, shooting, jumping or drowning; (x) left a suicide note; and (xi) previous attempts. The more items scored positively on this list for any individual, the greater the risk of eventual suicide. For example, attempters with two to five factors had a suicide rate of 6.8 per 1000 while those with ten or more had a suicide rate of over 60 per 1000. Subsequent studies have confirmed most of these factors as important predictors of risk and have added further items. For example, in two investigations, attempters who subsequently killed themselves had far less often experienced the break-up of the relationship with a partner in the year before the attempt and a major row during the 48 h preceding the attempt than had survivors (Pallis *et al*, 1982; Hawton & Fagg, in preparation), probably reflecting the fact that crises concerning relationships often resolve whereas other more chronic difficulties (e.g. poor physical health) may have a poorer prognosis. Two further factors which merit special attention when assessing suicide risk in this population are the *dangerousness of attempts* and *suicidal intent*.

Dangerousness of attempts

There has been considerable debate about whether anything can be inferred about the risk of subsequent suicide from the medical dangerousness of an attempt. For example, Fox & Weissman (1975) found that, while the medical effects of overdoses were greater than the medical effects of self injuries among suicide attempters, the degree of suicidal intent (i.e. wish to die) was generally lower among self-poisoners than those who deliberately injured themselves. Similarly, in a study of a larger series of attempters in Edinburgh, Rosen (1970) found that medical seriousness of attempts did not correlate with psychiatric seriousness (i.e. suicidal intent). In contrast, Pallis & Barraclough (1977), using retrospective assessments of the potential seriousness of attempts made by people who subsequently killed themselves, showed that the danger of the method used in the attempts was a positive, if somewhat weak, predictor of eventual suicide. Nevertheless, the controversy surrounding this question means that dangerousness alone is not a very useful basis for estimating suicide risk. However, it can be useful if the attempter's knowledge of the danger involved in the attempt is taken into account (Beck *et al*, 1975*a*). Thus, while Beck *et al* found that the correlation between suicidal intent and the medical danger in their overall series of attempters was very low, the correlation was very high when they examined separately the attempters who were able to assess acurately the probable outcome of their attempts if treatment had not been received.

Suicidal intent

This refers to the degree of apparent intention to die that is associated with an attempt. Several scales have been developed for assessing suicidal intent, of which the Suicidal Intent Scale (Beck *et al*, 1974*a*) is the most well known, having proved itself very useful in both research and clinical work. It has two sections: the first includes questions concerning the circumstances of the act, and the second includes questions concerning the subject's report about his/her state of mind at the time of the act. Scores on this scale have been shown to correlate with: (i) scores on the Tuckman and Youngman Scale of suicide risk (Pallis & Sainsbury, 1976); (ii) the medical seriousness of attempts in those aware of the dangers involved (Beck *et al*, 1975*a*); (iii) some aspects of depression, especially hopelessness (Minkoff *et al*, 1973; Wetzel, 1976; Dyer & Kreitman, 1984), which, as discussed earlier, is a predictor of eventual suicide in depressed patients with suicidal ideas; and (iv) risk of eventual suicide (Pierce, 1981). Furthermore, patients who make repeated attempts of increasing severity of suicidal intent appear to be at particular risk of suicide (Pierce, 1984).

In the assessment of suicide risk following an attempt, therefore, it is worthwhile taking into account social, demographic and clinical factors like those highlighted by Tuckman & Youngman, suicidal intent as measured objectively and, but only in those with knowledge about the potential risks, the medical danger involved in the attempt. Pallis and colleagues have developed composite scales for assessing suicide risk which include both an assessment of suicidal intent and of sociodemographic and clinical factors, items in the scale being weighted and cut-off points provided for varying degrees of risk (Pallis *et al*, 1984). However, weighting probably limits the practical usefulness of these measures. In everyday clinical work, psychiatrists are probably best advised to take account of all the factors that have been shown to characterise those at risk of suicide, without necessarily completing specific scales, although the Suicidal Intent Scale is worth using routinely. In addition, patients should be asked about their attitude to the future and whether they think they are likely to repeat the act in the near future. Some patients will deny their true intentions; in such cases the psychiatrist should be alerted to this by other factors (e.g. the dangerousness of the current attempt, the apparent hopelessness of the person's situation). Other factors which should be considered include whether or not active support will be available after the patient leaves hospital, the extent to which the patient will engage in after-care, and the likelihood that such help will be beneficial. Suicide risk is, of course, only one aspect of assessment of suicide attempters. A complete assessment should also take account of all the other problems that this population commonly faces, including, for example, relationship difficulties, family problems, alcoholism and other psychiatric disorders.

This review has summarised some of the more important findings from the substantial body of information that is now available concerning factors which can assist clinicians when assessing suicide risk. Clearly, statistical findings from studies of groups of patients can only help clinicians to a limited extent when considering individual patients. In addition, the special characteristics of each patient and his or her circumstances, including the availability of a suitable method for suicide (e.g. a firearm), always require attention. Nevertheless, it is worthwhile for the clinician to have in mind the information that has been provided by research and to ask himself in each case how closely the characteristics of the individual resemble those of people known to be likely to kill themselves.

While the low base-rate of suicide in even the highest risk groups will always limit the discriminatory power of predictive factors, further research is necessary to extend and refine our knowledge of risk factors. Other questions concerning suicide risk which require attention include, for example, how do suicidal ideas fluctuate during the course of an illness, what therapeutic strategies have an immediate effect on suicidal ideation, and do cognitive therapies diminish the extent of future suicidal behaviour by modifying vulnerability to experiencing hopelessness?

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References

Asberg, M., Traskman, L. & Thoren, P. (1976) 5-HIAA in the cerebrospinal fluid: a biological suicide predictor? *Archives of General Psychiatry*, 33, 1193–1197.

Barraclough, B. (1981) Suicide & epilepsy. In *Epilepsy and Psychiatry* (eds E. H. Reynolds & M. R. Trimble). Edinburgh: Churchill Livingstone.

—— & Pallis, D. J. (1975) Depression followed by suicide: a comparison of depressed suicides with living depressives. *Psychological Medicine*, 5, 55–61.

——, Bunch, J., Nelson, B. & Sainsbury, P. (1974) A hundred cases of suicide: clinical aspects. *British Journal of Psychiatry*, 125, 355–373.

Beck, A. T., Ward, C. H. & Mendelson, M. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

——, Schuyler, D. & Herman, J. (1974*a*) Development of suicidal intent scales. In *The Prediction of Suicide* (eds A. T. Beck, H. L. P. Resruk & D. J. Lettieri). Maryland: Charles Press.

——, Weissman, A., Lester, D. & Trexler, L. (1974*b*) The measurement of pessimism: the Hopelessness Scale. *Journal of Consulting and Clinical Psychology*, 42, 861–865.

——, Beck, R. & Kovacs, M. (1975*a*) Classification of suicidal behaviours: I. Quantifying intent and medical lethality. *American Journal of Psychiatry*, 132, 285–287.

——, Kovacs, M. & Weissman, A. (1975*b*) Hopelessness and suicidal behaviour: an overview. *Journal of the American Medical Association*, 234, 1146–1149.

——, ——, & —— (1979) Assessment of suicidal intention: the scale for suicide ideation. *Journal of Consulting and Clinical Psychology*, 47, 343–352.

——, Steer, R. A. Kovacs, M. & Garrison, B. (1985) Hopelessness and eventual suicide: a 10 year prospective study of patients hospitalised with suicidal ideation. *American Journal of Psychiatry*, 145, 559–563.

Brown, G. W. & Harris, T. (1978) *Social Origins of Depression*. London: Tavistock.

Buglass, D. & Horton, J. (1974) A scale for predicting subsequent suicidal behaviour. *British Journal of Psychiatry*, **124**, 573–578.

Bunch, J. (1972) Recent bereavement in relation to suicide. *Journal of Psychosomatic Research*, **16**, 361–366.

Cohen, S., Leonard, C., Farberow, N. & Schneidman, E. S. (1964) Tranquillizers and suicide in the schizophrenic patient. *Archives of General Psychiatry*, **11**, 312–321.

Copas, J. B. & Robin, A. (1982) Suicide in psychiatric in-patients. *British Journal of Psychiatry*, **141**, 503–511.

Coryell, W. (1981) Obsessive-compulsive disorder and primary unipolar depression: comparisons of background, family history, course and mortality. *Journal of Nervous and Mental Disease*, **169**, 220–224.

——, Noyes, R. & Clancy, J. (1982) Excess mortality in panic disorder: a comparison with primary unipolar depression. *Archives of General Psychiatry*, **39**, 701–703.

Dorpat, T. L. & Ripley, H. S. (1960) A study of suicide in the Seattle area. *Comprehensive Psychiatry*, **1**, 349–359.

——, Jackson, J. K. & Ripley, H. S. (1965) Broken homes and attempted and completed suicide. *Archives of General Psychiatry*, **12**, 213–216.

Drake, R. E. & Ehrlich, J. (1985) Suicide attempts associated with akathisia. *American Journal of Psychiatry*, **142**, 499–501.

——, Gates, C., Cotton, P. G. & Whitaker, A. (1984) Suicide among schizophrenics: who is at risk? *Journal of Nervous and Mental Disease*, **172**, 613–617.

Dyer, J. A. T. & Kreitman, N. (1984) Hopelessness, depression and suicidal intent in parasuicide. *British Journal of Psychiatry*, **144**, 127–133.

Fernando, S. & Storm, V. (1984) Suicide among psychiatric patients of a district general hospital. *Psychological Medicine*, **14**, 661–672.

Fox, K. & Weissman, M. (1975) Suicide attempts and drugs: contradiction between method and intent. *Social Psychiatry*, **10**, 31–38.

Gale, S., Mesnikoff, A., Fine, J. & Talbott, J. A. (1980) A study of suicide in state mental hospitals in New York City. *Psychiatric Quarterly*, **52**, 201–213.

Guze, S. B. & Robins, E. (1970) Suicide among primary affective disorders. *British Journal of Psychiatry*, **117**, 437–438.

Hawton, K. (1978) Deliberate self-poisoning and self-injury in the psychiatric hospital. *British Journal of Medical Psychology*, **51**, 253–259.

Hawton, K. & Fagg, J. Suicide and other causes of death following attempted suicide (in preparation).

Herold, R. C., Beck, A. T., Berchick, R. J., Brown, G. & Steer, R. A. Hopelessness as a predictor of long-term suicide in psychiatric outpatients (in preparation).

James, I. P. (1967) Suicide and mortality amongst heroin addicts in Britain. *British Journal of Addiction*, **62**, 391–398.

Kessel, N. & McCulloch, W. (1966) Repeated acts of self-poisoning and self-injury. *Proceedings of the Royal Society of Medicine*, **59**, 89–92.

Kreitman, N. (1982) How useful is the prediction of suicide following parasuicide? In *New Trends in Suicide Prevention* (eds J. Wilmotte & J. Mendlewicz). Basel: Karger.

Langley, G. E. & Bayatti, N. N. (1984) Suicides in Exe Vale Hospital, 1972–1981. *British Journal of Psychiatry*, **145**, 463–467.

McClure, G. M. G. (1984) Trends in suicide rate for England and Wales 1975–80. *British Journal of Psychiatry*, **144**, 119–126.

McMahon, B. & Pugh, T. F. (1965) Suicide in the widowed. *American Journal of Epidemiology*, **81**, 23–31.

Miles, C. P. (1977) Conditions predisposing to suicide: a review. *Journal of Nervous and Mental Disease*, **164**, 231–246.

Minkoff, K., Bergmann, E., Beck, A. T. & Beck, R. (1973) Hopelessness, depression and attempted suicide. *American Journal of Psychiatry*, **130**, 455–459.

Morgan, H. G. & Priest, P. (1984) Assessment of suicide risk in psychiatric in-patients. *British Journal of Psychiatry*, **145**, 467–469.

Motto, J. A. (1980) Suicide risk factors in alcohol abuse. *Suicide and Life Threatening Behavior*, **10**, 230–238.

Murphy, G. E. & Robins, E. (1967) Social factors in suicide. *Journal of the American Medical Association*, **199**, 303–308.

——, Armstrong, J. W., Hermele, S. L., Fischer, J. R. & Clendenin, W. W. (1979) Suicide and alcoholism: interpersonal loss confirmed as a predictor. *Archives of General Psychiatry*, **36**, 65–69.

Ovenstone, I. M. K. & Kreitman, N. (1974) Two syndromes of suicide. *British Journal of Psychiatry*, **124**, 336–345.

Pallis, D. J. & Sainsbury, P. (1976) The value of assessing intent in attempted suicides. *Psychological Medicine*, **6**, 487–492.

—— & Barraclough, B. M. (1977) Seriousness of suicide attempt and future risk of suicide: A comment on Card's paper. *Omega*, **8**, 141–149.

——, ——, Levey, A. B., Jenkins, J. S. & Sainsbury, P. (1982) Estimating suicide risk among attempted suicides: I. The development of new clinical scales. *British Journal of Psychiatry*, **141**, 37–44.

——, Gibbons, J. S. & Pierce, D. W. (1984) Estimating suicide risk among attempted suicides: II. Efficiency of predictive scales after the attempt. *British Journal of Psychiatry*, **144**, 139–148.

Pierce, D. W. (1981) The predictive validation of a suicidal intent scale: a five year follow-up. *British Journal of Psychiatry*, **139**, 391–396.

—— (1984) Suicidal intent and repeated self-harm. *Psychological Medicine*, **14**, 655–659.

Pitts, F. N. & Winokur, G. (1966) Affective disorder—VII: alcoholism and affective disorder. *Journal of Psychiatric Research*, **4**, 37–50.

Platt, S. (1984) Unemployment and suicidal behaviour: a review of the literature. *Social Science and Medicine*, **19**, 93–115.

Pokorny, A. D. (1964) Suicide rates in various psychiatric disorders. *Journal of Nervous and Mental Disease*, **139**, 499–506.

Robins, E., Gassner, S., Kayes, J., Wilkinson, R. H. & Murphy, G. E. (1959*a*) The communication of suicidal intent: a study of 134 consecutive cases of successful (completed) suicide. *American Journal of Psychiatry*, **115**, 724–733.

——, Murphy, G. E., Wilkinson, R. H., Gassner, S. & Kayes, J. (1959*b*) Some clinical considerations in the prevention of suicide based on a study of 134 successful suicides. *American Journal of Public Health*, **49**, 888–899.

Roose, S. P., Glassman, A. H., Walsh, B. T., Woodring, S. & Vital-Herne, J. (1983) Depression, delusions and suicide. *American Journal of Psychiatry*, **140**, 1159–1162.

Rosen, D. H. (1970) The serious suicidal attempt: epidemiological and follow-up study of 886 patients. *American Journal of Psychiatry*, **127**, 764–770.

Roy, A. (1982*a*) Suicide in chronic schizophrenia. *British Journal of Psychiatry*, **141**, 171–177.

—— (1982*b*) Risk factors for suicide in psychiatric patients. *Archives of General Psychiatry*, **39**, 1089–1095.

—— (1983) Suicide in depressives. *Comprehensive Psychiatry*, **24**, 487–491.

Sainsbury, P. (1955) *Suicide in London. Maudsley Monograph No. 1*. London: Chapman & Hall.

Salmons, P. H. (1984) Suicide in high buildings. *British Journal of Psychiatry*, **145**, 469–472.

Shepherd, D. M. & Barraclough, B. M. (1980) Work and suicide: an empirical investigation. *British Journal of Psychiatry*, **136**, 469–478.

Sims, A. (1984) Neurosis and mortality: investigating an association. *Journal of Psychosomatic Research*, **28**, 353–362.

—— & Prior, P. (1978) The pattern of mortality in severe neuroses. *British Journal of Psychiatry*, 133, 299–305.

Sletten, I. W., Brown, M. L., Evenson, R. C. & Altman, H. (1972) Suicide in mental hospital patients. *Diseases of the Nervous System*, 33, 328–334.

Smith, R. (1985) Occupationless health. 'I couldn't stand it any more': suicide and unemployment. *British Medical Journal*, 291, 1563–1566.

Stanley, M. & Mann, J. J. (1983) Increased serotonin-2 binding sites in frontal cortex of suicide victims. *Lancet*, ii, 214–216.

——, Virgilio, J. & Gershon, S. (1982) *Science*, 216, 1337–1338.

Traskman, L., Asberg, M., Bertilsson, L. & Sjostrand, L. (1981) Monoamine metabolites in CSF and suicidal behavior. *Archives of General Psychiatry*, 38, 631–663.

Tuckman, J. & Youngman, W. F. (1963) Identifying suicide risk groups among attempted suicides. *Public Health Reports*, 78, 763–766.

—— & —— (1968) A scale for assessing suicide risk of attempted suicides. *Journal of Clinical Psychology*, 24, 17–19.

van Praag, H. M. (1983) CSF 5-HIAA and suicide in non-depressed schizophrenics. *Lancet*, ii, 977–978.

Wetzel, R. D. (1976) Hopelessness, depression and suicide intent. *Archives of General Psychiatry*, 33, 1069–1073.

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Changes in Auditory P3 Event-related Potential in Schizophrenia and Depression

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Event-related potentials during a two-tone discrimination task were recorded in 24 schizophrenic patients, 16 depressed patients and 59 control subjects. Recordings were made when patients were medication-free. Fourteen schizophrenic and 13 depressed patients were retested at 1 and 4 weeks after the start of treatment, and 13 schizophrenic patients were also tested between 6 and 24 months after the initial recordings. In the schizophrenic group, the P3 latency was significantly prolonged compared with that in the control and the depressed groups, and remained unchanged both after 4 weeks treatment with therapeutic doses of neuroleptic drugs and at long-term follow-up. In the depressed group, the P3 latency did not differ from that of controls. P3 amplitude by contrast was reduced in both the acutely depressed and schizophrenic groups and following treatment became normal in the depressed group but remained reduced in the schizophrenic group. It is suggested that a prolonged P3 latency and reduced P3 amplitude indicate an impairment of auditory information processing in some patients with schizophrenia which is independent of the presence of acute psychotic symptoms and is not influenced by neuroleptic medication.

Electrophysiological studies employing a variety of perceptual tasks have reported changes in auditory event-related potentials which distinguish schizophrenic subjects from controls. The P3 component is a positive deflection recorded from the vertex approximately 300 m after a stimulus. The amplitude of this wave reflects both the unexpectedness of the signal, and the subject's awareness of the relevance of the signal in carrying out a simple cognitive task. The latency of P3 is a measure of the stimulus evaluation time (Kutas *et al*, 1977; Squires *et al*, 1977; Ford *et al*, 1979). A consistently reported observation in schizophrenic subjects is a reduction in the amplitude of P3 with auditory stimuli (Roth & Cannon, 1972; Levit *et al*, 1973. Verleger & Cohen, 1978; Roth *et al*, 1980; Steinhauer & Zubin, 1982; Baribeau-Braun *et al*, 1983; Pfefferbaum *et al*, 1984) and visual stimuli (Pass *et al*, 1980; Brecher & Begleiter, 1983). These changes are unlikely to be secondary to peripheral or brainstem changes (Pfefferbaum *et al*, 1980). Earlier studies have reported no change in P3 latency in schizophrenia, but Pfefferbaum *et al* (1984) have recently reported that in a group of 20 schizophrenic patients the P3 latency was significantly longer than in a group of 115 control subjects.

Changes in the amplitude and latency of other components of auditory event-related potential have also been reported in schizophrenia. N1, a negative wave in the range 90–150 ms post-stimulus, and P2, a positive deflection at 160–220 ms post-stimulus, are generally regarded as reflecting changes in selective attention (Hillyard & Kutas, 1983). Roth & Cannon (1972) found a shorter P2 latency and reduced amplitude which correlated negatively with medication dose in a group of 21 schizophrenic patients. In a group of 20 schizophrenics, Baribeau-Braun *et al* (1983) found N1 amplitude to be larger in patients on small doses of medication compared with those on higher doses. In contrast, Levit *et al* (1973) and Steinhauer & Zubin (1982) found no changes in the amplitudes of N1 and P2 and no correlation between amplitudes and medication dosage. Finally, Pfefferbaum *et al* (1984) reported a reduced amplitude of N1 but not P2 and a shorter P2 latency in patients with schizophrenia.

Changes in amplitude and latency of auditory event-related potentials are not specific for schizophrenia. P3 amplitude is reduced in patients with depression (Levit *et al*, 1973; Roth *et al*, 1981; Pfefferbaum *et al*, 1984). Shagass *et al* (1977, 1978) measured event-related potential following auditory, visual and somatosensory stimuli in 102 medication-free psychotic patients and 28 controls. In the three sensory modalities, the amplitudes of late components of evoked responses were reduced in both schizophrenic and depressed psychotic patients and were normal in non-psychotic patients regardless of diagnosis. The conclusion was that changes in N1 and P2 components reflected an impairment of attention related to psychosis in general and was not peculiar to schizophrenic patients.

Several factors may be responsible for the differing reports of changes in event-related potentials in schizophrenic patients, and these include variation in patient selection, states of medication and differing stimulus paradigms. This present study was designed to measure changes in auditory event-related potentials in patients with acute schizophrenia or depressive psychosis, and to see whether these changes disappeared or persisted with clinical improvement and response to treatment. Changes due to attentional difficulties in acute psychosis could thus be separated from changes reflecting more permanent underlying abnormalities in schizophrenia or depression.

Method

The study included 24 patients admitted to the Royal Edinburgh Hospital who fulfilled research diagnostic criteria (Spitzer *et al*, 1978) for schizophrenia and 16 patients who fulfilled the criteria for major depressive disorder. All patients were interviewed using the Present State Examination (Wing *et al*, 1974) and clinical ratings including the Hamilton Depression Score (HDS) (Hamilton, 1960) and the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962) were completed on each day that auditory-evoked responses were recorded. A diagnosis was reached independently by at least two psychiatrists using information from case notes in addition to the diagnostic interview. The signed consent of the patient and their nearest relative for the study was obtained in all cases.

Patients had received no antidepressant drugs or oral major tranquillisers for at least 3 months before initial testing, and no depot neuroleptic drugs for at least 1 year, with the exception of two patients with schizophrenia who had each received intramuscular neuroleptic medication (flupenthixol 20 mg) 6 weeks previously and one patient 3 months before testing. Five schizophrenic and four depressed patients requiring sedation were treated with amylobarbitone sodium (60–200 mg) within 3 days and not less than 10 h before testing. In 14 of the 24 patients with schizophrenia, event-related potentials were recorded before treatment and at 1 and 4 weeks after treatment. These patients were treated as follows: chlorpromazine (6), chlorpromazine and flupenthixol (4), haloperidol (1), fluphenazine decanoate (1), haloperidol and flupenthixol (1), trifluoperazine hydrochloride (1).

Thirteen patients with schizophrenia consented to be retested between 6 and 24 months (mean 13 months) after their initial assessment. Eleven patients could not be contacted or withhheld consent. Nine of these 13 patients were amongst those who had been tested at 1 and 4 weeks after treatment. At long-term follow-up, the group were receiving medication as follows: flupenthixol (9), chlorpromazine (2), pimozide (1), no medication (1). Two had never left hospital, two were re-admitted with an exacerbation of symptoms, five were living at home but unemployed and four were in employment.

In 13 depressed patients, recordings were obtained before treatment and 1 month after the start of treatment which was as follows: amitriptyline (7), cognitive therapy without drugs (2), monoamine oxidase inhibitor and lithium (2), ECT and amitriptyline (1), ECT alone (1). There were 59 control subjects, who included hospital staff, students and volunteers recruited through a local general practice. The control subjects had no history of physical or psychiatric illness and had been free of all medication for at least 12 months before testing.

Event-related potential recording

Event-related potentials were recorded soon after admission and repeated at 7–10 days and at 4 weeks after the start of treatment. A Nicolet CA1000 clinical signal averaging computer and NIC1009 P-300 stimulator were used (Nicolet Biomedical Instruments, Madison, Wisconsin). Subjects performed a two-tone auditory discrimination task while reclining in a chair in a sound-attenuated room. Bipolar recordings were made by way of a silver/silver chloride disc electrode secured at the CZ position and an indifferent ear-clip electrode attached to the left ear. The earth electrode was attached to the right ear. The electrode impedence was less than 2 kΩ. Sounds were delivered binaurally through headphones and the entire testing procedure lasted about 30 min. Subjects were instructed to count silently infrequent high-pitch tones of 1500 Hz randomly presented in a series of low-pitch tones of 1000 Hz. The ratio of high- to low-pitch tones was 1:9. The stimulus rate was 1.1/s, intensity was 65 db binaurally, and tone duration was 20 ms with a rise/fall time of 9.9 ms. The EEG was amplified 10 000 times with a bandpass of 1–100 Hz (−3 dB attenuation points of 12 dB/octave roll-off). The responses to frequent and rare tones were averaged separately. The digitizer sample rate was 100 kHz. The sensitivity was 50 μV. An artefact reject circuit excluded trials in which the voltage exceeded 45 μV. A total of 500 trials were averaged with a sweep time of 750 ms and a 75-ms pre-stimulus baseline. Two separate recordings were carried out at each testing. The baseline was estimated from the averaged response to frequent tones over the 75 ms before stimulus.

Individual peaks, designated N1, P2, N2, P3 (Fig. 1), were identified by visual inspection of the pen-recorded trace by a consultant neurophysiologist (D.M.McI.) who was not informed of the subject's diagnosis. N1 was the maximum negativity in the region of 70–120 ms and P2 the maximum positive deflection between 140 and 230 ms in the average response to frequent stimuli. P3 was the positive wave between 265 and 500 ms in the averaged response to rare stimuli and N2 was the negative deflection immediately preceeding P3. Latencies were measured at the maximum of amplitude using a cursor on the Nicolet CA1000 visual display. In those cases where the P3 deflection did not form a single sharp peak, the latency was measured as the point

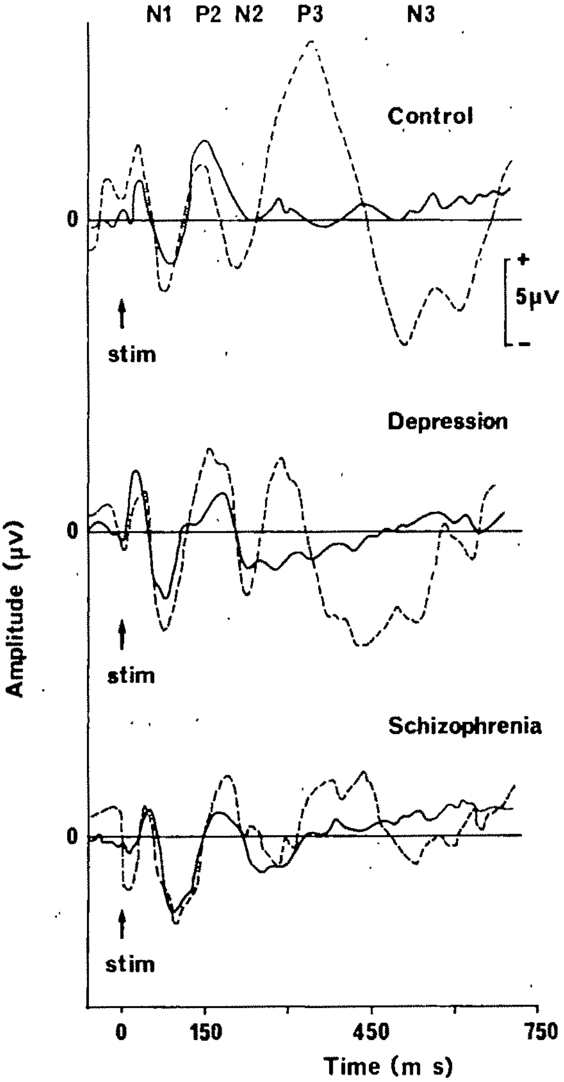

FIG. 1 Representative recordings of auditory event-related potentials obtained during a two-tone discrimination task from a control subject, a patient with major depressive disorder and a patient with schizophrenia. The continuous line is the response to frequent (1000 Hz) tones and the dotted line is the response to rare (1500 Hz) tones. In depression, P3 amplitude is reduced. In schizophrenia, P3 shows both a reduction in amplitude and an increase in latency.

of intersection of the tangents to the upgoing and downgoing slopes, measured on the pen-recorded trace. Amplitudes were measured from baseline to peak from the pen-recorded trace.

Results

Table I shows the age and sex distributions of all subjects and the clinical ratings of the patient groups. In each group, ages were normally distributed. In the control group, the latency and amplitude of the evoked potentials did not correlate significantly with age. Total BPRS scores were similar in both patient groups before treatment was started.

TABLE I
Age and sex distribution and clinical ratings of patients

	Male:female	*Mean age (range) (years)*	*BPRS mean ± s.e. (range)*	*Hamilton score mean ± s.e. (range)*
Control (59)	27:32	38 (19–59)		
Schizophrenic (24)	14:10	34 (19–57)	20.3 ± 1.5 (4–33)	11 ± 1.5 (1–30)
Depressed (16)	5:11	41 (19–60)	19.9 ± 2.2 (4–41)	24.3 ± 1.5 (17–37)

The latencies and amplitudes of the components of the evoked potentials were tabulated separately for the three subject groups. The results were then transposed on to the Edinburgh University mainframe computer and analysed using the *Statistical Package for the Social Sciences* (Nie *et al*, 1970). The data conformed to normal distribution and were analysed by one-way analysis of variance. The significance of differences between means was tested using the Scheffe procedure. However, in the analysis of P3 latency, the variances of the three groups were not homogeneous even after log transformation and differences between groups were tested separately using the Kruskall–Wallis test. Both the parametric and non-parametric analyses gave similar results.

Tables II and III show the mean latencies and amplitudes of N1, P2, N2 and P3 before treatment. The latencies of N2 and P3 were prolonged in the schizophrenic group and not in the depressed group compared with in control subjects. In 12 of the schizophrenic patients, P3 latency was greater than 2 s.d. from the mean of the controls. None of the depressed group came outside that range. The amplitudes of N1, P2, N2 and P3 were reduced in both depression and schizophrenia, but this reached significance only for P3 (ANOVA: $F = 11.95$, $P < 0.001$). Table IV shows the mean values for latency and amplitude of N1, P2, N2 and P3 in 14 patients with schizophrenia, in whom recordings were obtained before treatment and at 1 and 4 weeks after treatment began. The mean BPRS score was 20.9 ± 1.8 (range 16–33) before treatment, 21.0 ± 1.1 (range 16–27) at 1 week and 8.6 ± 1.7 (range 0–23) at 4 weeks. The change in BPRS over 4 weeks was significant ($P < 0.005$; paired t-test). One week after treatment, the latencies and amplitudes of the auditory responses were not altered significantly, but after 4 weeks, N1 amplitude was reduced and P2 amplitude was increased ($P < 0.01$; paired t-test). The latency and amplitude of N2 and P3 in schizophrenic patients did not change with treatment.

Table V shows the mean latencies and amplitudes of evoked responses before and during treatment in 13 depressed patients. The mean Hamilton Score was 24.7 (range 17–37) before treatment, and 6.0 (range 0–15) after

TABLE II
Latencies (means ± s.e.) (ms)

	N1	*P2*	*N2*	*P3*
Control (59)	91.5 ± 1.0	165.3 ± 2.1	213.8 ± 2.8	300.2 ± 2.9
Schizophrenic (24)	95.7 ± 2.1	161.9 ± 2.6	246.6 ± 6.8*	335.8 ± 7.8**
Depressed (16)	89.2 ± 2.5	159.8 ± 4.5	228.8 ± 6.1	301.4 ± 4.6

*N2: schizophrenic > depressed, $P<0.05$; schizophrenic > control, $P<0.001$.
**P3: schizophrenic > depressed, $P<0.001$; schizophrenic > control, $P<0.001$ (Kruskall-Wallis test).

TABLE III
Amplitudes (mean ± s.e.) (μV)

	N1	*P2*	*N2*	*P3*
Control	3.7 ± 0.22	4.3 ± 0.21	3.3 ± 0.28	8.9 ± 0.37
Schizophrenia	3.1 ± 0.39	3.5 ± 0.25	2.4 ± 0.37	5.5 ± 0.69**
Depression	3.1 ± 0.41	3.8 ± 0.53	2.7 ± 0.38	6.5 ± 0.83*

*Control > Depression, $P<0.01$.
**Control > schizophrenia, $P<0.001$ (Scheffe).

TABLE IV
Effect of antipsychotic medication on event-related potentials in 14 schizophrenic patients

	Latency (ms) (mean ± s.e.)				*Amplitude (μV) (mean ± s.e.)*			
	N1	*P2*	*N2*	*P3*	*N1*	*P2*	*N2*	*P3*
Before treatment	100.0 ± 2.7	150 ± 3.6	244 ± 9.0	343 ± 7.9	2.6 ± 0.39	2.9 ± 0.24	2.9 ± 0.53	5.2 ± 0.79
1 week after treatment started	98.6 ± 2.5	161 ± 4.1	261 ± 7.3	335 ± 9.0	2.9 ± 0.37	3.1 ± 0.28	3.2 ± 0.49	5.3 ± 0.75
4 weeks after treatment started	89.4 ± 5.8	160 ± 5.5	236 ± 9.1	339 ± 6.2	1.9 ± 0.31*	3.6 ± 0.27*	2.5 ± 0.39	5.4 ± 0.79

*N1 reduced, $P<0.01$ (paired Student t-test); P2 increased, $P<0.01$ (paired Student t-test).

TABLE V
Effect of treatment on event-related potentials in 13 depressed patients

	Latency (ms) (mean ± s.e.)				*Amplitude (μV) (mean ± s.e.)*			
	N1	*P2*	*N2*	*P3*	*N1*	*P2*	*N2*	*P3*
Before treatment	89.5 ± 2.5	162 ± 5.0	227 ± 5.4	297 ± 4.5	3.6 ± 0.41	3.8 ± 0.48	3.1 ± 0.75	6.7 ± 0.97
1 week after treatment started	90.5 ± 4.1	161 ± 4.1	224 ± 7.7	300 ± 6.5	3.1 ± 0.34	3.5 ± 0.5	3.2 ± 0.8	7.6 ± 0.82
4 weeks after treatment started	97.4 ± 3.5	162 ± 4.4	223 ± 7.1	301 ± 8.4	2.8 ± 0.44**	4.6 ± 0.41	4.1 ± 0.91	8.3 ± 0.72*

*P3 amplitude increased, $P<0.05$ (paired Student t-test).
**N1 amplitude reduced, $P<0.005$ (paired Student t-test).

4 weeks of treatment. After 4 weeks of treatment, there was reduction in N1 amplitude ($P<0.005$; paired t-test). P3 amplitude was also increased to 8.3 ± 0.72 μV ($P<0.05$; paired t-test) at which value it did not differ significantly from that in the control group (8.9 ± 0.37 μV).

Long-term follow-up of patients with schizophrenia

The mean P3 latency of this group of 13 patients at initial assessment, when medication-free and acutely ill, was 341 ± 36 ms and the mean P3 amplitude was 6.6 ± 3.7 μV. When retested an average of 13 months later (range 6–24 months), the P3 latency was 342 ± 32 ms and the amplitude was 7.8 ± 4.3 μV. The mean BPRS score was 19.5 (range 4–33) before treatment and 5.9 (range 0–14) at follow-up. Long-term neuroleptic treatment was thus not accompanied by significant changes in the P3 of patients with schizophrenia.

P3 wave form

In the schizophrenic group, the P3 waveform tended not only to be of lower amplitude but was broadened with a less clearly identifiable peak. The flattened waveform persisted following clinical recovery.

The effect of amylobarbitone sodium sedation

Five patients with schizophrenia and four patients with depression had received treatment with amylobarbitone sodium within 3 days but not less than 10 h before testing. The total dosage ranged from 60 to 600 mg. There was no significant difference in latency or amplitude of evoked response between the amylobarbitone-treated group and the completely drug-free patients.

Illness duration and P3 latency

The average number of years from onset of first psychotic illness in the schizophrenic group was 6.6 (range 3 months to ~~20 years~~). The correlation between P3 latency and illness duration was not significant ($r=0.32$; Spearman's Rank Correlation).

Clinical ratings and evoked potentials

For the 24 schizophrenic patients tested before treatment, the Pearson's product moment correlations were calculated for P3 latency and amplitude with the total score and individual items of the BPRS and the total Hamilton Score. P3 latency correlated positively with the total BPRS score ($r=0.5$, $P<0.02$) and, of the 19 individual items which constitute the BPRS, only one (the symptom 'emotional withdrawal and deficiency in relating to others') had a significant positive correlation with P3 latency ($r=0.48$, $P<0.03$). In contrast, P3 amplitude was found to be related to mood disturbance. In patients with schizophrenia, there was a significant negative correlation between P3 amplitude and total Hamilton Score ($r=-0.5$, $P<0.01$) and, of the 19 individual items of the BPRS, P3 amplitude correlated negatively with 'depressive mood' ($r=-0.47$, $P<0.03$) and motor retardation ($r=-0.44$, $P<0.05$). In contrast, in the group of 16 depressed patients, P3 latency and amplitude did not correlate significantly with the total Hamilton Score, total BPRS score or any of the individual items in the BPRS schedule.

Discussion

The main finding in this study is that, in a group of patients with schizophrenia, the latency of P3 was longer than that in either depressed patients or controls and is in keeping with the report of Pfefferbaum *et al* (1984). In 12 out of the 24 schizophrenic subjects tested, the latency of P3 was in excess of 2 s.d. from the mean for the control group. These latency differences remained unchanged after 4 weeks of treatment with neuroleptics and at long-term follow-up, at which times there was marked improvement in clinical symptoms. The significant positive correlation between P3 latency in schizophrenia not only with the total BPRS score but also on the individual rating of emotional withdrawal and deficiency in relating to others contrasted with the poor correlation between P3 latency and positive symptoms of acute psychoses, such as the presence of hallucinations, affective symptoms and paranoid ideation.

P3 latency in the schizophrenic group of patients was not related to age, the duration of illness nor the use of amylobarbitone sodium for initial sedation. The constancy of the P3 latency and amplitude changes following treatment and partial remission of psychiatric symptoms, suggests that these measures in schizophrenia reflect an underlying disorder of the processing of auditory information and are not merely the result of attentional deficits accompanying an acute psychiatric illness. As a group, the depressed patients had a mean BPRS score similar to the schizophrenic group, and in three of the depressed patients normal P3 latencies were recorded despite auditory hallucinations being reported during the period of testing.

In groups of schizophrenic patients, significant abnormalities have been detected in a variety of other psychophysiological measures, including skin conductance responsivity (Gruzelier & Venables, 1972), pursuit eye movements (Holzman *et al*, 1973) and the P50 wave of the auditory-evoked response using a conditioning-testing paradigm (Adler *et al*, 1982). A relative between abnormal eye-tracking movements and skin conductance habituation (Bartfai *et al*, 1983) and between eye-tracking dysfunction and P50 response (Siegel *et al*, 1984) has been reported in schizophrenic subjects. Furthermore, both the P50 response and eye-tracking movements are abnormal in a significant proportion of close relatives of schizophrenics. The P300 response shares several characteristics in common with the skin conductance response (Roth, 1983) and further systematic studies into the relationship between P3 and other psychophysiological abnormalities in patients and their relatives may clarify the role of physiological tests in the identification of a subgroup of schizophrenics, who appear to have, as a trait marker, a defect in the processing of and response to auditory and possibly other modes of input.

There was a tendency for the amplitudes of N1, P2, N2 and P3 to be reduced in both depressives and schizophrenics, although significance between group means was reached only for P3. When the effect of treatment in both groups was examined using paired samples, a significant reduction in the amplitude of N1 and an increase in P2 was measured. It is difficult to interpret changes in these wave forms, since the inter-stimulus interval is one of the major determinants of amplitude of N1 and P2, and the interval of 1.1 s used in this study is well below the optimum 8-s interval required for maximum evoked potential size, and under these conditions an amplitude change may be a reflection of an altered recovery curve rather than a true amplitude change (Lader, 1977). However, a possible explanation for the reciprocal changes in N1 and P2 is that treatment may be followed by an attentuation of the broad negative wave (Nd) which extends beyond N1 into the latency range of P2 and which Hillyard & Kutas (1983) have shown to be generated in selective attention tasks.

A reduction in P3 amplitude and an increase in P3 latency, although capable of distinguishing a group of patients with schizophrenia from patients with major depressive disorder and control volunteers, are changes which are not specific to schizophrenia. Similar alterations in P3 are found in Alzheimer's but not Korsakoff's dementia (Pfefferbaum *et al*, 1984; St Clair *et al* (1985) and in mental handicap of Down's syndrome and fragile X syndrome (Blackwood & St. Clair, in preparation). Although the sensitivity and specificity of auditory-evoked potential changes in schizophrenia await to be defined, these results suggest the presence of a stable abnormality in auditory information processing in a considerable proportion of patients with schizophrenia.

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References

Adler, L. E., Pachtman, E., Franks, R. D., Pecevich, M., Waldo, M. C. & Freedman, R. (1982) Neurophysiological evidence for a defect in neuronal mechanisms involved in sensory gating in schizophrenia. *Biological Psychiatry*, **17**, 639–654.

Baribeau-Braun, J., Picton, T. W. & Goselin, J. Y. (1983) Schizophrenia: a neurophysiological evaluation of abnormal information processing. *Science*, **219**, 874–876.

Bartfai, A., Levander, S. E. & Sedvall, G. (1983) Smooth pursuit eye movements, clinical symptoms, CSF metabolites, and skin conductance habituation in schizophrenic patients. *Biological Psychiatry*, **18**, 971–985.

Brecher, M. & Begleiter, H. (1983) Event-related brain potentials to high-incentive stimuli in unmedicated schizophrenic patients. *Biological Psychiatry*, **18**, 661–674.

Ford, J. M., Roth, W. T., Mohs, R. C., Hopkins, W. F. & Kopell, B. S. (1979) Event-related potentials recorded from young and old adults during a memory retrieval task. *Electroencephalography and Clinical Neurophysiology*, **47**, 450–459.

Gruzelier, J. H. & Venables, P. H. (1972) Skin conductance orienting activity in a heterogeneous sample of schizophrenics. *Journal of Nervous and Mental Disease*, **155**, 277–287.

Hamilton, M. (1960) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–296.

Hillyard, S. A. & Kutas, M. (1983) Electrophysiology of cognitive processing. *Annual Review of Psychology*, **34**, 33–61.

Holzman, P. S., Proctor, L. R. & Hughes, D. W. (1973) Eye-tracking patterns in schizophrenia. *Science*, **181**, 179–181.

Kutas, M., McCarthy, G. & Donchin, E. (1977) Augmenting mental chronometry: the P300 as a measure of stimulus evaluation time. *Science*, **197**, 792–795.

Lader, M. (1977) Effects of psychotropic drugs on auditory evoked potentials in man. In *Auditory Evoked Potentials in Man. Psychopharmacological Correlates of EPS* (ed. J. E. Desmedt). Basel: Karger.

Levit, R. A., Sutton, S. & Zubin, J. (1973) Evoked potential correlates of information processing in psychiatric patients. *Psychological Medicine*, **3**, 487–494.

Nie, N. H., Hull, C. H. & Jenkins, J. G. (1970) *Statistical Package for the Social Sciences*. New York: McGraw Hill.

Overall, J. E. & Gorham, D. R. (1962) The Brief Psychiatric Rating Scale. *Psychological Reports*, **10**, 799–812.

Pass, H. L., Klorman, R., Salzman, L. F., Klein, R. H. & Kaskey, G. B. (1980) The late positive component of the evoked response in acute schizophrenics during a test of sustained attention. *Biological Psychiatry*, **15**, 9–20.

Pfefferbaum, A., Horvath, T. B., Roth, W. T., Tinklenberg, J. R. & Kopell, B. S. (1980) Auditory brain stem and cortical evoked potentials in schizophrenia. *Biological Psychiatry*, **15**, 209–223.

——, Wenegrat, B. G., Ford, J. M., Roth, W. T. & Kopell, B. S. (1984) Clinical application of the P3 component of event-related potentials. II. dementia, depression and schizophrenia. *Electroencephalography and Clinical Neurophysiology*, **59**, 104–124.

Roth, W. T. (1983) A comparison of P300 and skin conductance response. In *Tutorials in ERP Research* (eds A. W. K. Gaillard & W. Ritter). Amsterdam: North Holland.

—— & Cannon, E. H. (1972) Some features of the auditory evoked response in schizophrenics. *Archives of General Psychiatry*, **27**, 466–471.

——, Pfefferbaum, A., Horvath, T. B., Berger, P. A. & Kopell, B. S. (1980) P3 reduction in auditory evoked potentials of schizophrenics.

——, ——, Kelly, A. F., Berger, P. A. & Kopell, B. S. (1981) Auditory event-related potentials in schizophrenia and depression. *Psychiatric Research*, **4**, 199–212.

SHAGASS, C., STRAUMANIS, J. J., ROEMER, R. A. & AMADEO, M. (1977) Evoked potentials of schizophrenics in several sensory modalities. *Biological Psychiatry*, **12**, 221–235.

SHAGASS, C., ROEMER, R. A., STRAUMANIS, J. J. & AMADEO, M. (1978) Evoked potential correlates of psychosis. *Biological Psychiatry*, 13, 163–184.

SIEGEL, C., WALDO, M., MIZNER, G., ADLER, L. E. & FREEDMAN, R. (1984) Deficits in sensory gating in schizophrenic patients and their relatives. *Archives of General Psychiatry*, **41**, 607–612.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, 35, 773–782.

SQUIRES, N. K., DONCHIN, E., SQUIRES, K. C. & GROSSBERG, S. (1977) Bisensory stimulation. Inferring decision related processes from the P300 component. *Journal of Experimental Psychology*, 3, 199–315.

ST CLAIR, D. M., BLACKWOOD, D. H. R. & CHRISTIE, J. E. (1985) P3 and other long latency auditory evoked potentials in Alzheimer's dementia and alcoholic Korsakoff syndrome. *British Journal of Psychiatry*, **147**, 702–706.

STEINHAUER, S. & ZUBIN, J. (1982) Vulnerability to schizophrenia: Information processing in the pupil and event-related potential. In *Biological Markers in Psychiatry and Neurology* (eds E. Usdin & I. Hanin). London: Pergamon Press.

VERLEGER, R. & COHEN, R. (1978) Effects of certainty, modality shift and guess outcome on evoked potentials and reaction times in chronic schizophrenics. *Psychological Medicine*, **8**, 81–93.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms.* London: Cambridge University Press.

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Nuclear Magnetic Resonance Brain Imaging in Chronic Schizophrenia

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Patients with chronic schizophrenia were examined by nuclear magnetic resonance imaging of the brain. Subgroups of the syndrome with high positive or high negative symptom scores and ventricular dilatation were compared with each other and with normal controls in respect of regional spin lattice relaxation time (T_1) changes. Significant differences were not observed between the schizophrenic subgroups and controls but there were significant differences between the subgroups themselves. The presence of tardive dyskinesia was associated with increased T_1 of the basal ganglia. The significance of these results is discussed in relation to findings using other techniques.

The development of newer imaging techniques has allowed the investigation of patients with schizophrenia in a manner hitherto difficult to achieve. Early reports by Johnstone *et al* (1976) demonstrated the presence of increased ventricular size and cognitive impairment in a group of chronic schizophrenics. Younger patients (Trimble & Kingsley, 1978; Benes *et al*, 1982) were shown to have normal ventricular dimensions. Weinberger *et al* (1979, 1980) did not find any correlation between ventricular size and length of illness, but found that those with atrophy had a poorer premorbid adjustment and a poorer response to neuroleptics.

In monozygotic twin studies, Reveley *et al* (1982) observed that the schizophrenic proband had enlarged ventricles compared with those in controls, suggesting that this is not a result of medication or chronicity and furthermore (Reveley *et al*, 1984) that it is a feature of those patients with a history of perinatal morbidity.

Golden *et al* (1980) showed a relationship between those with increased ventricular size, cognitive impairent and the presence of reduced X-ray density especially in the left anterior half of the brain.

Regional spin lattice relaxation time (T_1) changes obtained during nuclear magnetic resonance (NMR) imaging employing the spin-warp technique of Johnson *et al* (1981) have been demonstrated to alter in a number of pathological processes affecting the brain. These vary very widely and occur to a substantial degree in tumours, areas of demyelination and vascular lesions and, to a lesser degree, in Alzheimer's disease (Besson *et al*, 1985*a*) and idiopathic Parkinson's disease (Besson *et al*, 1985*b*). Thus, changes in T_1 may be used as an indicator of the site of pathological change. This study investigates patients with schizophrenic illness to determine:

(a) whether there are areas of the brains of schizophrenics which have altered T_1 compared with that of normals;
(b) whether there is a relationship between clinical profile, ventricular size and T_1 change;
(c) whether schizophrenic patients with tardive dyskinesia have changes in T_1 differing from those without.

Method

Twenty-three patients with a diagnosis of schizophrenia (DSM-III) were imaged. There were 20 males and three females, with a mean age of 36 years (Range 21–58 years) and a mean duration of illness of 12 years (range 1–36 years). All patients were receiving a dosage of neuroleptics considered optimal for their clinical state. The controls were 15 normal subjects, 12 males and three females, with a mean age of 36 years (range 21–54 years).

Imaging was carried out using the Aberdeen Mark II NMR imager (Hutchison *et al*, 1980), field strength 0.08 T, resonance frequency 3.4 MHz. Midline saggital and coronal sections were taken. From the sagittal section, regions of interest were mapped in the genu and splenium of the corpus callosum and in the thalamus; and from the coronal section, regions of central white matter of the temporal lobe and medial temporal structures corresponding to the inferior limb of the limbic system were mapped. Transverse sections commencing at the orbitomeatal level and moving cranially at 1-cm intervals were taken and regions of interest mapped in the basal ganglia, frontal and parietal white matter of both sides. The area, pixel count coordinates and mean ± s.d. T_1 values were obtained for each region of interest by computerised recall of the original acquired data.

Ventricular size was rated by visual examination of the images by an independent radiologist who was rating blind to the other aspects of the study. Ventricular size was rated on a four-point scale, with 0 indicating normal size and 3 the largest ventricles of the sample.

Subjects were rated for positive and negative symptoms by a semi-structured interview using a modified schedule of Krawiecka *et al* (1977). The presence of delusions, auditory hallucinations, incongruity of affect and altered mood were included in rating as positive symptoms, and blunted affect, psychomotor retardation, poverty of speech and incoherence or irrelevance of speech as negative. Each patient was examined for the presence of tardive dyskinesia using the items of the AIMS scale.

Results

Ventricular dilatation, clinical characteristics and regional T_1 values

Twelve patients had normal ventricular size and 11 had dilated ventricles. Of these 11, four were severe, one moderate and six were mild.

Comparing the normal and the dilated ventricular groups, there was no difference in the T_1 values in any of the regions of interest measured. With regard to the clinical features, there was no difference in the level of positive symptoms between the two groups, but the abnormal group had a slight increase in negative symptom score but this was not statistically significant. Those with dilated ventricles had a longer history of illness (mean 17 ± 4 years, compared with those with normal ventricles: 9 ± 2 years, NS) and were older (42 ± 4 years compared with 30 ± 3 years, $P = 0.02$). Those with dilated ventricules had slightly increased numbers with tardive dyskinesia, but this was not statistically significant.

Clinical subgroups based on cluster analysis of positive and negative symptom scores

Patients were rated on the basis of their positive and negative symptom scores on the Krawiecka scale and clustered into four groups on the basis of whether they scored high or low on the positive symptom and negative symptom dimensions. High positive patients had a mean score of 2.6 (range 0–4) and low positive patients had a mean score of 7.4 (range 5–10). Low negative patients had a mean score of 3.4 (range 2–4) and high negative patients had a score of 7.2 (range 5–10). Using this method four groups were obtained:

(1) low positive, high negative ($n = 6$);
(2) low positive, low negative ($n = 8$);
(3) high positive, low negative ($n = 4$);
(4) high positive, high negative ($n = 5$).

The T_1 characteristics of each region of interest measured were compared in each of the four groups. Group 1, characterised by a high negative score, had significantly increased T_1 (283 ± 5 ms) in the left frontal white matter compared with that in group 3 (265 ± 4 ms, $P < 0.05$) which is characterised by low negative scores. Significant differences were not found between the other groups, nor when each group was compared with controls (274 ± 4 ms).

Groups 3 and 4, characterised by high positive scores, had significantly increased T_1 in left medial temporal structures (368 ± 12 and 362 ± 11 ms respectively) compared with that in group 1 (348 ± 16 ms, $P < 0.05$) — characterised by low positive scores. Significant differences were not found between the other groups, nor when each group was compared with controls (360 ± 10 ms).

The ventricular sizes were examined in the four groups. In group 1 ($n = 6$), three patients had normal ventricles and three abnormal (one mildly dilated, one moderately dilated and one severely dilated). In groups 2 ($n = 8$), six were normal and two mildly abnormal. In group 3 ($n = 4$), three were normal and one mildly dilated, and in group 4 ($n = 5$), none were normal, two mildly dilated and three severely dilated. The most abnormal group (group 4) were characterised by high positive and high negative scores, and those with high negative scores alone (group 1), the next most abnormal, in terms of ventricular dilatation. Using the generalisation of Fisher's Exact Probability Test, the groups were different at the $P < 0.05$ level, the major contribution being from group 4.

Basal ganglia and tardive dyskinesia

Schizophrenics with tardive dyskinesia were compared with those without and with normal controls. The T_1 measures of all regions of interest, except for the basal ganglia, did not differ between these three groups. Differences were found in the putamen–globus pallidus of the basal ganglia (Fig. 1).

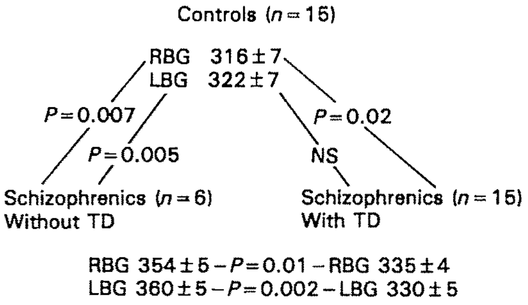

FIG. 1 Right and left basal ganglia T_1 (ms, mean ± s.e.).

Patients with tardive dyskinesia had significantly higher T_1 values than had normal controls. There were also significant differences between those with tardive dyskinesia and those without.

Tardive dyskinesia did not correlate with positive or negative symptom score, age or length of history. Three of the six patients with tardive dyskinesia had normal ventricles and three had mild or moderate ventricular dilatation. Patients with ventricular dilatation did not have statistically significantly higher tardive dyskinesia scores than those without ventricular dilatation.

Conclusion

The spin lattice relaxation time measured during NMR imaging is a sensitive indication of pathological processes. The precise nature of the pathological process is not disclosed accurately by these measures, but they are nevertheless useful in morphological siting of pathological lesions.

This study confirms previous reports that there is a subgroup of patients with schizophrenia who have enlarged ventricles, and these tend to have a high level of negative symptoms. Those with high negative and high positive scores tend to have the largest ventricles and are presumably the most severely ill.

High negative scorers have increased T_1 in the left frontal region in comparison with that of low scorers. This is of interest in view of the reputed reductions in cerebral blood flow and glucose utilisation in that region in positron emission tomographic (PET) studies (Buchsbaum *et al*, 1982) and the reduced X-ray density in computerised tomographic (CT) studies (Golden *et al*, 1980). However, these differences do not differ significantly from controls.

The negative symptoms in chronic schizophrenia bear a certain likeness to the frontal lobe syndrome seen in organic states. X-ray CT and NMR studies raise the possibility of morphological change in the frontal area. This is compatible with the reduced glucose utilisation and reduced cerebral blood flow in the frontal regions observed during PET studies by Buchsbaum *et al* (1982). Thus, the reduction in metabolic activity may be linked to structural change.

High positive symptom scorers have increased T_1 in the left medial temporal area relative to low positive scorers but not to controls. These schizophrenic subgroups do not differ from controls in respect to left medial temporal T_1 changes, but differ from each other.

All patients had received neuroleptic medication for prolonged periods of time and at varying dosage. Length of illness as a general rule relates to length of neuroleptic treatment. Tardive dyskinesia score does not correlate with length of illness. T_1 changes are found both in patients with and without tardive dyskinesia, although they were greater in the former. Therefore these changes may relate either to the presence of neuroleptic *per se* or to some change occurring as a result of the neuroleptic which may present to a lesser degree in patients without tardive dyskinesia and more pronounced in those with tardive dyskinesia and suggest an underlying pathological process associated with this disorder.

It is of interest that the neuroleptic-free patients of Buchsbaum *et al* (1982) showed reduced glucose metabolism in the central grey matter areas (mainly caudate) mainly on the left. There were no drug-free patients in this study, but any change in T_1 of the schizophrenic group without tardive dyskinesia only reached significance on the right. The striking differences occur in patients with tardive dyskinesia and suggest a location of pathology of this disorder in the basal ganglia. Such changes are also present in patients without tardive dyskinesia who are on medication. As yet, we do not know whether these changes are present in untreated schizophrenics, and whether therefore they are a feature of the disease which is present in greater magnitude in those with tardive dyskinesia from onset, or acquired later.

References

BENES, F., SUNDERLAND, P., JONES, B. D., LE MAY, M., COHEN, B. M. & LIPINSKI, J. F. (1982) Normal ventricles in young schizophrenics. *British Journal of Psychiatry*, **141**, 90–93.

BESSON, J. A. O., CORRIGAN, F. M., FOREMAN, E. I., EASTWOOD, L. M., SMITH, F. W. & ASHCROFT, G. W. (1985*a*) NMR imaging in dementia. *British Journal of Psychiatry*, **146**, 31–35.

——, MUTCH, W. J., DOWNIE, A. W., SMITH, F. W., CORRIGAN, F. M., ASHCROFT, G. W. & CROSHER, G. A. (1985*b*) Das Verhältnis zwischen M. Parkinson und Demenzzustanden untersucht mithilfe von Protonen-NMR-Abbildungsparametern. *Zeitschrift für Gerontologie*, **18**, 322–324.

BUCHSBAUM, M. S., INGVON, D. H., KESSLER, R., WATERS, R. N., CAPPELLETTI, J., VAN KAMMEN, D. P., KING, A. C., JOONSON, J. L., MANNING, R. G., FLYNN, R. W., MANN, L. S., BUNNEY, W. E. & SOKOLOFF, L. (1982) Cerebral glucography with positron tomography. *Archives of General Psychiatry*, **39**, 251–260.

GOLDEN, C. J., GRABER, B., COFFMAN, J., BAG, R. A., NEWLIN, D. B. & BLOCH, S. (1980) Structural brain deficits in schizophrenia. *Archives of General Psychiatry*, **38**, 1014–1017.

HUTCHISON, J. M. S., EDELSTEIN, S. A. & JOHNSON, G. (1980) A whole body NMR imaging machine. *Journal of Physics E: Scientific Instruments*, **13**, 947–955.

JOHNSON, G., HUTCHISON, J. M. S. & EASTWOOD, L. M. (1981) Instrumentation for NMR spin warp imaging. *Journal of Physics E: Scientific Instruments*, **15**, 74–79.

JOHNSTONE, E. C., CROW, T. J., FIRTH, C. D., HUSBAND, J. & KREEL, L. (1976) Cerebral ventricular size and cognitive impairment in schizophrenia. *Lancet*, **ii**, 924–926.

KRAWIECKA, M., GOLDBERG, D. & VAUGHN, M. (1977) A standardised psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

REVELEY, A. M., REVELEY, M. A., CLIFFORD, C. A. & MURRAY, R. M. (1982) Cerebral ventricular size in twins discordant for schizophrenia. *Lancet*, **i**, 540–541.

——, —— & MURRAY, R. M. (1984) Cerebral ventricular enlargement in non-genetic schizophrenia: a twin study. *British Journal of Psychiatry*, **144**, 89–93.

TRIMBLE, M. R. & KINGSLEY, D. (1978) Cerebral ventricular size in chronic schizophrenia. *Lancet*, **i**, 278–279.

WEINBERGER, D. R., TORREY, E. F., NEOPHYTES, A. N. & WYATT, B. J. (1979) Lateral cerebral enlargement in chronic schizophrenia. *Archives of General Psychiatry*, **36**, 735–739.

WEINBERGER, D. R., BIGELOW, L. B., KLEINMAN, J. E., KLEIN, S. T., ROSENBLATT, J. E. & WYATT, R. J. (1980) Cerebral ventricular enlargement in chronic schizophrenia. *Archives of General Psychiatry*, **37**, 11–13.

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Tiapride in the Long-term Management of Alcoholics of Anxious or Depressive Temperament

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Thirty-two chemically dependent alcoholics with significant levels of anxiety or depression were admitted to a double-blind randomised study in which the effect of the substituted benzamide tiapride was compared with that of placebo over a 6-month period. Twenty patients completed the study. Assessments included relevant biochemical and haematological tests, drinking levels and associated behaviour, expressed satisfaction with various areas of life, the Crown–Crisp Experiential Index of neurotic symptoms and questionnaires on self-esteem and alcohol dependence. The results indicated that in comparison with the placebo group, patients treated with tiapride drank less and had longer periods of abstinence. This was associated with improvements in laboratory tests, reduction in neurotic symptoms, gains in self-esteem and increased levels of expressed satisfaction with life situation. The drug was well tolerated and no deleterious effects were noted, suggesting its potential usefulness for this patient group.

This paper reports a study assessing the efficacy of tiapride as a treatment agent in chemically dependent alcoholics with significant symptoms of anxiety or depression.

Treatment of chemically dependent alcoholics is not universally successful and depressed or anxious alcoholics present special problems. The use of antidepressants carries the risk of facilitating suicide in a high-risk group, and the long-term use of minor tranquillisers not only increases the risk of suicide but also enhances dependency, both to alcohol and to the minor tranquilliser. Thus, those patients are frequently denied the benefit of drugs which might alleviate some of the symptoms likely to encourage further drinking on their part.

Tiapride (N-diethylaminoethyl 2-methoxy-5-methylsulphonyl benzamide) is a member of the substituted benzamide group of drugs which include metoclopramide (an antiemetic) and sulpiride (an antipsychotic). It is a selective dopaminergic receptor blocking agent (Costall & Naylor, 1977), but differs from typical neuroleptics in that it is not sedative (Jenner & Marsden, 1979) and does not cause Parkinsonism (Jenner *et al*, 1978). In common with typical neuroleptics, no anticonvulsant effect has been reported (Parent *et al*, 1978). It is not a drug of addiction (La Barre, 1978).

The drug has attracted favourable attention in the literature on the management of alcoholism. It has been found to have beneficial effects on tremor, agitation, gastrointestinal disturbance, appetite and craving for alcohol. Favourable results have been reported on the symptoms of anxiety (Marx, 1981; Bonnaffoux *et al*, 1982; Peyramond, 1982) and on symptoms of depression (Bonnaffoux *et al*, 1982; Stecchini & Corrias, 1982). In an earlier study from our own unit (Murphy *et al*, 1983), tiapride was found to have beneficial effects on symptoms of anxiety and depression. It was observed that the drug did not affect vigilance and there was a suggestion that it alleviated craving for alcohol. Freour (1979) reported facilitation of abstinence with the drug and Bonnaffoux *et al* (1982) reported an improvement in relationships within the work and family setting.

The non-addictive nature of the drug, its beneficial effects on symptoms of anxiety and depression and the possibility that it might lessen craving for alcohol suggested its usefulness in the long-term management of alcoholics of anxious or depressive temperament.

Treatment of alcoholism aims not only to promote abstinence but also to improve the alcoholic's ability to function in a social setting and to increase satisfaction, both with regard to relationships with significant others and with general life-style. Therefore, the study was designed to take account of those factors.

Method

The study was double-blind and placebo-controlled with random allocation to groups. Consecutive male admissions to the detoxification section of an alcohol treatment unit in 1983 and 1984 were admitted to the study, after routine detoxification procedures had been completed, if they fell in the age range 25–65 years, were assessed clinically chemically dependent on alcohol, gave current evidence of high levels of anxiety or depression, scored more than 30 on the Crown–Crisp Experiential Index (Crown & Crisp, 1979) towards the end of their period of detoxification and whose life history gave evidence of a tendency to react to adverse life situations with manifestations of anxiety or depression. Informed consent was obtained from all patients, who were then allocated randomly to an active group who received tiapride 100 mg *t.i.d.* for a period of 6 months or to a placebo group who received one identical dummy tablet *t.i.d.* for the same period.

At the time of admission to the study and at closure, a battery of laboratory investigations were carried out (haemoglobin, mean corpuscular volume, platelet count, total bilirubin, alkaline phosphatase, aspartate transaminase, γ-glutamyl transpeptidase and albumin).

At intake, each patient was interviewed by a sociologist and two schedules were completed. The first schedule collated demographic information about the patient and, taking as point of reference the six months prior to admission, data relating to employment, the use of medical, psychiatric or alcohol facilities, the use of social agencies and criminal involvement. The drinking history over the previous 6 months was examined in detail and data recorded in relation to the number of days drinking in the previous 6 months, the quantity drunk on drinking days, the complications and consequences of drinking and the social complications of drinking. The total years of heavy drinking and the number of years since excessive drinking had been regarded as a problem were also recorded.

The second schedule was completed by both the patient and the sociologist. Assessments were made of the quality of the relationships with spouses, relatives and friends, and of the degree of satisfaction with employment status, accommodation, use of leisure time and physical and mental health. At this time, the patient completed a questionnaire assessing self-esteem (Litman *et al*, 1983), a questionnaire assessing dependence on alcohol, the Severity of Alcohol Dependence Questionnaire (SADQ) (Stockwell *et al*, 1979) and the Crown–Crisp Experiental Index. Each patient was re-interviewed at monthly intervals throughout the trial, when compliance with treatment was assessed by estimating the number of tablets left and a further 1-month's supply of tablets was issued. Significant changes in social functioning were noted and details of drinking behaviour over the previous month were recorded.

At completion of the study, the schedules were completed — on this occasion with reference to the 6-month study period. The self-esteem index and the Crown–Crisp Experiential Index were also repeated.

Data collected at monthly follow-up interviews were not analysed separately but were used to improve the accuracy of the final rating schedules. The pre- and post-treatment scores of pathological investigations were analysed statistically using the Statistical Analysis System (SAS) and the data from the schedules using the SAS system backed up where appropriate by non-parametric Willcoxon tests.

Results

A total of 32 patients entered the study, of whom 13 received tiapride and 19 placebo. Seven of the latter failed to comply with follow-up; they were all of no fixed abode. Of the active group, two patients were lost as they removed from the area, 2 patients of no fixed abode did not comply with the protocol and one was withdrawn because of intercurrent physical illness (congestive cardiac failure) unrelated to trial treatment. Those who dropped out were similar to the others in age and in length of drinking history but appeared to be more disturbed. For instance, they were more neurotic, more dependent on alcohol and over the preceding 6 months had longer periods of unemployment, spent more time in hospital and had suffered more arrests. However, the main difference was that 75% of them were homeless, as opposed to 15% of those who completed.

A comparison of the active group (8 patients) with the placebo group (12 patients) found them to be well matched in respect of demographic data, drinking history, severity of dependence and on various biochemical parameters. None of the differences between the groups was statistically significant.

Conventional liver function tests and routine haematology were carried out before and after treatment in both the tiapride and placebo groups. Those patients treated with tiapride showed beneficial changes on all tests performed and in the case of mean corpuscular volume the change achieved statistical significance (94.75 ± 7.76 *vs* 87.50 ± 6.55 fl). In contrast, the patients treated with placebo showed no change in mean corpuscular volume (96.75 ± 6.63 *vs* 96.27 ± 6.32 fl), an increase in alkaline phosphatase (85.75 ± 28.56 *vs* 101.30 ± 102.62 IU/litre), and a fall in albumin levels (42.42 ± 7.22 *vs* 36.10 ± 3.21 g/litre), these adverse changes no doubt reflecting continued drinking.

Changes in various drinking parameters before and after treatment in the tiapride and placebo groups are listed in Table I. In the tiapride group there were significant improvements in the number of days abstinent, physical complications of drinking and average daily intake on a heavy drinking day. All other changes were in a beneficial direction. These improvements were not mirrored in the placebo group and in some instances changes have been adverse. It will be seen from a comparison of columns 1 and 3 that the groups were well matched prior to treatment and there was no significant difference between the groups on any variable.

Pre- and post-treatment scores obtained on the Crown–Crisp Experiential Index and Self-esteem Scale in the tiapride and placebo groups are listed in Table II. A comparison of pre-treatment scores in the tiapride and placebo group shows no significant differences between the groups. In the case of the tiapride group, there were significant beneficial changes in all measures, whilst no change was observed in the case of the placebo group.

Table III lists the percentage of patients expressing improvement in level of satisfaction with various social and health functions in both tiapride and placebo groups. It is clear that the patients in the active treatment group were markedly more satisfied with their level of functioning after

TABLE I

Indices of drinking before and after treatment in the tiapride and placebo groups

Items (mean ± s.d.: range)	Tiapride Pre-treatment (n = 8)	Tiapride Post-treatment (n = 8)	Placebo Pre-treatment (n = 12)	Placebo Post-treatment (n = 12)
Average daily intake of ethanol (units) on a heavy drinking day	44.38 ± 23.25 (16–86)	22.5 ± 16.69** (0–48)	41.92 ± 22.18 (14–92)	39.67 ± 29.27 (0–92)
Total days abstinent in preceding 6 months	36.13 ± 48.48 (0–126)	102.88 ± 70.34* (28–182)	40.33 ± 41.34 (0–133)	57.42 ± 66.99 (0–182)
Physical complications	3.38 ± 1.41 (1–5)	1.88 ± 1.64* (0–4)	4 ± 0.95 (3–6)	3.83 ± 1.99 (0–6)
Social complications	2.88 ± 5.06 (0–15)	1.25 ± 2.19 (0–6)	4.5 ± 7.05 (0–26)	8.08 ± 11.08 (0–32)
Use of medical and social facilities	28.13 ± 14.09 (1–46)	20.5 ± 14.83 (3–46)	18.5 ± 17.38 (3–66)	23.17 ± 17.34 (7–57)

*$P<0.02$; **$P<0.01$.

treatment as compared to the situation prior to treatment. In the case of the placebo group, improvement in satisfaction was, at best, modest. In a number of instances, the differences in improved satisfaction between groups achieved statistical significance.

Additional statistical comparisons were made of score changes before and after treatment across the tiapride and placebo groups on all variables, with the following results. In the case of pathological parameters, the fall in mean corpuscular volume was significantly greater in the tiapride group. In the case of drinking variables, the tiapride group fared better at a statistically significant level than the placebo group in respect of the average amount of alcohol consumed on a heavy drinking day, in respect of physical complications experienced and in the use made of medical and social facilities. In respect of the Crown–Crisp Experiential Index, the tiapride group fared better than the placebo group on all indices at highly statistically significant levels.

TABLE II

Assessment of neuroticism and of self-esteem before and after treatment in the tiapride and placebo groups

Items (Mean ± s.d.: range)	Tiapride Pre-treatment (n = 8)	Tiapride Post-treatment (n = 8)	Placebo Pre-treatment (n = 12)	Placebo Post-treatment (n = 12)
Crown-Crisp Experiential Index				
Neuroticism	51 ± 11.76 (34–66)	32.25 ± 14.15** (9–55)	51.83 ± 10.91 (34–70)	50.33 ± 18.38 (15–80)
Free-floating anxiety and phobic anxiety	15 ± 5.86 (8–26)	9 ± 4.63* (1–14)	16.33 ± 4.38 (11–23)	16.33 ± 6.47 (1–26)
Depression	10.5 ± 3.34 (7–15)	5.75 ± 3.92** (0–11)	9.75 ± 3.19 (2–14)	10 ± 3.74 (4–16)
Self-esteem scale	48.63 ± 8.57 (40–65)	40.88 ± 7.02** (30–54)	54.33 ± 7.95 (38–71)	51.42 ± 12.47 (34–73)

*$P<0.02$; **$P<0.01$.

TABLE III

Comparison of percentage of patients expressing improvement in level of satisfaction with various social and health functions following treatment in the tiapride and placebo groups

	Tiapride (n=8) %	*Placebo (n=12)* %	*Fisher's probability score*
Relationships			
Patient-rating	62.5	25	0.11
Interviewer-rating	75	16.6	0.01**
Accommodation			
Patient-rating	25	8.3	0.34
Interviewer-rating	25	8.3	0.34
Use of day			
Patient-rating	50	16.6	0.13
Interviewer-rating	50	16.6	0.13
Use of leisure			
Patient-rating	75	16.6	0.01**
Interviewer-rating	75	16.6	0.01**
Physical health			
Patient-rating	37.5	8.3	0.15
Interviewer-rating	50	8.3	0.05*
Mental health			
Patient-rating	87.5	25	0.009**
Interviewer-rating	87.5	25	0.009**

*$P<0.02$; **$P<0.01$.

Discussion

This study was designed to assess the efficacy of tiapride as an aid in the longer-term management of chemically dependent alcoholics of anxious or depressive temperament. The severity of their symptoms of anxiety and/or depression is made evident from their pre-treatment scores on the Crown–Crisp Experiential Index (Table II), which all fall in the range of high pathology. Subjects scoring in this range would normally be treated either with conventional antidepressants or with minor tranquillisers. However, there is a reluctance on the part of clinicians to use these drugs in the alcoholic population, particularly since alcoholics are known to be a high-risk group in respect of suicide. Antidepressants are commonly used as suicidal agents and have peculiar dangers in that respect. The use of minor tranquillisers also increases the risk of suicide and carries the risk of substituting dependence on drugs for dependence on alcohol. There is also evidence (Smith & Wesson, 1983) that the use of alcohol increases the likelihood of developing dependency to benzodiazepines and it is probable that the use of benzodiazepines increases the ease with which dependence on alcohol may develop. Thus, these patients are usually deprived of the benefit of drugs which might alleviate some of the symptoms likely to encourage further drinking on their part.

Tiapride is not a drug of addiction and is therefore unlikely to enhance the development of chemical dependence on alcohol. It is relatively safe even if taken in high dosage and there is no evidence of synergistic reactions if combined with alcohol. Thus, the danger of successful suicide with the drug is less pressing. Its known antianxiety and antidepressant effects, coupled with the facts that it does not affect vigilance and may alleviate craving for alcohol, suggest its usefulness in the management of this patient group.

That subjects in this group are difficult to treat is confirmed from a consideration of our placebo group. Both tiapride and placebo groups, in addition to medication, had the benefit of regular supportive follow-up interviews conducted by therapists specialising in the treatment of alcoholism. Despite this, the placebo group showed no significant improvement over a 6-month period in a battery of pathological tests, in ratings of neuroticism, depression and self-esteem or in various measures of drinking status. Furthermore, improvement in their level of satisfaction with various measures of social functioning was, at best, modest.

The results of this study indicate that the tiapride group fared considerably better than did the placebo group. A consideration of changes in a battery of pathological tests before and after treatment offered no evidence of any deleterious effect of the drug on any of the parameters measured. In the case of the mean corpuscular volume, there was indeed a significant post-treatment shift in a beneficial direction.

The tiapride-treated group showed highly significant improvements in ratings of neuroticism, anxiety and depression as assessed by the Crown–Crisp Experiential Index, improvements which were not seen in the placebo group. In both groups, there was an improvement in self-esteem over the trial period, but only in the case of the tiapride group was the change of statistical significance.

With regard to various measures of drinking status over the treatment period, the tiapride group showed a significant increase in total days abstinent over a 6-month period and showed significantly less in the way of physical complications over that period. Their use of medical and social facilities and the number of social complications which they suffered were also reduced, although these did not achieve statistical significance. There was a highly statistically significant reduction in the amount of ethanol taken on a heavy drinking day. In contrast, the placebo group showed only modest improvement in respect of total days abstinent over the trial period, and their use of medical and social facilities increased, as did the number of social complications which they suffered.

Some indication that these improvements in drinking levels and in neurotic symptoms were accompanied by improved satisfaction with their life situation emerges from a consideration of Table III. In all instances, the tiapride group expressed much improved satisfaction in these areas whilst, at best, the placebo group made only modest gains.

This study is open to the criticism that the numbers are small, that the research questionnaires used by the sociologist have not been validated but rely on rather obvious face validity and that the research design did not allow complete confidence that patients were fully compliant with medication. Nonetheless, the results seem clear-cut. The tiapride group drank less, had longer spells of abstinence, improved significantly on measures of neuroticism and expressed greater satisfaction with their social situation and with their physical and mental health.

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References

BONNAFFOUX, D., JEANPARIS, F., BOURGEAT, P. & LAPORTE, P. (1982) Follow-up treatment after alcohol withdrawal: results after two years in 200 cases. *Semaine des Hôpitaux de Paris*, **58**, 2079–2081.

COSTALL, D. & NAYLOR, R. J. (1977) Neuropharmacological indications of an antidyskinetic potential for tiapride. *Semaine des Hôpitaux de Paris*, **58**, 72–76.

CROWN, S. & CRISP, A. H. (1979) *Crown–Crisp Experiential Index*. Kent, UK: Hodder & Stoughton.

FREOUR, P. (1979) The fight against alcoholism and its consequences in general practice. *Semaine des Hôpitaux de Paris*, 55, 757–761.

JENNER, P. & MARSDEN, C. D. (1979) The substituted benzamides—a novel class of dopamine antagonists. *Life Sciences*, **25**, 479–486.

——, ELLIOTT, P. M. C., CLOW, A., REAVILL, C. & MARSDEN, C. D. (1978) A comparison of *in vitro* and *in vivo* dopamine receptor antagonism produced by substituted benzamide drugs. *Journal of Pharmacy and Pharmacology*, **30**, 46–48.

LA BARRE, J. (1978) Rapport au sujet des propriétés non toxicomanogenes du tiapride (1347). *Semaine des Hôpitaux de Paris*, 54, 461–462.

LITMAN, G., STAPLETON, J., OPPENHEIM, A. N. & PELEG, M. (1983) An instrument for measuring coping behaviours in hospitalized alcoholics: implications for relapse, prevention, treatment. *British Journal of Addiction*, **78**, 269–277.

MARX, A. (1981) Out-patient treatment of chronic alcoholism with tiapride. *Semaine des Hôpitaux de Paris*, **57**, 1347–1348.

MURPHY, D. J., SHAW, G. K. & CLARKE, I. (1983) Tiapride and chlormethiazole in alcohol withdrawal: a double-blind trial. *Alcohol & Alcoholism*, **18**, 227–237.

PARENT, M., JADOT, M. & TOUSSAINT, C. (1978) Tiapride and alcohol withdrawal symptoms. *Revue Médicale de Liège*, **33**, 672–677.

PEYRAMOND, A. (1982) Comparative cross-over clinical trial of tiaprid versus meprobamate in agitated states. *Semaine des Hôpitaux de Paris*, **54**, 542–545.

SMITH, D. E. & WESSON, D. R. (1983) Benzodiazepine dependency syndromes. *Journal of Psychoactive Drugs*, **15**, 85–95.

STECCHINI, M. & CORRIAS, G. (1982) Treatment of alcoholic patients with tiapride. *Semaine des Hôpitaux de Paris*, **58**, 2724–2726.

STOCKWELL, T., HODGSON, R., EDWARDS, G., TAYLOR, C. & RANKIN, H. (1979) The development of a questionnaire to measure severity of alcohol dependence. *British Journal of Addiction*, **74**, 79–87.

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Depression in the Elderly Living in the Community

Its Presentation and Features

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A prevalence of depression of 13% was found among 139 coloured persons aged 65 years and over, living in the community in Cape Town, which accords with figures elsewhere. A high rate of hypochondriasis was found, but there was a low rate of suicidal intent, which could be explained by good social support. Observed depression and a depressed mood occurred in almost all depressed patients; as defined in the Present State Examination, these items appear to be an excellent screen for depression.

A comprehensive survey of the psychiatric, physical and psychosocial status of non-institutionalised, coloured residents of Cape Town, aged 65 and over, was undertaken in 1982 (Ben-Arie *et al*, 1983; Elk *et al*, 1983; Nash & Meiring, 1983; Jacobs *et al*, 1984). They comprise a population group, living mainly in the Cape, who developed from the intermarriages of indigenous people and settlers (Gillis *et al*, 1968). This article examines the clinical features of depression in this group of elderly residents, and explores associated factors.

The clinical features of depression in the elderly have been summarised by Stenback (1980): depressed mood, somatic complaints and hypochondriasis are common, as are anxiety, irritability and loss of interest, but there is uncertainty about the prevalence of guilt and self-reproach. It has been argued that depression in old age is typified less by guilt than by somatic complaints, but Post is reported (Stenback, 1980) as having found symptoms of guilt and self-reproach in almost half his patients.

Epidemiological psychiatry has focused on the reliability and validity of psychiatric 'case' identification (Wing *et al*, 1981), and there is a particular problem in recognising depression in the elderly because it may present with features similar to dementia. To overcome such difficulties, research workers have turned to the use of instruments with proven validity and reliability for use in the elderly, such as the Comprehensive Assessment and Referral Evaluation Interview Schedule (CARE) (Gurland, 1980) and the Geriatric Mental State Examination (Henderson *et al*, 1983). The Present State Examination (PSE) (Wing *et al*, 1974; Wing & Sturt, 1978) is particularly useful because it is not age-specific and enables easier comparison with other age groups (Murphy, 1982, 1983).

Regardless of the instruments chosen, it is necessary to present data in a standard manner (Gurland *et al*, 1983; Henderson *et al*, 1983), while simultaneously respecting the uniqueness of the population under observation (Gutman, 1980).

Method

The survey as a whole

Selection of subjects

Names were obtained from geriatric registers kept by the City Health Department for the Eastern Zone of Cape Town. These are comprehensive registers of all persons of pensionable age (over 60 for females and 65 for males) compiled by community health nurses employed by the Department; they are continually updated by house-to-house visits. The Eastern Zone was chosen for sampling as it covers a representative range of income and social groups, as well as being the largest municipal zone.

A subsample selected at random was checked for representativeness by door-to-door enquiries, and consisted of evaluation of one street in each of ten areas chosen at random. A total of 20 consecutive houses per selected street were visited, and a list was drawn up of all people aged 65 and over. This was then compared with the relevant street registers, and there was a 75% agreement, without a particular pattern of non-correspondence in terms of age, sex, area and income variables. Using a table of random numbers, 150 respondents of 65 years and over were selected from the same subsample (Elk *et al*, 1983). The total number of elderly people on the Eastern Zone register was 9617, and the sampling rate was therefore greater than one in 64.

Procedure

Psychosocial data and subjective assessments of physical health were obtained through home visits by a team of six research workers (clinical psychologists and social workers) specially trained in the use of the instruments chosen for collection of the data. Within 2 weeks, respondents were examined physically by a team of doctors and assessed psychiatrically at community health centres by ten psychiatrists and clinical psychologists, specially trained for this purpose. There was one refusal for the study as a whole, and ten subjects did not have the psychiatric examination, one having died shortly after initial assessment and another, being deaf and blind, could not be assessed by the instruments used. The remainder refused to attend clinics or to allow domiciliary follow-up for a variety of personal reasons.

Assessment methods

The Present State Examination (PSE) (Wing *et al*, 1974) has been found to be reliable in many in-patient and population studies (Wing *et al*, 1977, 1978). It consists of 140 ratable items, from which syndromes can be generated by the CATEGO computer program. When the number, type and severity of symptoms cross the threshold of an index of definition (ID) (Wing & Sturt, 1978), a tentative diagnosis according to the eighth revision of the *International Classification of Diseases* is generated.

The Social Performance Index (Hurry & Sturt, 1980) is a measure of coping, and examines performance in seven well-defined areas (household, employment, money management, child care, intimate and non-intimate relationships, and social presentation). Respondents are rated only on items which apply to them, and ratings based on the person's report of the family's expectations, which has the disadvantage of losing direct comparability of scoring. A total impairment score is calculated for each respondent.

The Mini-Mental State Examination (MMSE) (Folstein *et al*, 1975) was developed for the assessment of intellectual impairment by a standard short rating scale and was mainly intended for use in elderly patients. However, in this investigation, it was adapted for use in a community survey. In view of the high illiteracy rate in the population studied, which is known to bias results (Anthony *et al*, 1982), these were interpreted conservatively, and an additional criterion of impaired social performance was required before a person could be rated as demented (Ben-Arie *et al*, 1983).

Selection of subjects for the current study

The current study examined differences in symptoms between depressed and 'well' respondents, i.e. those not given a diagnosis by the psychiatric ratings used. Respondents with a level of cognitive impairment likely to affect assessment ($n = 18$) were excluded using the MMSE and Social Performance Scale, as were those with diagnoses other than depression ($n = 10$). Diagnostic decisions were based on a combination of PSE findings and independent clinical assessment (Ben-Arie *et al*, 1983). Diagnoses other than depression or dementia included paranoid states ($n = 3$), schizophrenia ($n = 2$), mania ($n = 1$), and personality disorders ($n = 4$). No neurotic conditions (other than neurotic depression) were diagnosed, but 5% of the respondents (15% of males and 0.9% of females) were rated as being dependent on alcohol which, however, was not used as a criterion for exclusion. The depressed group, as based on the PSE/CATEGO tentative diagnosis, consisted of 23 respondents (16.5% of the respondents who were assessed psychiatrically). This group was re-analysed because it is possible to generate a tentative CATEGO diagnosis of depression on the basis of a single anxiety symptom (anxiety, panic attacks or anxiety on examination). Furthermore, the threshold ID of 5, which is sufficient for a tentative CATEGO diagnosis to be made, may tend to over-diagnose in population studies (Wing *et al*, 1981).

Therefore, the PSE protocols of the 23 respondents with a CATEGO tentative diagnosis of depression were examined further by two independent psychiatrists, experienced in the PSE but blind to the ID level. The raters agreed independently that the same four respondents were not clinically depressed; all had an ID of 5 (three scored on anxiety symptoms alone, and one had anxiety symptoms plus mild slowness of speech and observed depression). Therefore, these cases were excluded from further analysis, and their symptom profiles are shown in the Appendix.

The final samples for comparison thus consisted of 19 clinically depressed respondents (13.7%) and 88 with no psychiatric diagnosis (63.3%).

Results

Description of the group as a whole

The respondents were in general a 'young old' group (modal age category: 65–69 years) and mainly female (73%). Approximately one-third were married, half widowed and the remainder single or divorced. Over 70% had had less than 7 years of schooling, 21% never having been to school. Personal income was generally equal to the government old-age pension, and 79% came from families where the breadwinner(s) had been unskilled labourers. The vast majority of respondents were either retired (66%) or housewives (26%), with only 1% in full-time and 6% in part-time employment. A total of 96% were living with family and the remainder with friends—none were living alone. Nutrition was considered to be generally satisfactory, as judged from a haematological analysis and dietary history, and in the few cases where malnutrition was found, this was associated with alcoholism or major physical illness (Jacobs *et al*, 1984).

Findings with regard to depression

All statistical analyses of the data were done by the likelihood ratio chi-square, unless otherwise stated.

Demographic features

There were no significant associations between depression and demographic features such as age, sex, marital status, previous occupation and personal and pooled family income.

Psychiatric symptoms

Of the 19 depressed respondents, six had a 'threshold' ID of 5, the remainder scoring 6 or more.

For the purpose of comparison between the depressed and non-depressed groups, psychiatric symptoms were dichotomised on a present/absent basis. Table I shows a comparison in frequency of symptoms making up the syndromes leading to the CATEGO tentative diagnosis of depression.

Table II shows that PSE symptoms not leading to a tentative diagnosis of depression which significantly differentiated the two groups. Where there were differences in the frequency of symptoms, these were always more common in the depressed group.

TABLE I

Frequency of PSE symptoms used to reach CATEGO tentative diagnosis of depression

Syndrome	*Symptoms*	*% frequency in no diagnosis group*	*% frequency in depressed group*
Simple depression (SD)	19 Inefficient thinking	1.1	31.6**
	23 Depressed mood	5.7	94.7**
	24 Hopelessness	2.3	52.6**
	25 Suicidal plans or acts	0.0	5.3
	121 Depression on examination	1.1	78.9**
Special features of depression (ED)	29 Self-depreciation	0.0	11.1*
	32 Guilty ideas of reference	0.0	10.5*
	33 Guilt	0.0	10.5*
	51 Dulled perception	0.0	0.0
	54 Lost affect	0.0	5.3
Other symptoms of depression (OD)	27 Morning depression	2.3	57.9**
	34 Loss of appetite	8.0	36.8**
	37 Early waking	4.7	52.6**
	38 Loss of libido	2.3	5.3
	39 Premenstrual exacerbation	N/A	N/A
Depressive delusions and hallucinations (DD)	61 (2) Depressive hallucinations	0.0	10.5*
	88 Delusions of guilt	0.0	0.0
	91 Hypochondriacal delusions	0.0	0.0
	92 Delusions of catastrophe	0.0	5.3
General anxiety (GA)	11 Anxiety	2.3	26.3**
	14 Panic attacks	0.0	5.3
	120 Anxiety on examination	2.3	31.6**

*$P<0.05$; **$P<0.001$.

TABLE II

Frequency of PSE symptoms not used to reach CATEGO tentative diagnosis of depression: significant differences between depressed and no diagnosis group ($P<0.05$)

Syndrome	*Symptoms*	*% frequency in no diagnosed group*	*% frequency in depressed group*
Obsessional neurosis (ON)	46 Obsessional ideas and rumination	0.0	16.7
Slowness (SL)	110 Slowness and under-activity	0.0	21.1
Ideas of reference (IR)	31 Simple ideas of reference	2.3	27.8
Tension (TE)	5 Tension pains	17.6	52.6
	7 Muscular tension	2.3	33.3
	8 Restlessness	5.8	31.6
Lack of energy (LE)	36 Subjective anergia	10.3	47.4
	4 Worrying	8.2	78.9
	6 Tiredness	21.4	47.4
Worrying etc (WO)	10 Nervous tension	16.3	72.2
	21 Neglect through brooding	1.2	15.8
	35 Delayed sleep	15.1	42.1
Irritability (IT)	40 Irritability	11.5	73.7
Hypochondriasis (HY)	9 Hypochondriasis	2.3	26.3
Situational anxiety (SA)	15 Situational autonomic anxiety	3.4	16.7
Olfactory hallucinations (OH)	68 Olfactory hallucinations	0.0	10.5
	12 Anxious foreboding	1.1	21.1

Previous psychiatric treatment

There were no differences between the two groups on reports of previous psychiatric treatment.

Use of medication and physical complaints

A significantly greater percentage of depressed respondents were on 'minor tranquillisers' (15.8% as opposed to 1.1% $P<0.01$) and on vitamins (26.3% as opposed to 8%, $P<0.05$). No respondent in either group was on antidepressants, nor were there other differences in the use of medication. Significant differences were found in the area of subjective physical complaints; of the depressed group, 31% reported stiffness in the joints to the extent that it affected their daily activities as opposed to 7% in the psychologically well group ($P<0.01$). This finding was not significantly related to osteoarthritis on physical examination or to antirheumatic drug use. Of the depressed respondents, 58% ($n=11$) as opposed to 8% of the well respondents ($n=7$) felt at least moderately incapacitated by a variety of other physical complaints such as chronic cough, varicose veins, dizziness and hypertension ($P<0.001$). Visual difficulties of at least a moderately incapacitating nature were reported by 53% of the depressed, as against 27% of the non-diagnosed respondents ($P<0.05$). This finding was supported by the fact that the significantly more depressed had problems with visual acuity on examination (90 *vs* 65%, $P<0.05$).

No significant differences between the groups were found in reports of pain, dyspnoea, urinary and faecal incontinence, hearing difficulties, or other pathology detected on physical examination. Six respondents were found to have carcinomas; one of these fell into the depressed group.

Associated psychosocial features

Social performance. The depressed group was significantly more impaired in overall social performance than was the well group (22% mean disability rating *vs* 11%; Mann-Whitney $U=598.5$, $P<0.05$), experiencing difficulty of at least moderate intensity in the areas of household management (HM), child management (CM) and non-intimate relationships (NIR) (HM: 67 *vs* 27%, $P<0.01$, $n=103$; CM: 62 *vs* 10%, $P<0.001$, $n=54$; NIR: 42 *vs* 16%, $P<0.05$, $n=104$). Different totals for items arise because not all sections of the Social Performance Index were applicable to all respondents.

On multi-dimensional analysis, it emerged that over half of the depressed respondents (56%) had difficulty with household management, together with reported stiffness and other physical complaints. Only 7% of the control group reported difficulties in these areas. Assessments of household management performance took physical problems into account, since this is assessed by using the person's expected level of physical ability as a baseline.

Discussion

The prevalence of depression in elderly persons living in the community as shown in this study (13.7%) is similar to that of other investigations (Kay & Bergmann, 1980; Stenback, 1980). The putative protective effect of stable and supportive social groups in preventing depression does not seem to have lowered its rate in this population (World Health Organisation, 1983). The combined use of the PSE and independent clinical judgment probably makes the identification of depression as accurate as is possible for a community survey. In this study, the random sample revealed a higher proportion of females (73%) than of males. This is in keeping with, but higher than the proportion of females in this population in the Greater Cape Town area—61% in the 1980 census. Our sampling procedure was extremely rigorous and all possible precautions were taken to eliminate sampling errors. The difference may be due to the fact that only one particular region of Cape Town was studied, or it may be a chance finding. However, as we found no significant differences between males and females in the factors studied (and, in any case, sex ratios are probably not relevant to the purpose of the study, which is the phenomenology of depression), it would seem reasonable to regard the high proportion of females as probably not very noteworthy.

The absence of other forms of neurosis in this study bears out the contention that depression is "the neurotic disorder of old age" (Kay & Bergmann, 1980). None of the depressed respondents was receiving antidepressant medication, but significantly more were on tranquillisers and vitamins than were the controls. It would appear that their emotional distress had been recognised by clinicians, but not diagnosed as depression. This may be because anxiety and agitation (worry, nervous tension and irritability) were found in over 70% of depressed cases on the PSE. In addition, tension pain was present in significantly more of the depressed respondents, and this may also have impaired the recognition of depression by clinicians.

However, on direct enquiry, as the PSE requires, depressed mood was present in 94.6% and observed depression in almost 80% of the respondents given a PSE/clinical diagnosis of depression. Thus, if one asks about depression and carefully examines for it, a clear depressive affect may well be found; these PSE items in combination serve as an excellent screen for depression.

With particular reference to the use of the PSE and CATEGO program in community surveys of depression, it is worth noting that in all cases where the program gave a tentative diagnosis of depression which was not confirmed by clinical review, the Index of Definition was low (5). In most of these, a tentative diagnosis of depression had been made by the CATEGO program, taking account of anxiety symptoms alone, and this is seen as a defect in the program. It confirms Wing's contention (1983) that CATEGO tentative diagnoses must be reviewed by taking clinical information into account. Our findings make it clear that this is particularly so in depression with a borderline Index of Definition.

With regard to the presentation of depression, although hopelessness occurred as a symptom in just over half the depressed respondents, suicidal plans (which might be expected to be associated) were present in only one case (5.3%). The low rate of suicidal intent is particularly interesting in view of the high rate of alcoholism in the males of this cohort (15%) since these are commonly associated (Gillis, 1969); only one of our alcoholic subjects was rated as depressed, and he did not express suicidal intent. Suicidal tendencies are also said to be common in depressives with hypochondriacal symptoms (Post, 1965), but, although the latter was frequent (in 26%), it was not associated with suicidal intent. A possible explanation for the lower suicidal intent is that since most of our subjects were not isolated (the majority lived in close families and the rest had considerable social support), a known important risk factor was diminished (Berkman & Syme, 1979).

Loss of libido, unlike other vegetative symptoms, was present in only one respondent (5.3%). This low prevalence may be artefactual, because the majority of respondents, being widowed or single, did not live in situations where sexual desires were likely to be stimulated or opportunities offered (Newman & Nichols, 1960). Furthermore, young raters in a community survey of elderly respondents may not have enquired about sexual desires and fantasies in any depth.

Depressive delusions were not significantly present in the depressed group; this may be because when the condition was severe enough for delusions to develop, the person had probably been admitted to hospital. Depressive delusions are likely to be very rare in a community sample, occurring only in severe clinical depression (depressive delusions occur in about 28% of depressed inpatients [Eagles, 1983]). A similar explanation probably applies also to the low frequency of depressive symptoms, such as dulled perception and loss of affect.

In this group as a whole, depressed as well as non-depressed, there was a high rate of pathological symptoms, particularly those of 'neurosis'. Tension pains, subjective anergia, tiredness, nervous tension, delayed sleep and irritability were present in between 10 and 21% of the non-depressed group and were significantly more prevalent in the depressed. Amongst the latter, the most prevalent symptom ('worry') occurred in about 80%. This high rate of 'neurotic symptoms' has been discussed by Ben-Arie *et al* (1983), but accords with other investigations on the aged, as reviewed by Simon (1980). These symptoms are found commonly in epidemiological surveys, and particularly relevantly in a study of coloured persons as a whole (Gillis *et al*, 1968), but are rarely sufficiently severe to impair functioning or to lead to a rating of psychiatric illness (Bebbington *et al*, 1980).

To some extent, however, the high rate of symptoms may reflect the aggregation of a high level of physical ill-health in this population (Nash & Meiring, 1983), decreasing opportunities and mobility associated with age, and also possibly a degree of frustration as a result of living under adverse socioeconomic circumstances for many years.

In social performance, the depressed group showed significantly more impairment in coping with household tasks, management of children and relating to acquaintances, although there was no difference in their tidiness, dress and general hygiene. What impressed the raters was the generally good physical state of all the respondents, including the depressed, and the adequacy of their nutrition (Jacobs *et al*, 1984). This was felt to be due to the care and support given by families and friends.

This study indicates that the PSE and CATEGO program are effective in detecting depression in a community survey of the elderly, particularly where the Index of Definition is above 5 and clinical information is taken into account. It is also possible that depression is often missed by clinicians because of the more obvious presentation of symptoms such as anxiety or tension pain. Furthermore, clinicians may have diagnosed depression in some cases, but have considered minor tranquillisers to be a suitable form of medication. However, a specific enquiry will bring depression to light (if present), and this should be sought for when elderly persons complain of a variety of non-specific or unexplained physical symptoms, together with increasing difficulty in coping with usual life tasks.

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Appendix

PSE profiles of respondents receiving an ID of 5 and a CATEGO tentative diagnosis of depression but excluded from analysis of clinical grounds

Note: PSE item numbers and names are given. Where an item is followed by (2), the respondent scored 2 on this. In all other cases a score of 1 was obtained.

Respondent A: 1. Subjective evaluation of physical health; 10. Nervous tension; 35. Delayed sleep; 66. Visual hallucinations (clear consciousness); 120. Observed anxiety; 121. Observed depression; 131. Slowness of speech.

Respondent B: 4. Worry; 5 Tension pains; 6. Tiredness (2); 10. Nervous tension; 22. Recent loss of interest; 40. irritability (2); 120. observed anxiety.

Respondent C: 1. Subjective evaluation of physical health (2); 4. Worry; 6. Tiredness; 8. Restlessness and fidgeting; 9. Hypochondriasis; 10. Nervous tension; 11. Free-floating autonomic anxiety; 28. Social withdrawal; 35. Delayed sleep; 36. Subjective anergia and retardation; 40. Irritability (2).

Respondent D: 5. Tension pains; 6. Tiredness; 11. Free-floating autonomic anxiety; 12. Anxious foreboding with autonomic accompaniments; 17. Specific phobias; 18. Avoidance of autonomic anxiety; 111. Observed agitation; 120. Observed anxiety.

References

Anthony, J. C., Leresche, L., Niaz, U., Von Korff, M. R. & Folstein, M. F. (1982) Limits of the "mini-mental state" as a screening test for dementia and delirium among hospital patients. *Psychological Medicine*, 12, 397-408.

Bebbington, P., Hurry, J. & Tennant, J. C. (1980) Recent advances in the epidemiological study of minor psychiatric disorders (Editorial). *Journal of the Royal Society of Medicine*, 73, 315-317.

Ben-Arie, O., Swartz, L., Teggin, A. F. & Elk, R. (1983) The coloured elderly in Cape Town—a psychosocial, psychiatric and medical community survey. Part II. Prevalence of psychiatric disorders. *South African Medical Journal*, 64, 1056-1061.

Berkman, L. F. & Syme, S. L. (1979) Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology*, 109, 186-204.

Eagles, J. M. (1983) Delusional depressive in-patients, 1892 to 1982. *British Journal of Psychiatry*, 143, 558-563.

Elk, R., Swartz, L. & Gillis, L. S. (1983) The coloured elderly in Cape Town—a psychosocial, psychiatric and medical community survey. Part I. Introduction and psychosocial data. *South African Medical Journal*, 64, 1017-1022.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) "Mini-Mental State": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12, 189-198.

Gillis, L. S. (1969) The mortality rate and causes of death of treated chronic alcoholics. *South African Medical Journal*, 43, 230-232.

——, Lewis, J. B. & Slabbert, M. (1968) Psychiatric disorder amongst the coloured people of the Cape Peninsula. *British Journal of Psychiatry*, 114, 1575-1587.

Gurland, B. J. (1980) The assessment of the mental health status of older adults. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

——, Copeland, J., Kuriansky, J., Kelleher, M., Sharpe, L. & Dean, L. L. (1983) *The Mind and Mood of Aging*. London: Croom Helm.

Gutman, D. (1980) Observations on culture and mental health in later life. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Henderson, A. S., Duncan-Jones, P. & Finlay-Jones, R. A. (1983) The reliability of the Geriatric Mental State Examination. *Acta Psychiatrica Scandinavica*, 67, 281-289.

Hurry, J. & Sturt, E. (1980) Social performance in a population sample: relation to psychiatric symptoms. Paper presented at World Psychiatric Association Symposium—"What is a Case?", London.

Jacobs, P., Richards, J. D. M. & Ben-Arie, O. (1984) The coloured elderly in Cape Town—a psychosocial, psychiatric and medical community survey. Part IV. Haematological values. *South African Medical Journal*, 65, 16-18.

Kay, D. W. K. & Bergmann, K. (1980) Epidemiology of mental disorders among the aged in the community. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Murphy, E. (1982) Social origins of depression in old age. *British Journal of Psychiatry*, 141, 135-142.

—— (1983) The prognosis of depression in old age. *British Journal of Psychiatry*, 142, 111-119.

Nash, E. S. & Meiring, P. de V. (1983) The coloured elderly in Cape Town—a psychosocial, psychiatric and medical community survey. Part III. A survey of physical disorders and disabilities. *South African Medical Journal*, 64, 1089-1094.

Newman, G. & Nichols, C. R. (1960) Sexual activities and attitudes in older persons. *Journal of the American Medical Association*, 173, 33-35.

Post, F. (1965) *The Clinical Psychiatry of Late Life*. Oxford: Pergamon Press.

Simon, A. (1980) The neuroses, personality disorders, alcoholism, drug use and misuse, and crime in the aged. In *Handbook of Mental Health and Aging* (eds. J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Stenback, A. (1980) Depression and suicidal behavior in old age. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Wing, J. K. (1983) Use and misuse of the PSE. *British Journal of Psychiatry*, 143, 111-117.

—— & Sturt, E. (1978) The PSE-ID CATEGO system: Supplementary manual. (Mimeo) Institute of Psychiatry, London.

——, Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

——, Nixon, J. M., Mann, S. A. & Leff, J. P. (1977) Reliability of the PSE (Ninth Edition) used in a population study. *Psychological Medicine*, 7, 505-516.

——, Mann, S. A., Leff, J. P. & Nixon, J. M. (1978) The concept of a 'case' in psychiatric population surveys. *Psychological Medicine*, 8, 203-217.

——, Bebbington, P. & Robins, L. N. (Eds.) (1981) *What is a Case? The Problem of Definition in Psychiatric Community Surveys*. London: Grant McIntyre.

World Health Organisation (1983) *Depressive Disorders in Different Cultures*. Geneva: WHO.

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A Long-term Retrospective Follow-up Study of Patients Treated with Prophylactic Lithium Carbonate

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Patients suffering from unipolar and bipolar affective illness, who began treatment with prophylactic lithium carbonate during a 5-year period, were followed up and 59 out of 101 interviewed. Most had been taking lithium for at least 13 years: 49% had a complete remission, 41% a partial but significant response, and 10% no response. No specific individual or illness factor was found to correlate with favourable outcome, and no correlation between average serum lithium level and outcome. No side-effects could be associated specifically with the long-term use of lithium, but there was a surprisingly high incidence of clinical hypothyroidism.

This paper presents the results of a long-term retrospective follow-up study of a sample of patients given lithium carbonate as prophylaxis against recurrent affective disorders. Since the first classical papers of Baastrup & Schou (1967, 1968*a*,*b*; Schou, 1968) there have been a number of similar studies, using the so-called 'mirror-image' technique to assess the efficacy of prophylactic lithium. In addition, several controlled, random-assignment, double-blind studies have clearly established the efficacy of prophylactic lithium in both unipolar and bipolar patients (Coppen *et al*, 1963, 1971; Baastrup *et al*, 1970; Prien *et al*, 1971, 1973*a*,*b*, 1974; Cundall *et al*, 1972; Persson, 1972; Mendlewicz *et al*, 1973; Stallone *et al*, 1973; Dunner *et al*, 1982; Kane *et al*, 1982; Peselow *et al*, 1982).

The limitations of this type of retrospective study have been described (Blackwell & Shepherd, 1968; Blackwell, 1969), but this particular study is the first to follow a large cohort of patients over a period of 17 years, with detailed follow-up data being established first in 1971 and then 12 years later, in 1983. Thus, useful information has been obtained regarding long-term efficacy and side-effects, despite the fact that the data could not be subjected to a rigorous 'mirror-image' analysis.

Method

The sample

The subjects were a consecutive series of 101 patients, the first to be treated with prophylactic lithium by one consultant psychiatrist working in north London; all began lithium therapy between 1966 and 1971. Prior to that time, all had suffered from several episodes of depression and/or hypomania. The criteria for defining these episodes were that specific symptoms of depressive or hypomanic mood were elicited by the clinician, which were of at least 2 weeks duration and of sufficient severity as to impair the subject's usual functioning, though not necessarily requiring admission to hospital. There were distinct intervals between these episodes when the subject's mood state was normal for at least 4 weeks.

Assessments of the patients

Initial status

A detailed examination of their hospital and clinic case notes was carried out to identify characteristics of this group at the time of initial lithium prescription. This included:

(a) demographic details (age, sex, socioeconomic and marital status, employment record);
(b) family history of physical and psychiatric illness;
(c) initial diagnosis and detailed psychiatric history prior to commencing lithium treatment, including age of onset, length and pattern of illness, previous treatment, characteristics of illness including the presence or absence of specific symptoms, frequency of relapses, suicide attempts.

Follow-up status

In 1971, the entire cohort of patients who had been taking lithium for up to 5 years was reviewed (Benaim & Lappin, 1973). They were assigned to one of three outcome groups, depending on whether their response to lithium was complete, partial or insignificant. These outcome groups were defined as follows:

(a) complete response, i.e. there had been no episodes of hypomania or depression requiring treatment whilst the patient was taking lithium;
(b) partial response, i.e. some episodes of illness occurred whilst the patient was taking lithium, but these were less often and produced less handicap (in terms of need for additional medication, admission to hospital, duration of episode, time away from work) than those prior to lithium treatment;
(c) insignificant response, i.e. there was no detectable change in frequency or severity of relapses after lithium treatment was begun.

In 1983, attempts were made to trace these patients again via out-patient clinics, general practitioners, family practitioner committees and informal sources. All were asked to attend for interview, with the option of being interviewed in their home if unable to travel. The interview included collection of the following data: demographic details, psychiatric and physical morbidity since beginning lithium treatment, details of lithium therapy (average dose, length of time for which the drug was taken, individual patterns of taking treatment, compliance, reasons for discontinuation), employment history, marital history and social functioning since commencing treatment.

In order to assess their mental state at the time of interview, the ninth edition of the Present State Examination (PSE) was administered (Wing *et al*, 1974).

Analysis

The course of each individual's illness whilst on lithium was thus examined in terms of episodes of depression or hypomania, number of admissions, severity of the recorded episodes, frequency of episodes, suicide attempts and ability to function adequately at work and at home. A comparison was then made with the pre-lithium data for each individual and with the data obtained in 1971, the latter to assess the stability of the response. These comparisons were analysed using the chi-squared test.

Results

Of the 101 patients, 59 were interviewed in 1983: 20 were dead (detailed information regarding this group was sought from case notes, general practitioners and relatives), two were living abroad, four refused to be interviewed and 16 were never traced.

Characteristic of the initial sample

Sociodemographic details

The original group comprised 47 men and 54 women, their ages ranging from 19 to 75 years, the mean age at commencing lithium treatment being 45 years. A total of 74 were married, nine divorced, 15 single and three widowed. According to the Registrar General's classification, 22% were social class I, 49% social class II, 20% social class III non-manual, 7% social class III manual and 2% social class IV.

Diagnosis

At the time of starting lithium, 35 patients were classed by one of the authors (S. B.) as having unipolar affective disorder, 65 bipolar affective disorder and one a schizo-affective disorder.

Family history of psychiatric illness

A positive family history of bipolar illness was found in 61 subjects, and in all but one of these, their own diagnosis was also that of bipolar illness. There was a family history of unipolar illness in 46, which was evenly distributed amongst subjects with both diagnoses. There was a family history of completed suicide in 18, of which 14 were bipolar subjects. Nine had a family history of alcoholism; all of these were bipolar subjects.

Age of onset of illness

In 30, the age of onset of illness was less than 25 years, in 34 it was between 26 and 35 years, in 22 it was between 36 and 45 years, and in 15 it was over 45 years. There was no significant difference in age of onset of illness between those subjects with bipolar and those with unipolar disorders.

Characteristics of illness prior to lithium

The characteristics of illness before treatment with lithium included on average 8.5 depressive episodes (range 0–80), 3.8 admissions for depression (range 0–30), 2.7 hypomanic episodes (range 0–16), 2.1 admission for hypomania (range 0–9). The average frequency of episodes was 19 months.

The length of illness prior to lithium treatment varied from 1 month to 30 years, with a mean of 5 years. The frequency of occurrence of episodes was no different between unipolar and bipolar subjects.

Analysis of preliminary data

All the preliminary data were examined to see whether the original population could be defined by distinct subgroups, clustering in terms of sex, age of onset of illness, symptoms or family history. Using the chi-squared test to compare subgroups, it was not possible to demonstrate any significant clusters.

Follow-up data (1983)

Patients not available

No follow-up data were available for 22 patients. This group was not significantly different from the remainder of the original population in terms of any initial variables, as determined from their case notes.

Patients who had died

Twenty patients had died by 1983. The causes of death were: suicide (6), cancer (5), myocardial infarction (4), accident (2), cerebrovascular accident (1), respiratory failure (1) and pulmonary embolus (1). At the time of death, eight of these were known to still be taking lithium. In comparing the 20 dead subjects with the rest of the original population, there was no major differentiating characteristic of statistical significance. All six who committed suicide had been diagnosed as having bipolar illness, in each case characterised by severe depressions which prevented them from working or functioning socially; five had shown a partial response to lithium and one had shown no response. The average age at time of death was 47 years (range 30–56).

Patients who were interviewed

A total of 59 patients were re-interviewed in 1983. On examination of their mental state, including use of the PSE, none of these were found to have clinically significant psychiatric signs or symptoms, either at the time of interview or in the preceding 4 weeks. It was therefore presumed that the data which they provided at those interviews were valid.

Lithium usage

Their average age at starting lithium was 45 years (range 19–75). Figure 1 shows the length of time for which these patients had regularly taken lithium; most had taken it for more than 13 years.

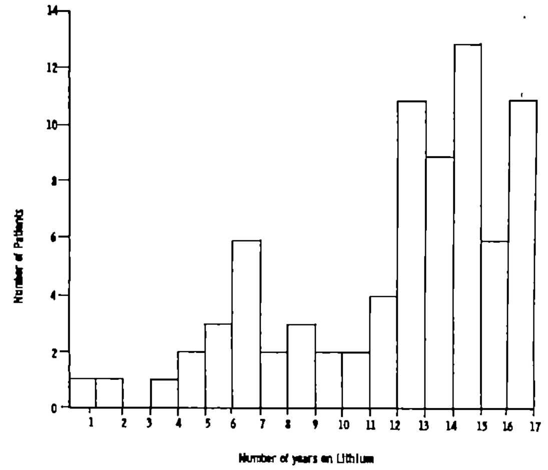

FIG. 1. Length of time for which patients had taken lithium (total = 957 patient-years on lithium).

The mean daily dose was 800 mg (range 250–1600 mg), mostly as single daily dose preparations. The mean recorded serum level, approximately 12 h post-dose, was 0.7 mmol/litre (range 0.3–1.2 mmol/litre). Since lithium was first prescribed for them, 61% had taken the drug continuously. The reasons given by the others for stopping lithium were: unwillingness to take long-term treatment (12), unacceptable side-effects (11), the treatment considered ineffective by the doctor (4), concomitant physical illness (3) and in order to become pregnant (1). Twenty-nine of the total 31 who stopped taking lithium suffered relapses, and 19 subsequently recommenced their treatment. Two subjects (both with a unipolar illness) have remained free of recurrence since stopping lithium and have now been well, without medication, for over 6 years.

Outcome

As described above, the subjects were all assigned to one of three outcome groups, both in 1971 and in 1983. In 1971, (when 101 subjects were included), 48% had shown a complete response, 42% a partial response and 10% an insignificant response. In 1983, (when 59 subjects were re-examined), 49% had shown a complete, 41% a partial and 10% an insignificant response. In the 12 years since these patients had been reviewed, the proportions in each group had not changed and there was no movement between outcome groups of the 59 patients followed up in 1983.

Having categorised the patients in terms of their response to lithium, all the preliminary and follow-up data were examined to see whether there were any variables which correlated with outcome (using the chi-squared test). No association was found between outcome and any of the variables examined, which included: age, sex, family history, age of onset of illness, diagnosis, previous treatment, previous physical history, nature and severity of their pre-lithium illness, dose of lithium, average serum level and concurrent treatment or side-effects.

During the period of lithium treatment, the marital and socioeconomic status of these patients had remained similar to that pre-lithium, but a higher proportion were able to work fulltime (36 compared with 12 before lithium).

At the same time of follow-up, four subjects were found to be living in institutions (one in a long-stay ward of a psychiatric hospital, three in old people's homes).

Other treatment

Additional treatment was given concurrently to 43 (73%) of these patients at some point during their lithium treatment. In the case of patients who had no further episodes of affective illness after starting lithium, the additional treatment given was either hypnotic drugs or psychotherapy. The percentages receiving other forms of treatment, compared with the pre-lithium figures, are shown in Table I.

Side-effects

The side-effects recorded in the case notes, or reported on detailed questioning at interview of the 59 patients, were as follows: tremor (23 subjects), subjective memory loss (23), weight gain (21), polyuria or polydipsia (21), loss of creativity (11), diarrhoea (10), hypothyroidism (10), goitre (7), nausea (5), impotence (5), gastric pain (4) and psoriasis (4). Hypothyroidism was diagnosed biochemically by routine measurement of plasma T_3 and T_4 and of TSH. Eleven subjects stopped taking lithium because of side-effects. None of the 59 patients seen in 1983 had any known renal disease and no other side-effects which might be specifically associated with prolonged use of lithium were detected.

TABLE I
Percentages of patients receiving other forms of treatment

Other treatment given	*Prior to lithium treatment (mean 5 years)*	*Concurrent with lithium treatment (mean 11 years)*	*P value*
Tricyclic antidepressants	88	61	<0.01
Monoamine oxidase inhibitors	43	19	<0.01
Barbiturates	69	12	<0.01
Benzodiazepines	70	47	<0.01
Phenothiazines	62	34	<0.01
Electroconvulsive therapy	58	12	<0.01
	(4–180 treatments)	(3–12 treatments)	<0.01
Psychotherapy/analysis	31	20	0.1

Pregnancies

One woman became pregnant whilst taking lithium, and this pregnancy was terminated for social reasons. One other woman stopped lithium in order to become pregnant; she became hypomanic after the birth, and lithium treatment was re-instituted.

There were no reports of infertility amongst these subjects, although their age and illness meant that most were not wishing to have more children. The wives of four of the male subjects became pregnant; there were no reports of physical or psychological problems in these children.

Discussion

This study presents retrospective data from a large cohort of patients taking prophylactic lithium over a period of up to 17 years. Potential bias was introduced, in that 22 patients were either not traced or would not be interviewed. It is possible that these subjects were those in whom lithium had not produced a favourable response, and that they were therefore unwilling to cooperate with the study. This group of patients is also unusual in the marked bias towards social classes I and II, which is thought to be due to the inclusion of patients referred from outside London and even from other countries, at a time when lithium was not widely used or available. These latter patients were predominantly from the higher social classes.

Despite these methodological shortcomings, the results obtained do reveal several interesting points. First, 50% of patients showed a complete remission whilst on prophylactic lithium, 40% showed a partial but significant response and 10% a negligible response. This response was true of both unipolar and bipolar patients, and the prophylactic effect of lithium carbonate did not diminish with time in the patients who showed a good response.

The percentage of patients who show no apparent response to prophylactic lithium varies considerably in published studies. However, all studies agree that there is a significant proportion of both unipolar and bipolar patients who will relapse whilst taking lithium carbonate. This study has followed subjects over a long period of time, and has indicated that the subjects can be categorised according to response within the first 5 years of treatment. On examining the same subjects 12 years later, none had moved from one outcome group to another. It has previously been suggested by Dunner & Fieve (1974) that if patients are followed for long enough, they will all ultimately have a further affective episode, but this study did not confirm that hypothesis.

Secondly, in the bipolar group of patients, lithium was equally effective in preventing hypomanic and depressive episodes. This has been a subject of debate for some years, since Prien *et al* (1971, 1973*a*,*b*, 1974) found that lithium had a greater effect in preventing the manic phase of illness rather than the depressive phase. However, Baastrup *et al* (1970) and Coppen *et al* (1963) have found essentially similar prophylactic effects against the manic and depressive phases in their bipolar patients, and the results of this study, with a larger number of patients, confirm that finding.

Thirdly, these results do not show that a positive family history of bipolar illness indicates a more favourable response to prophylactic lithium; nor were we able to isolate any other specific factor which was correlated significantly with outcome. This conflicts with the strong correlation found by Mendlewicz *et al* (1973) between positive family history of bipolar illness and good response to prophylactic treatment. However, using a life-table method of analysis on a cohort of 120 patients, Dunner *et al* (1976*a*) found no association between negative family history and failure of lithium prophylaxis. In addition, on analysis of the relationship between sex, age at treatment, diagnosis, number of previous episodes, age of onset of illness and outcome of treatment, they concluded that there was no significant association between any of these factors and outcome. Our long-term study substantiated this evidence and also demonstrated no correlation between eventual outcome and certain other factors, including previous physical history, previous treatment, severity of illness before treatment, symptoms before treatment, dose, serum level and occurrence of side-effects.

Fourthly, only three of our subjects could be defined as 'rapid cyclers' (four or more affective episodes per year), and all of these seemed to derive considerable benefit from prophylactic lithium. Dunner & Fieve (1974) described this subgroup of manic-depressive patients and noted that they seemed not to respond to prophylactic lithium. However, in a later paper, Dunner *et al* (1976*b*) re-analysed the data from their sample of 'rapid cyclers' and concluded that lithium carbonate was effective in preventing hypomanic episodes in these patients.

Fifthly, the mean serum level, measured 12 h post-dose, was 0.7 mmol/litre in all three outcome groups, with no correlation between mean serum level and outcome. The guidelines concerning desirable plasma lithium levels for prophylaxis are still conflicting. A number of studies have indicated a higher rate of relapse among subgroups of patients maintained with average serum levels below 0.4 mmol/litre (Hullin, 1980) and 0.8 mmol/litre (Prien & Caffey, 1976). Recently, however, Peselow *et al* (1982) examined a large cohort of patients maintained on prophylactic lithium alone and found that they were unable to demonstrate a minimum level of lithium below which prophylaxis against depression is ineffective. Sashidaran *et al* (1982) followed a cohort of 53 patients on lithium therapy and retrospectively detailed their serum level and mental state over a period of 3 years. They found that those who did not relapse during the trial had spent significantly less time with serum levels over 0.9 mmol/litre than had those who relapsed.

Finally, the side-effects found in our subjects have all been previously well documented. There was a surprisingly high incidence of clinical hypothyroidism, and further work is in progress to examine this group in more detail. We were not able to demonstrate any side-effects specifically associated with long-term lithium treatment, but in view of the association of tardive dyskinesia and long-term neuroleptic treatment, it is essential to continue to monitor closely those patients who have taken lithium for extended periods, in case of similar developments.

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References

Baastrup, P. C. & Schou, M. (1967) Lithium as a prophylactic agent against recurrent depression and manic-depressive psychosis. *Archives of General Psychiatry*, **16**, 162–172.

—— & —— (1968*a*) Prophylactic lithium. *Lancet*, **i**, 1419–1422.

—— & —— (1968*b*) Prophylactic lithium. *Lancet*, **ii**, 349–350.

——, Poulsen, K. S., Schou, M. *et al* (1970) Prophylactic lithium: double-blind discontinuation in manic-depressive and recurrent depressive disorders. *Lancet*, **ii**, 326–330.

Benaim, S. & Lappin, F. (1973) La terpaia con sali di litio nelle malattie maniaco-depressive. *Rivista Sperimentale di Frenatria*, **97**, 677–691.

Blackwell, B. (1969) Need for careful evaluation of lithium. *American Journal of Psychiatry*, **125**, 1131.

—— & Shepherd, M. (1968) Prophylactic lithium: another therapeutic myth? *Lancet*, **i**, 968–971.

Coppen, A., Peet, M., Bailey, J. *et al* (1963) Double-blind and open prospective studies of lithium prophylaxis in affective disorders. *Psychiatria Neurologia, Neurochiragia*, **76**, 500–510.

——, Noguera, R., Bailey, J. *et al* (1971) Prophylactic lithium in affective disorders. *Lancet*, **ii**, 275–279.

Cundall, R. L., Brooks, P. W. & Murray, L. G. (1972) A controlled evaluation of lithium prophylaxis in affective disorders. *Psychological Medicine*, **2**, 308–311.

Dunner, D. L. & Fieve, R. R. (1974) Clinical factors in lithium carbonate prophylaxis failure. *Archives of General Psychiatry*, **30**, 229–233.

——, Fleiss, J. L. & Fieve, R. R. (1976*a*) Lithium carbonate prophylaxis failure. *British Journal of Psychiatry*, **129**, 40–44.

——, Stallone, F. & Fieve, R. R. (1976*b*) Lithium carbonate and affective disorders. *Archives of General Psychiatry*, **33**, 117–120.

——, —— & —— (1982) Prophylaxis with lithium carbonate: an update. *Archives of General Psychiatry*, **39**, 1344–1345.

Hullin, R. P. (1980) Minimum serum lithium levels for effective prophylaxis. In *Handbook of Lithium Therapy* (ed. F. N. Johnson). Lancaster: MTP Press.

Kane, J. M., Quitkin, F. M., Rifkin, A. *et al* (1982) Lithium carbonate and imipramine in the prophylaxis of unipolar and bipolar illness. *Archives of General Psychiatry*, **39**, 1065–1069.

Mendlewicz, J., Fieve, R. & Stallone, F. (1973) Relationship between effectiveness of lithium therapy and family history. *American Journal of Psychiatry*, **130**, 1011–1013.

Persson, G. (1972) Lithium prophylaxis in affective disorders. *Acta Psychiatrica Scandinavica*, **48**, 462–479.

Peselow, E. D., Gulbenkian, G., Dunner, D. L. *et al* (1982) Relationship between plasma lithium levels and prophylaxis against depression in bipolar I, bipolar II and cyclothymic patients. *Comprehensive Psychiatry*, **23**, 176–180.

Prien, R. F. & Caffey, E. M. (1976) Relationship between dosage and response to lithium prophylaxis in recurrent depression. *American Journal of Psychiatry*, **133**, 567–570.

——, —— & Klett, C. J. (1971) Lithium carbonate. *Diseases of the Nervous System*, **32**, 521–531.

——, —— & —— (1973*a*) Prophylactic efficacy of lithium carbonate in manic-depressive illness. *Archives of General Psychiatry*, **28**, 337–341.

——, Klett, C. J. & Caffey, E. M. (1973*b*) Lithium carbonate and imipramine in the prevention of affective episodes. *Archives of General Psychiatry*, **29**, 420–425.

——, Caffey, E. M. & Klett, C. J. (1974) Factors associated with lithium responses in the prophylactic treatment of bipolar manic-depressive illness. *Archives of General Psychiatry*, **31**, 189–192.

Sashidaran, S. P., McGuire, R. J. & Glen, A. I. M. (1982) Plasma lithium levels and therapeutic outcome in the prophylaxis of affective disorders: a retrospective study. *British Journal of Psychiatry*, **140**, 619–622.

Schou, M. (1968) Lithium in psychiatric therapy and prophylaxis. *Journal of Psychiatric Research*, **6**, 67–95.

Stallone, F., Shelley, E., Mendlewicz, J. *et al* (1973) The use of lithium in affective disorders: a double-blind study of prophylaxis in bipolar illness. *American Journal of Psychiatry*, **130**, 1006–1010.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

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The Effect of Carbamazepine and Lithium on Remission from Affective Illness

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Times in remission from affective illness were prolonged during treatment with lithium carbonate and carbamazepine, but lithium was the more effective. Similar global scores for the two drugs suggested that other factors may determine their acceptability.

The salts of lithium are used for the treatment of recurrent affective disorders to reduce the frequency and severity of illness, but their low therapeutic index and tendency to induce side- and toxic effects in some individuals have led to the search for substances with the same therapeutic properties and fewer disadvantages. Recent studies have suggested that carbamazepine may be an alternative to lithium in this context (Takezabi & Hanaoka, 1971; Okuma *et al*, 1973, Inanaga *et al*, 1981; Ballenger & Post, 1980; Kishimoto *et al*, 1983; Nolen, 1983), and a pilot comparison of these two drugs is reported here.

Method

The protocol was for a randomised double-blind retrospective/prospective study of the mood-stabilising properties of lithium and carbamazepine in respect of time in remission from affective illness. Many of the patients knew about and were aware of the early symptoms of relapse in their case, and our normal practice is to give them the option of taking antidepressant drugs temporarily at these times. This option was retained for ethical reasons, and for those patients who did not fulfil the criteria of full relapse (i.e. a Hamilton depression score (1967) of less than 10) during this period, the presence of this attenuated and treated minor episode was not counted as interrupting time in remission. Thus, the trial is really that of the effect of lithium and carbamazepine on this mode of management of affective illness. This design might have biased the results, but the option to take antidepressants temporarily was taken up by 61% on lithium and 63% on carbamazepine.

Patients were accepted into the trial if: (1) they were experiencing mania or a major depressive episode, using the criteria of DSM-III, (2) there was a well-defined recorded previous episode, and a known time of recovery from which the time in remission without the benefit of a prophylactic could be estimated. The previous period in remission had not been 'covered' by putative prophylactic drugs (including tricyclic or MAOIs) either because the patient was coming *de novo* to this form of therapy or, thinking he no longer needed it, had defected from such treatment. Of the 52 accepted for the trial, only seven were withdrawn for the combined reasons of having moved away or non-compliance.

These criteria also applied for subsequent episodes of illness. Remission was timed from the beginning of therapy with lithium or carbamazepine, which was usually at the end of the second week after achieving scores of 10 or less on the Montgomery–Åsberg (1979) or 3 or less on the Modified Manic State scales (Blackburn *et al*, 1977). If relapse occurred within 3 weeks of starting lithium or carbamazepine, that person was taken out of the trial; this applied to only two patients.

Side-effects were rated non-blindly and globally by the prescribing medical staff, using the scale of mild, moderate and severe. Only volunteered complaints which were rated as moderate or severe were counted, and these occurred in six of the 18 on lithium and three of the 19 on carbamazepine (χ^2, NS).

The global ratings for patients (self-rating) and doctors had three choices: the same or worse/improved/completely well. The doctors completed their rating at the end of the trial period for that patient; all subjects in the analysis completed and returned their self-rating scales. The form was sent by post to avoid—as far as possible—the tendency of the patient to want to please the staff.

All patients underwent a physical examination and the following tests: haemoglobin, red cell count, white cell count, urea, electrolytes, creatinine clearance, urine for protein/casts, thyroid profile, serological tests to exclude syphilis, skull/chest X-ray, ECG (and liver function tests in some). None with evidence of organic illness was accepted. All the patients included gave informed consent.

The drugs used were tablets of lithium carbonate (400 mg) and carbamazepine (200 mg), with appropriate placebo preparations for each. The patients were given an active drug and the opposite placebo (lithium [or its placebo] in split dosage (*b.i.d.*) and carbamazepine [or its placebo] at night). Active and placebo drugs were increased in parallel. Change in initial dosage was made every 5–7 days until plasma levels of 0.4–0.9 mmol/litre for lithium and 5–12 μg/ml for carbamazepine were reached in samples taken 12 ± 0.5 h after the previous night's dose.

Antimanic or antidepressant drugs were given as required for a relapse. Lithium was discontinued if the episode was manic and haloperidol was to be used; otherwise, both drugs were carried on through the illness.

The mean times the patients were observed are given in Table I. No patients were admitted to the trial from 6 months before it was due to terminate, and no data were included unless the period of observation of the patient had exceeded the previous cycle length (time between onset or index and preceding illness). Since the therapies on trial were started at the beginning of the period of remission, the

TABLE I
Patients entering and completing the trial

	Lithium	*Carbamazepine*
Number	18	19
Diagnosis: unipolar	10	7
bipolar	8	12
Males	11	12
Females	8	7
Average time in trial (months)	20	16
Ages (years): 20–29	3	3
30–39	2	3
40–49	4	4
50–59	5	4
60+	6	5

comparison used was that of the time in remission rather than the length of cycle.

Patients were assessed in the index illness and then weekly for 4 weeks for psychometry and blood levels (for some patients, a fifth drug assay was included). They were then reassessed every 2 months for repeat drug assays, psychometry and white cell count. At 6-month intervals, the thyroid profile, blood urea and creatinine clearance tests were repeated. Subjects were asked to make additional visits if they experienced mood changes lasting more than 3 days or had problems with their drugs.

The protocol excluded patients with white cell counts of 3.5×10^9/litre and any whose counts fell below this level, but this did not occur in the sample studied.

The medical staff who rated the patients were separate from those who prescribed, checked blood levels, altered dosage, and monitored laboratory tests and side-effects. These side-effect scores were assessed non-blindly by the prescribing doctors. The other ratings were made blindly, and the raters limited their enquiries to this task. The maintenance of blindness of the ratings was tested by a guess score at the end of the trial. There were 23 correct guesses out of 38 (NS), which suggests that double-blind conditions had been maintained, at least as far as the raters were concerned.

However, the same did not apply to the patients. Some were able to 'break the code', probably because of familiarity with the side-effects of lithium. This seemed inevitable in a prolonged trial. Despite the correct guesses by some, when it became clear that patients were working out their drug therapies correctly, they were asked not to, and apparently did not reveal this information to the raters. Allocation to the two treatments was by a series of random numbers.

If two episodes occurred, the trial was terminated for that patient. A total of 52 patients were selected for the trial and agreed to participate, of which 37 completed. The reasons for losses and additional exclusions from the trial were: intolerance to drug during the start of therapy (one on lithium and one on carbamazepine), one case of hypothyroidism during therapy (lithium), relapse in first 3 weeks of therapy (two cases). There were seven cases of non-compliance or of moving away, making a total of 15 patients accepted for the trial but not completing it.

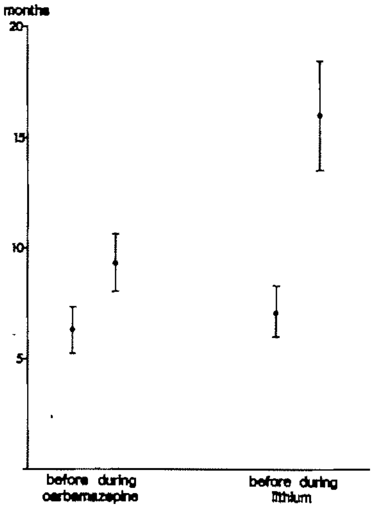

FIG 1 Times in remission (means ± s.e.). Carbamazepine: $P<0.001$; lithium: $P<0.001$.

Results

Table I gives the ages, sex, diagnoses and average times of observation in the patients who completed the trial. The mean times in remission before and after each drug are illustrated in Figure 1. The mean time (±s.e.) before starting lithium carbonate was 7.1 ± 1.1 months and was 16.0 ± 2.4 while taking this drug. The mean (±s.e.) for carbamazepine was 6.3 ± 1.0 before the 9.4 ± 1.3 afterwards. Both differences were significant ($P<0.001$ in each case, t-test for paired observations). The 'additional time' in

TABLE II
Global assessments

	Self-rated		*Independent rater*	
	Lithium	*Carbamazepine*	*Lithium*	*Carbamazepine*
Failure/no change	5	4	3	3
Significant improvement	12	13	7	10
Well	1	2	8	6

There were no significant differences between the drugs.

remission while on lithium was 9.3 ± 2.0 months (mean ± s.e.) as compared to an extra 3.3 ± 0.8 months on carbamazepine, so that lithium was significantly the better drug ($P < 0.001$, t-test). A shorter time of observation would be expected if patients relapsed more rapidly with carbamazepine, and this was observed.

Discussion

From the now considerable work on lithium and from the view that affective illnesses tend to occur more frequently with age (Angst *et al*, 1969; Grof *et al*, 1970), it is to be anticipated that cycle length and time in remission will decrease with succeeding episodes. In neither group did this occur, and in fact time in remission was increased. Lithium, with or without additional medication for minor deterioration, was significantly superior in this respect to carbamazepine.

Our conclusion from this preliminary study has confirmed the observation that lithium significantly lengthens time in remission, while the effect of carbamazepine is more modest. However, it is an alternative to be considered in patients who have been intolerant of lithium or whose illness does not respond to this drug. The similarity of global scores may also point to other factors which may enter into the tolerance to and acceptability of the two drugs, such as the particular pattern of even mild side-effects, e.g. tremor and polyuria, which it was felt was the means by which patients 'broke the code'.

The reason that this particular design was chosen was partly because in a retrospective/prospective study it is difficult sometimes to pinpoint time of onset of a previous illness. Another reason was that prophylactic therapy cannot be started with carbamazepine during treatment if this consists of electroconvulsive therapy (ECT). As it is our practice to give ECT, wait to see if recovery is complete, then give one or two more if the patient is relapsing, an individual patient may be 2 or 3 weeks into remission before a prophylactic drug is started. The most appropriate comparative measure is therefore remission time in patients in whom carbamazepine or lithium is started 2 or 3 weeks into this period.

Also, one of the criteria for entry was a clearly defined and recorded time of recovery from a previous episode. Thus, it was possible to calculate reasonably accurate times in remission and to determine from the recent and therefore accessible history the time of relapse into the current episode.

The design might be criticised on the grounds that additional therapy was given for relapses and that blindness to therapy on the part of the patients was lost. The option to use additional therapy for episodes of illness was retained for ethical reasons and was taken by similar proportions of patients in the two groups. As regards breaking of the code by the patient, in our view this is inevitable in a long trial where one of the drugs — in this case lithium — has distinctive identifying side-effects.

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References

ANGST, J., DITTRICH, A. & GROF, P. (1969) Course of endogenous affective psychosis and its modification by prophylactic administration of imipramine and lithium. *International Pharmacopsychiatry*, **2**, 1–11.

BALLENGER, J. C. & POST, R. M. (1980) Carbamazepine in manic-depressive illness. A new treatment. *American Journal of Psychiatry*, **137**, 782–790.

BLACKBURN, I. M., LOUDON, J. B. & ASHWORTH, C. M. (1977) A new scale for measuring mania. *Psychological Medicine*, **7**, 453–458.

GROF, P., SCHOU, M., ANGST, J., BAASTRUP, P. & WEIS, P. (1970) Methodological problems of prophylactic trials in recurrent affective disorders. *British Journal of Psychiatry*, **116**, 599–619.

HAMILTON, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychiatry*, **6**, 278–296.

INANAGA, K., OTSUKI, S., SARAI, K., TAKATASHI, R., HAZAMA, H., MORI, A. & WATANABE, M. (1981) A preliminary double-blind study of the efficacy of carbamazepine in prophylaxis of manic depressive illness. *Psychopharmacology*, **73**, 95–96.

KISHIMOTO, A., OGURA, C., HAZAMA, H. & INOUE, K. (1983) Long term prophylactic effects of carbamazepine in affective disorders. *British Journal of Psychiatry*, **143**, 327–331.

MONTGOMERY, A. S. & ÅSBERG, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, **134**, 382–389.

NOLEN, W. A. (1983) Carbamazepine, a possible adjunct or alternative to lithium in bipolar disorder. *Acta Psychiatrica Scandinavica*, **67**, 218–225.

OKUMA, T., KISHIMOTO, A., INOUE, K., MATSUMOTO, H., OGURA, A., MATSUSHITA, T., NAKAO, T. & OGURA, C. (1973) Antimanic and prophylactic effects of carbamazepine on manic depressive psychosis. *Folia Psychiatrica and Neurologica Japonica*, **27**, 283–297.

TAKEZAKI, H. & HANAOKA, M. (1971) The use of carbamazepine in the control of manic depressive psychosis and other manic depressive states. *Journal of Clinical Psychiatry*, **13**, 173–182.

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Suicide and Parasuicide Among Further Education Students in Edinburgh

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During the period 1979–1982 the incidence of suicide and parasuicide for students aged 15–24 years was found to be lower than that of others of the same age in Edinburgh. A comparison of student parasuicides with matched non-student parasuicides shows similar parasuicide repetition rates, but some differences in respect of precipitants (twice as many students reporting no major event prior to the episode), agent of poisoning (analgesics more commonly used by students; hypnotics, tranquillisers and barbiturates, by controls), and social and clinical characteristics (a greater proportion of controls reporting parasuicide in the family, a criminal record and being the victim of violence).

Much published research appears to confirm the popular view that students are excessively prone to suicide. Among male students at Cambridge, Oxford and University College, London, in the post-war years, suicide rates were significantly in excess of those expected for the relevant age group (Carpenter, 1959). Even the reduced rate of 10 per 100 000 among Oxford University males during 1966–1976 (Hawton *et al*, 1978) was twice as high as the rate for males aged 15–24 in England and Wales at the mid-point in the study (1971). However, in the only study of its kind, Hawton *et al* (1978) reported lower parasuicide inception rates among students compared to non-students.

This study reports the results of an investigation into the incidence of suicide and parasuicide among students in further education in Edinburgh over the period 31 January 1979 to 31 December 1982. Clinical and sociodemographic characteristics of student parasuicides are compared with those in a matched sample of non-student (control) parasuicides. Results are discussed in the light of findings reported in previous studies.

Method

Suicide

Using information provided by the General Register Office (Scotland) a count was made of all 'suicides' (ICD codes E950–E959) or 'undetermined' (E980–E989) deaths among students aged 15 years and over and usually resident in the city of Edinburgh. 'Undetermined' deaths are included because these tend to resemble suicides more closely than accidents (Holding & Barraclough, 1978). Three major problems arise when using this data source. Firstly, the deceased person may have been in the secondary, rather than tertiary, education sector. Unfortunately, it was not possible to ascertain the name of the institution where the person was a student, nor details of year of study, course or degree. Secondly, the person may have been studying outside Edinburgh but have committed suicide in the city. If his/her 'usual address' was held by GRO to be the parental home in Edinburgh, then this death would be included in the sample. Thirdly, any Edinburgh student committing suicide outside Scotland was not recorded. (However, deaths of Edinburgh residents occurring outside Edinburgh, but elsewhere within Scotland, are included.) While it is not possible to calculate precisely the net effect of these possible errors, they probably result in an underestimate of the frequency of student suicide in the city.

Parasuicide

Parasuicide is defined as 'a non-fatal act in which an individual deliberately causes self-injury or ingests a substance in excess of any prescribed or generally recognised therapeutic dosage' (Kreitman, 1977). This definition excludes intoxication with alcohol alone. Information was collected on all identified cases of parasuicide involving students currently resident in Edinburgh and registered at an institution of further education in the city, who were admitted to the Regional Poisoning Treatment Centre (RPTC) at the city's Royal Infirmary during the study years. Over 90% of all adult patients admitted to hospital from an Edinburgh address for treatment of self-poisoning are received by the RPTC (Holding *et al*, 1977). All referrals are admitted regardless of the severity of their physical condition (Matthew *et al*, 1969), but some must be missed. The RPTC also receives cases of self-injury, although these are less systematically referred than the cases of self-poisoning and are therefore likely to be a less representative sample of the total number of such cases. Kennedy & Kreitman (1973) have shown that parasuicides admitted to the RPTC are representative of all medically treated parasuicides. Mindful that an unknown number of student parasuicides will not have been included in this hospital sample, I made enquiries at each institute of further

TABLE I

Parasuicide rates (per 100 000) among students and non-students aged 15–24 years in Edinburgh City, 1979–1982 (term-times only)

	1979			*1980*			*1981*			*1982*			*1979–1982*		
	n Parasuicides	*Population*	*Rate*	*n Parasuicides*	*Population*	*Rate*	*n Parasuicides*	*Population*	*Rate*	*n Parasuicides*	*Population*	*Rate*	*Mean n Parasuicides*	*Mean population*	*Mean rate*
Males															
Students	7	10 882	64	5	10 857	46	4	10 402	38	7	10 853	64	5.75	10 749	53
Non-students	57	30 401	187	61	30 426	200	80	30 685	261	73	29 478	248	67.75	30 254	224
Females															
Students	9	8696	103	12	8726	138	11	8923	123	17	9040	188	12.25	8846	138
Non-students	102	32 212	317	106	32 182	329	123	31 647	389	94	30 691	306	106.25	31 683	335

education in the city. However, very few cases came to light; all were already included in the sample.

A control group of non-student parasuicides was drawn from the RPTC archival data set. Each parasuicide was matched with the next parasuicide admitted to the RPTC who fulfilled the following criteria: resident in Edinburgh, not a student, same gender and same age (within one year). Comparisons between students and non-students were made on all sociodemographic, clinical, treatment and social problem variables routinely recorded for RPTC admissions. The significance of differences between groups was assessed by means of the McNemar Test (Siegel, 1956). In addition, information was gathered on the total number of admissions in the two groups over the study period. Finally, an attempt was made to compare groups on the presence and type of any major life event preceding the episode. Information was taken from letters written by the RPTC psychiatrist to the person's general practitioner on his/her discharge from the ward.

Epidemiology of suicide and parasuicide among students

The calculation of suicide and parasuicide rates for students is complicated by three problems. Firstly, those studying in Edinburgh are only definitely resident in the town for approximately 30 weeks of the year; during the vacations they may be living elsewhere. Secondly, students show a wide age range. A third problem concerns the amount of time spent weekly in connection with studies at a further education institution, which varies from a few hours part-time on secondment to a full-time degree course. For these reasons, the numerator is restricted to students studying full-time, aged 15–24 years and whose suicide or parasuicide occurred during term-time. An additional difficulty lies in the fact that students are known to be unrepresentative of the general population of the same age in respect of social class distribution. Unfortunately, it has not proved possible to obtain a social class breakdown of the student population in Edinburgh, and information on the social class position of student suicides and parasuicides is also unavailable.

On the basis of estimates provided by the further education institutions and by the Education Department of the Scottish Office, the numbers of full-time students in Edinburgh aged 15–24 years were calculated for each year of the survey. Denominators for non-students were obtained by subtracting the estimated number of students from the estimated total for the whole age group (as provided by GRO). Because of the small numbers involved, only two mean suicide rates (one for students and one for controls) were calculated for the whole period, the total number of term-time suicides (and undetermined deaths) over the 4 years being divided by the relevant population estimates for 1981, the resultant fraction being expressed as a rate per 100 000. For parasuicide, a rate per 100 000 was calculated for each year based on the unduplicated count of persons aged 15–24 admitted to the RPTC during the 30 weeks of term-time (numerator) and the appropriate population estimates (denominator). The four rates were then averaged to give a mean parasuicide rate for the whole period. Parasuicide rates were calculated separately for males and females, and separately for students and non-students. It is important to note that both suicide and parasuicide rates cover the same 30 week periods (term-time) for students and non-students alike.

Significance tests are not provided or required when assessing differences between student and non-student rates. Numbers of suicides and parasuicides are either total counts or estimates of total counts, and no attempt is made to generalise from Edinburgh's experience to the rest of Great Britain.

Results

Suicide and undetermined death

Between 1979 and 1982 there were three student suicides in Edinburgh: a female, single, aged 21, died by carbon monoxide poisoning; a male, divorced, aged 38, died by jumping (this death occurred during the vacation); a male, single, aged 19, died by hanging; and one 'undetermined' death: a male, single, aged 18, who died by falling. None had a recorded admission to the RPTC. The mean 4-year suicide rate during term-time for the 15–24 year age group (omitting the male aged 38) was 2.6 ($n=2$) per 100 000 among students and 8.8 ($n=22$) among non-students. The mean rate for suicide and undetermined deaths combined was 3.8 ($n=3$) and 11.6 ($n=29$), respectively. Thus, students appear to have been less at risk of suicide and undetermined death than their non-student peers, at least during this 4-year period.

Parasuicide

During 1979–1982 91 students (27 male, 64 female) were admitted to the RPTC following an episode of deliberate self-harm. The mean age for males was 21.7 years (s.d. 4.0; range 17–34) and for females 20.7 years (s.d. 4.3; range 16–42).

Table I shows, separately for each year, the numbers of student and non-student parasuicides (on a term-time only basis) in the 15–24 age group, together with population estimates. Throughout the period, parasuicide was less frequent among students. Taking the mean 4-year rates, the relative risks were 1:4:2 for men and 1:2:4 for women.

While direct control for the likely skewed social class distribution among students has not been possible (see above), an indirect assessment of its confounding effect can be made, at least for males. During the study period, parasuicide rates among economically active 16–24-year-old males in Edinburgh ranged from an average of 110 per 100 000 in social classes I and II, to 537 in class V (calculated over a 30-week period). Thus the rate among professional men is double the rate of their student age peers. We can reasonably conclude that Edinburgh students have a lower parasuicide rate than other people of the same age and social class.

TABLE II

Distribution of student parasuicide by month of admission

Month	*Observed*	*Expected*[1]
January	12	8.3
February	8	7.5
March	10	8.8
April	6	6.6
May	9	8.2
June	4	7.6
July	3	7.2
August	3	9.3
September	5	7.8
October	15	6.2
November	9	7.5
December	7	6.0
	91	91.0

1. Based on the distribution of non-student admissions, 1979–1982
$\chi^2 = 24.4$, d.f. = 11, $P < 0.02$

The actual distribution of the 91 student parasuicides by month of admission was significantly different to the expected distribution based on non-student admissions over the study period ($\chi^2 = 24.4$, d.f. = 11, $P < 0.02$). The relevant data are provided in Table II. Two-thirds of admissions occurred during the period October to March (15 admissions recorded in October alone) and only one-third during the rest of the year (just four in June when most examinations are taken).

Parasuicide repetition was assessed among the 91 student parasuicides and their matched controls. Table III shows that nine persons in each group were readmitted to the RPTC. Students were thus no more likely to be readmitted than their matched controls. The total number of admissions was 113 among students (five persons admitted twice, two admitted four times, one admitted six times and one admitted seven times), and 104 among controls (six admitted twice, two admitted three times, one admitted four times).

Table IV lists the social and clinical variables on which significant differences ($P < 0.05$) between students and controls were found. More students were unmarried and living outside the family; they were also more likely to use non-prescribed drugs. A greater proportion of controls had been the victims of violence, reported an episode of parasuicides by a near relative, lived in overcrowded conditions and had a criminal record. Differences on a further 21 variables (listed in Table IV) did not reach statistical significance.

The most common method of parasuicide was self-poisoning, either alone (86 controls, 83 students) or in conjunction with self-injury (two controls and two students). Analgesics were used by more students ($n = 36$) than controls ($n = 22$); on the other hand, tranquillisers, hypnotics and barbiturates were more commonly taken by controls (29) than by students (9).

An analysis of major immediate precipitants reveals that nearly twice as many students (33) as controls (17) reported no precipitant, while many more controls (55) than students (35) referred to interpersonal problems. Academic difficulties were noted in only 18 cases (and in eight of these interpersonal problems were also present).

TABLE III

Readmission to RPTC during study period: students versus controls

		Students	
		Readmitted	*Not readmitted*
Controls	Readmitted	0	9
	Not readmitted	9	73

$n = 91$ matched pairs
$\chi^2 = 0$

TABLE IV

Clinical and social characteristics: significant differences between students and controls*

Variable	*Controls (n=91)*	*Students (n=91)*
Civil state		
Not married/cohabiting	62	85
Source of drugs		
Non-prescribed	18	36
Present household		
Not living with family	28	58
Violence		
Victim of excessive force in past 5 years	24	3
Episode of parasuicide		
By near relative	22	8
Overcrowding		
Yes	10	0
Criminal record		
In prison or conviction	18	5

*McNemar test, $P < 0.05$: n of pairs differs for each variable because of missing data.

No significant differences between groups were found in respect of agent of referral; type of admission; coma level; psychiatric diagnosis; personality diagnosis; alcoholism; alcohol at time of act; previous in-patient and out-patient psychiatric treatment; previous parasuicide; permanent separation from mother or father before age 15; use of excessive force in past 5 years; psychiatric treatment received by family member; completed suicide by near relative; serious debts; police proceedings threatened; psychiatric or social work aftercare recommended; habitual misuse of drugs.

Discussion

Contrary to the findings of previous studies, this investigation has revealed a lower frequency of suicide in the student population than among non-student age peers. It is possible that the Edinburgh rate is artificially low due to inadequacies in case identification procedures. However, the same limitation to deaths occurring in the city was also a feature of the Oxbridge surveys (Parnell, 1951; Rook, 1959; Hawton *et al*, 1978). Likewise, the inability to adjust for possible social class differences between student and non-student populations is common to both the Edinburgh and Oxbridge surveys. The discrepancy between this and previous studies is unlikely to be due to a secular trend towards lower suicide rates among students, since there has been a notable increase in suicide rates among adolescents and young adults over the past decade (McClure, 1984). Furthermore, an examination of suicide among students in Edinburgh over a 14-year-period (1968–1982) shows a tendency towards more deaths in the latter 7 years ($n=9$) than during the first half of the period ($n=3$). An alternative explanation for the findings might derive from the wide definition of 'student' employed in the present study. It may be that suicide risk is higher among university students compared to those studying in polytechnics, colleges of further education and teacher-training colleges. It was not possible to compute suicide (or parasuicide) rates according to the type of further education institution. However, even if both the term-time suicides which occurred during the period 1979–1982 were committed by university students, the mean suicide rate among such students would be 4.5 per 100 000, still considerably below previously reported rates.

The frequency of parasuicide was found to be lower among further education students in Edinburgh than among the rest of the 15–24 age group. Hawton *et al* (1978) reported a similar finding in Oxford in the 1970s, but were unable to control for social class variables. Indirect evidence suggests that the lower rate among students in Edinburgh was independent of any social class effect.

The matched-pairs analysis revealed that among those reporting some major precipitant of the parasuicidal episode, the majority of both groups cited interpersonal problems (argument, fight, conflict, actual or threatened termination of relationship), most commonly with a partner. Only eighteen students referred to academic difficulties, and in eight of these cases interpersonal problems were also present. The concentration of admissions at the beginning of the academic year (October, November) and their comparative rarity at the time of examinations (June) provides further evidence of the importance of social, rather than academic, factors in parasuicide among students. A similar conclusion was reached by Bernard & Bernard (1982). As Schwartz & Reifler (1980) suggested, membership in the young adult group (where social and family problems are commonplace) may be more important in understanding suicidal behaviour among college students than anything peculiar to the status of student. Investigations among school-age parasuicides has also demonstrated the relative insignificance of the school environment and its potential stresses in accounting for the child's behaviour (Kerfoot, 1979; Lumsden Walker, 1980).

A greater number of controls reported the occurrence of a major life event prior to parasuicide, a finding which might suggest more mental health problems among the students. However, there was no significant difference overall in the proportion of the two groups given a psychiatric or personality diagnosis. The finding could be an artefact of different reporting styles, including more denial of problems among the students, and to the limitations of the psychiatric discharge letters as a source of information on the topic. Nevertheless, it remains a possible marker of unidentified psychiatric morbidity in the student group. The finding of greater use of non-prescribed drugs (particularly analgesics) among students suggests that this group is less impulsive in planning the parasuicide and therefore perhaps also has a higher level of suicidal intent than their age peers. However, no data are available to confirm this suggestion, which is contrary to the finding by Hawton *et al* (1978) that student parasuicides are no more serious than others in their degree of suicidal intent. Whatever the 'seriousness' of the original parasuicide, the parasuicide repetition rate was shown to be identical in the two groups.

A final comment concerns the findings on the clinical and sociodemographic attributes of student and control groups. It was not surprising to discover that the student group contained a greater proportion of unmarried persons living outside the family home. Other significant findings may be accounted for by likely differences in the social class composition of the groups. Thus, receipt of violence, episode of parasuicide by near relative, overcrowding and criminal record have all been shown to be related to social class in the parasuicide population, with greatest prevalence found among the semi- and unskilled manual groups (Buglass, 1976). The difficulty of drawing any direct conclusion from the P values obtained in repeated significance tests on the same data, also has to be recognised.

On the basis of this evidence, it would appear reasonable to conclude that concern about the extent of suicidal behaviour among students may be misplaced. However, the discovery of a lower suicide rate in this group is unexpected and requires replication; and while the findings in relation to parasuicide confirm those reported from Oxford, they may not be generalisable to other cities or towns which have less well developed student health services. The data presented here should certainly not be used to justify a complacent attitude towards mental health problems in the student population.

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References

BERNARD, J. L. & BERNARD, M. L. (1982) Factors related to suicidal behavior among college students and the impact of institutional response. *Journal of College Student Personnel*, **23**, 409–413.

BUGLASS, D. (1976) The relation of social class to the characteristics and treatment of parasuicide. *Social Psychiatry*, **11**, 107–119.

CARPENTER, R. G. (1959) Statistical analysis of suicide and other mortality rates of students. *British Journal of Preventive and Social Medicine*, **13**, 163–174.

HAWTON, K., CROWLE, J., SIMKIN, S. & BANCROFT, J. (1978) Attempted suicide and suicide among Oxford University students. *British Journal of Psychiatry*, **132**, 506–509.

HOLDING, T. A. & BARRACLOUGH, B. M. (1978) Undetermined deaths—suicide or accident? *British Journal of Psychiatry*, **133**, 542–549.

——, BUGLASS, D., DUFFY, J. C. & KREITMAN, N. (1977) Parasuicide in Edinburgh—a seven year review 1968–74. *British Journal of Psychiatry*, **130**, 534–543.

KENNEDY, P. & KREITMAN, N. (1973) An epidemiological survey of parasuicide ('attempted suicide') in general practice. *British Journal of Psychiatry*, **123**, 23–34.

KERFOOT, M. (1979) Self-poisoning by children and adolescents. *Social Work Today*, **10** (45), 9–11.

KREITMAN, N. (ed.) (1977) *Parasuicide*. London: Wiley.

LUMSDEN WALKER, W. (1980) Intentional self-injury in school age children. *Journal of Adolescence*, **3**, 217–228.

MCCLURE, G. M. (1984) Recent trends in suicide among the young. *British Journal of Psychiatry*, **144**, 134–138.

MATTHEW, H., PROUDFOOT, A. T., BROWN, S. S. & AITKEN, R. C. B. (1969) Acute poisoning: organization and work-load of a treatment centre. *British Medical Journal*, **3**, 489–493.

PARNELL, R. W. (1951) Mortality and prolonged illness among Oxford undergraduates. *Lancet*, **i**, 731–733.

ROOK, A. (1959) Student suicides. *British Medical Journal*, **i**, 599–603.

SCHWARTZ, A. J. & REIFLER, C. B. (1980) Suicide among American college and university students from 1970–71 through 1975–76. *Journal of the American College Health Association*, **28**, 205–210.

SIEGEL, S. (1956) *Nonparametric Statistics for the Behavioural Sciences*. London: McGraw-Hill.

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Influence of the Day of the Week and the Weather on People Using a Telephone Support System

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The calls to a telephone counselling service at Louvain, during a 21-month period, from one telephone zone were analysed. Data were studied in relation to the type of weather, which was recorded every day. Calls made by women about health and interpersonal problems showed a relationship to the weather, while calls apparently motivated by masturbation intentions from male callers increased when the weather improved.

From ancient times, man has tended to link, mostly on the basis of subjective feeling, weather, season, and climate with health, mood, and behaviour (Licht, 1964), as indicated by many traditional proverbs and expressions (Pastor, 1934; Dufour, 1978). Weather is also an important factor in social life, illustrated in the importance attached to it in travel brochures and marketing.

However, there has been little scientific investigation into this matter, though a weather dependency or meteorological aetiology has been put forward for almost every disease (Tromp, 1980), and for some, a direct relationship with weather and climate can certainly be found. Few doubt the pathogenetic influence of long-term UV exposure on the skin (Roelandts and Degreef, 1980) or the influence of cold and foggy weather on symptoms of angina pectoris (Bullock & Hall, 1982; Feldman & Conti, 1982), but for many other diseases, research results are contradictory, and conclusive evidence is still missing. In psychiatry, though, a great deal of attention has been given to the possible influence of the weather and season on diseases: many reports are available on the seasonality of the incidence of depression, mania, suicide, and schizophrenia, but due to the numerous intervening and sometimes unknown variables, a convincing connecting theory has still to be drawn up (Hare *et al*, 1974; Symonds & Williams, 1976; Mawson & Smith, 1981; Meares *et al*, 1981; Hare, 1981; Parker & Walter, 1982; Shur, 1982; Rosenthal *et al* 1984; Watson *et al*, 1984).

By investigating the influence of the weather on the number and type of the calls to a telephone counselling service, we tried to investigate the possible influences of the weather on people with physical or psychosocial problems. We therefore analysed the calls to the service in Louvain, in relation to three types of weather.

The concept of telephone counselling service has existed for about 30 years in Europe. Though the original purpose was suicide prevention, it gradually developed into a more general counselling service for people with problems. In this context, a service was first set up in Belgium in 1959; there are now five in the Flemish part of the country, whereas the Walloon part has three. Our service (Tele-Onthaal) functions on the level of basic community health care. Its aim is to give telephone counselling and support, 24 hours a day; this is done by volunteers, supervised by a permanent staff. There are 100 members, on duty in shifts, who receive training, with follow-up, in giving prompt and accurate help to people with problems. For every call, the volunteer on duty completes a form, which gives a profile of the caller, including sex, age, marital status, and the type and reason for the call. A short summary of the conversation is made on the back. On this precoded form, the volunteer can check off one possibility for every item and up to two possibilities for problems dealt with during the conversation.

Method

This report is based on 23 000 calls, made to the telephone counselling service in Louvain during the period from 1 January 1981 to 30 September 1982. The fact that the volunteers on duty change five times a day removed any systematic bias in the completion of the forms. To ensure uniformity of weather conditions, only the 13 191 calls coming from the Louvain telephone zone were retained for study.

Following Poriau (unpublished theses 1972), we used the relative duration of sunshine as a parameter for the weather; he found that the sun, and particularly the number of hours of sunshine, is the most important factor in peoples' subjective appreciation of it. The relative duration of sunshine, i.e. the ratio between the real number of hours of sunshine and the potential hours that day (number of hours between sunrise and sunset) is independent of the season, and has the advantage that only one value for each day has to be taken into account. These observations were made at the Louvain Climatic Institute, and were valid within a radius of about 20 km, corresponding largely to the Louvain telephone zone.

The calls to the telephone counselling service were first studied according to the time of day and day of the week. Holidays were counted as Sundays, because they are subjectively felt to be the same as a Sunday. The unequal presence of the various days of the week in the experimental period forced us to make corrections, to transform the data into comparable material. This was done in two steps. Firstly, an average number of calls was calculated for each day of the week (first part of the formula below). This was then multiplied with the total number of days in the experimental period, and divided by the number of days in a week (second part of the formula). In this way, we calculated a number of calls (N′) for each day, as if each day were equally present in the experimental period.

$$\frac{\text{Number of calls on day X}}{\text{Frequency of day X}} \times \frac{\text{Total number of days (638)}}{\text{Number of days in a week}} = \text{N}'$$

Three types of weather were distinguished on basis of the relative duration of sunshine (RDS); when this was less than or equal to 10%, we designated the weather as 'bad' and called it Weather A. This occurred on 222 of the 638 days. When the RDS was equal to or greater than 60%, we designated the weather as 'beautiful', and called this type C; this occurred on 144 days. When the RDS was greater than 10% but less than 60%, the weather was designated as 'changeable', and was called type B, which was the case on 272 days. In the second part of the study the number and type of the calls were examined according to these types of weather. Corrections had to be made here in the same way and for the same reasons as for investigation of the calls by day of the week.

$$\frac{\text{Number of calls when type X}}{\text{Frequency of type X}} \times \frac{\text{Total number of days (638)}}{\text{Number of types (3)}} = \text{N}'$$

Finally, the calls were investigated according to the seasons and the months of the experimental period. Because the winter of 1981 and autumn of 1982 were not totally covered, they were excluded from the investigation by season. Corrections also had to be made because each season or month does not have the same number of days.

$$\frac{\text{Number of calls in season X}}{\text{Number of days in season X}} \times \frac{\text{Total number of days (549)}}{\text{Total number of seasons (6)}} = \text{N}'$$

$$\frac{\text{Number of calls in month X}}{\text{Number of days in month X}} \times \frac{\text{Total number of days (638)}}{\text{Total number of months (21)}} = \text{N}'$$

To determine the influence of the weather on the calls, a χ^2 test for goodness of fit was used: the expected frequencies were considered to be equal within one sex. Our Null Hypothesis (H_0) was that all spreading of the calls within one sex was based on coincidence. The limit to reject this hypothesis was set at $P<0.01$.

Results

In the first part of the study, it became clear that many more women than men call the service (1.6:1). In both sexes, an obvious increase in the number of calls is seen as the day goes on, with peak periods between 10 and 12 am, 2 and 4 pm, and late at night; there is a slack period in the early morning. All this is more striking in women's calls, but is statistically significant in both sexes (χ^2 male = 943.27; $P<0.001$ and χ^2 female = 1,735.04; $P<0.001$).

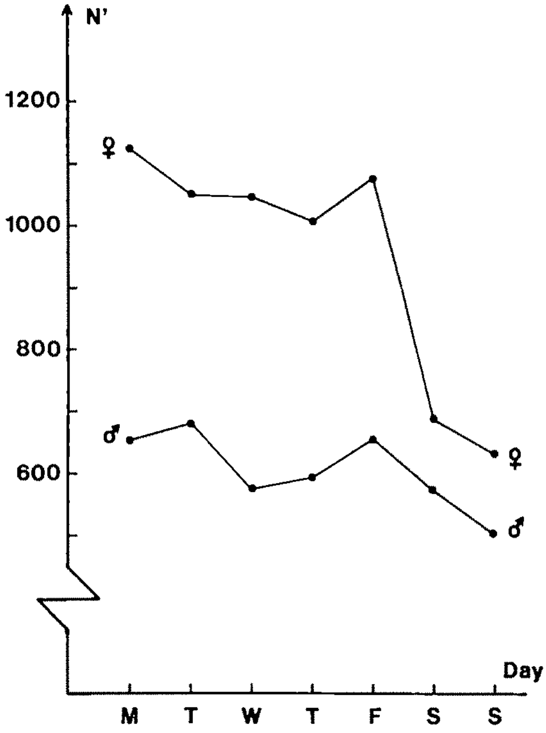

Fig. 1 Corrected number of calls (N′) to Tele-Onthaal according to the day of the week for male and female callers.

At weekends, there is a sudden decrease in the number of calls for both sexes (χ^2 male = 38.64; $P<0.001$ and χ^2 female = 183.43; $P<0.001$), which again is more striking in women's calls. On Monday, the number of calls increase again. The downward trend of the number of calls during the week, which could point to the need for airing problems accumulated during the weekend, was tested with the Spearman correlation test for trends within groups. This did not reach our criteria for significance (Spearman correlation for male −0.68; NS and for female −0.79; $P<0.05$).

The variation of the calls according to the three types of weather was statistically significant for women, with a strong decrease as the weather improved ($\chi^2 = 35.64$; $P<0.001$). Age-specific investigation here revealed no signs of weather dependency, but testing for marital status showed weather dependency. Divorced women and widows seem to call the

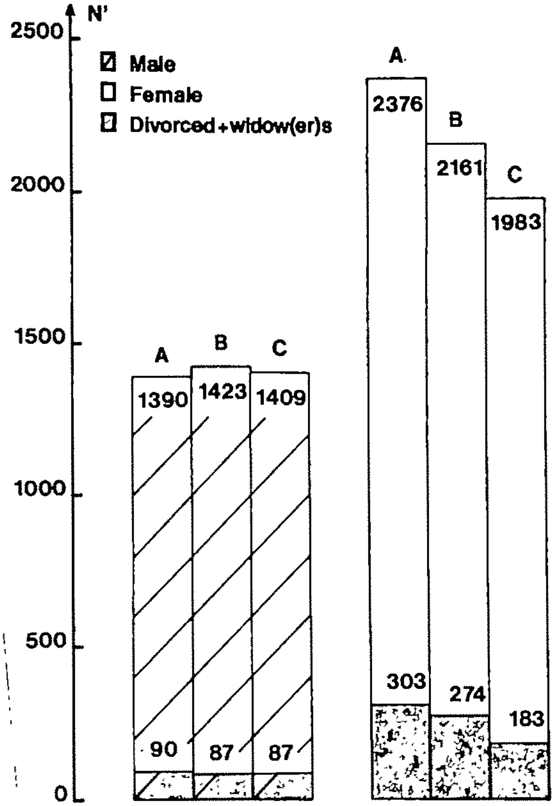

FIG. 2 Corrected number of calls (N') to Tele-Onthaal according to the type of weather for male and female callers. Subgroup widow(er)s and divorced. A = RDS ≤ 10%, B = RDS > 10% but < 60%, C = RDS ≥ 60%.

telephone service most when the weather is bad. This decreases when the weather is beautiful ($\chi^2 = 30.95$; $P < 0.001$). For divorced men and widowers, there was a similar tendency, although by no means statistically significant ($\chi^2 = 0.07$; NS).

Some callers are well known to the service; the frequency and pattern of their calls is entirely different from that of the other groups. This is mainly caused by the great increases in the number of calls concerning sexual problems when the weather improves and, in particular, calls from men whose intention is to masturbate while speaking to the telephone counsellor.

Calls concerning health problems increase significantly for women when the weather is bad ($\chi^2 = 27.15$; $P < 0.001$). In our investigation, this was not seen in men ($\chi^2 = 0.88$; NS). There is also an increase in calls from women complaining of relational problems with their partner when the weather gets worse ($\chi^2 = 22.27$; $P < 0.001$). For men, a similar tendency was found, although not statistically significant ($\chi^2 = 4.13$; NS).

In a third part of the study, we checked these results by investigating the calls by season and month, and this confirmed the results to a great extent. Calls concerning health and relational problems followed a seasonal pattern, with a significant increase in winter ($P < 0.001$). The remarkable increase in calls for masturbation from men during the summer corresponds with the results from the investigation of the calls for sexual problems according to the type of weather.

Discussion

The distribution of calls during the day and the week shows that people seeking help tend to telephone when they are sure to be undisturbed. In Belgium, and certainly in Louvain, women are the greatest part of the unemployed, and a large number of them are housewives; when husband and children are out, they thus have an opportunity to call undisturbed, and share their problems with the anonymous telephone counsellor. Many women lack possibilities for social contact and thus for airing their problems, partly due to the differing expectations, social obligations, and life styles of men and women. All this explains, at least in part, the fact that there is a greater number of calls from women, and that the increase in calls during peak periods is greater for women than for men.

Trenkle (1982) and Symonds & Williams (1976), among others, have suggested that women are more sensitive than men to weather, due to hormonal differences. However, even if this is true, other factors surely must be taken into account: many women remain indoors and postpone shopping, visits, and walks when the weather is bad. This can create loneliness, making a call to a telephone counselling service more probable. Women, furthermore, are often more sensitive to fashion than men, and their clothes are frequently less adapted to bad weather. Sun and warmth have become selling 'products' in many women's magazines, travel brochures, and fashion books. Therefore, women are more sensitive to weather parameters, in part due to the predominance in the media of an image of beautiful weather that influences them to live a certain style of life.

Loneliness is most felt when possibilities for contact are scarce, for instance because of bad weather. It is not surprising that it is at these times that divorced people and widow(er)s tend to call most to Tele-Onthaal.

The calls for sexual problems, most of them from well known male callers concerning masturbation, show a completely different pattern from that of other calls. The peak periods are seen when the weather is beautiful. Michael (1983) found the same tendency when he studied the variation in time of different forms of sexual violence in the United States.

It is not surprising that calls for health problems increase when the weather is bad. It is well known that the subjective complaints as well as the objective symptoms of many diseases increase in winter, or when the weather is bad.

Problems of relationships between partners seem to have the same tendency, again more striking in women's calls. People working out of home, who can bury themselves in their work or share their problems with colleagues, are likely to deal more easily with such problems.

The effect of bad weather can also partly be explained by territorial feelings. These become more prominent when one is obliged to stay indoors, sharing space with several people, so making the space smaller for each individual, and thus also restricting the possibilities for privacy.

The results of this study show that there is indeed an influence of the weather on calls to this telephone counselling service, particularly on calls from women concerning health and relationship problems. Though lack of opportunities for social contact or entertainment, and a greater concern with fashion may be relevant, this could indicate a greater sensitivity to the weather in women. In men, more of a weather influence was found in the calls relating to masturbation. It was found that women telephone Tele-Onthaal more than men, and that both sexes place importance on an undisturbed conversation with the anonymous telephone counsellor.

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References

BULLOCK, R. & HALL, R. (1982) The heart in the winter. *The Practitioner*, **226**, 465–466.

DUFOUR, L. (1978) *Calendriers et Croyances Populaires. Les Origines Magico-religieuses, les Dictons.* Paris: Librairie d'Amérique et d'Orient.

FELDMAN, R. L. & CONTI, C. R. (1982) Cold air exposure and angina pectoris. *American Heart Journal*, **104**, 173.

HARE, E. (1976) The season of birth of siblings of psychiatric patients. *British Journal of Psychiatry*, **129**, 49–54.

—— & MORAN, P. (1981) A relation between seasonal temperature and the birth rate of schizophrenic patients. *Acta Psychiatrica Scandinavica*, **63**, 396–405.

——, PRICE, J. & SLATER, E. (1974) Mental disorder and season of birth. A national sample compared with the general population. *British Journal of Psychiatry*, **124**, 81–86.

LICHT, S. (1964) *Medical Climatology. Physical Medicine Library VIII.* New Haven: Elisabeth Licht. 594–612.

MAWSON, D. & SMITH, A. (1981) Relative humidity and manic admission in the London area. *British Journal of Psychiatry*, **138**, 134–128.

MEARES, R., MENDELSOHN, F. & MILGROM-FRIEDMAN, J. (1981) A sex difference in the seasonal variation of suicide rate. A single cycle for men, two cycles for women. *British Journal of Psychiatry*, **138**, 321–325.

MICHAEL, R. P. & ZUMPE, D. (1983) Sexual violence in the United States and the role of season. *American Journal of Psychiatry*, **7**, 883–886.

PARKER, G. & WALTER, S. (1982) Seasonal variation in depressive disorders and suicidal death in New South Wales. *British Journal of Psychiatry*, **140**, 626–632.

PASTOR, E. (1934) *Deutsche Volksweisheit in Wetternregeln und Bauernsprüchen.* Berlin: Deutsche Landbuchhandlung.

ROELANDTS, R. & DEGREEF, H. (1980) Voor- en nadelen van zonnebaden. *Tijdschrift voor Geneeskunde*, **36**, 715–718.

ROSENTHAL, N. E., SACK, D. A., GILLIN, J. C., LEWY, A. J., GOODWIN, F. K., DAVENPORT, Y., MUELLER, P. S., NEWSOME, D. A. & WEHR, T. A. (1984) Seasonal affective disorders. *Archives of General Psychiatry*, **41**, 72–80.

SHUR, E. (1982) Season of birth in high and low genetic risk schizophrenics. *British Journal of Psychiatry*, **140**, 410–415.

SYMONDS, R. L. & WILLIAMS, P. (1976) Seasonal variation in the incidence of mania. *British Journal of Psychiatry*, **129**, 45–48.

TRENKLE, H. (1982) Wetter und Mensch. *Medizinische Welt*, **33**, 1285–1287.

TROMP, S. (1980) *Biometeorology. The Impact of the Weather and Climate on Humans and Their Environment.* London: Heyden.

WATSON, C. G., KUCALA, T., TILLESKJOR, C. & JACOBS, L. (1984) Schizophrenic birth seasonality in relation to the incidence of infectious diseases and temperature extremes. *Archives of General Psychiatry*, **41**, 85–90.

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Measuring the Rising Tide

How Many Dementia Cases will there be by 2001?

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Numerous surveys have attempted to estimate the prevalence of dementia. Despite difficulties of measurement, evidence suggests figures may be lower than is generally realised. Factors which are likely to affect prevalence rates are discussed. Forthcoming changes in the demography of old age indicate that increases in the prevalence of dementia may have been seriously underestimated.

The rapid increase in the number of elderly people, especially the very elderly, is widely recognised, and is likely to continue into the next century. By then there will be almost nine million people of 65 years of age or more in Britain, nearly half of whom will be over 75. Over a million will be at least 85 years old (OPCS, 1985).

This means almost inevitably a growth in the numbers of mentally ill old people, the so-called 'Rising Tide' (HAS, 1982). Information on the extent of mental illness, especially dementia, among the elderly is becoming available on an increasing scale, but as yet much remains scattered and relatively little-known. This paper attempts to bring together this literature, and to apply it to estimate the likely increase in dementia sufferers by the end of the century.

Problems in measuring the prevalence of dementia

Counting the number of dementia sufferers is not easy for the following reasons:

(i) There is no universally agreed criterion for what constitutes 'a case' (Davis, 1984); there are particular difficulties at the 'mild' end of the spectrum (Henderson, 1983; Cooper & Bickel, 1984; Henderson & Huppert, 1984). Impairment of intellectual function, especially memory, is a universally agreed feature of dementia, relatively easy to measure. Deterioration of behaviour is often also present but creates greater difficulty of measurement. Gilleard (1984) has an extended discussion of the clinical syndrome of dementia. Epidemiological surveys have used a range of techniques for case identification, including psychiatric interviews and cognitive tests. Gurland (1980) and Gardner (1984) summarise some of the more popular cognitive tests. Kay *et al* (1985) report on the use of a variety of tests and cognitive procedures. Jorm and Henderson (1985) suggest improvements in the use of one particular diagnostic tool, the DSM-III.

(ii) Demographic changes influence prevalence, in view of the age-related nature of dementia. It is often difficult to estimate precisely the denominator for any particular investigation.

(iii) Few studies have been sufficiently robust in their methodology to provide firm answers. Much dementia remains outside the service net (Williamson *et al* 1964; Parsons, 1965; Helgason, 1977), making any study drawing on service usage for its sample of limited value. Studies dealing only with populations known to particular services (e.g. Adolfsson *et al* 1981; Molsa *et al* 1982) are excluded from the present discussion. Services themselves are subject to enormous local variation. Reliable large-scale surveys are expensive and laborious to conduct, and therefore rare. Most existing studies have used samples numbered in hundreds rather than thousands; the number of identified dementia sufferers is therefore small and subject to experimental error. High refusal rates, as found in some studies, are another source of potential error.

(iv) A wide range of sociocultural factors may influence the number of dementia sufferers. The degree of isolation of elderly people from their relatives, for example, is influenced by changes in family size, family cohesiveness, housing styles etc. (Blazer, 1980) and this may be increasing in Britain (Roth & Mountjoy, 1980). Changes in nutrition, level of education, and other sociocultural factors may also influence the extent of dementia.

(v) In our present state of knowledge only an approximate answer can be given to the question of how long dementia patients survive, although one recent study (Diesfeldt, 1986) reports an average duration of 7.2 years for an institutionalised sample.

Findings in published studies on the prevalence of dementia in old age

Many previous writers on the subject (OHE, 1979; Roth & Mountjoy, 1980; Arie & Jolley, 1982; McCarthy, 1982; Norman, 1982; Pitt, 1982; Davis, 1984; Mahendra, 1984; Miller, 1984; Wilkin *et al*, 1984) have contented themselves with quoting from the work of Kay *et al* (1964, 1970) done in Newcastle in the early 1960s, as the most recent, or the most valued, or indeed the only work on the subject of the prevalence of dementia. However, a score of studies have been published subsequently, many with very different findings. Attempts to bring these studies together have been no more than partial: Kay & Bergmann (1980), for instance, list 17 studies, but only three of these have been published since 1970. (See also Mortimer *et al*, 1981; Gurland & Cross, 1982; Henderson, 1983; Mortimer, 1983; Henderson & Kay, 1984; Eastwood & Corbin, 1985).

The UK studies are as follows:

Kay *et al* (1970). Interviews with representative samples of people over 65 living at home in Newcastle-on-Tyne in 1960 and 1964. ($n = 758$); 47 cases (6.2%) were reckoned to have a firm diagnosis of Chronic Brain Syndrome, in nearly every case due to senile or arteriosclerotic dementia.

Gruer (1975). A 1-in-20 sample from Scottish borders, covering whole population, identified from executive council lists and hospital populations. Stratified for urban/rural, male/female and four age-groups. 762 out of 835 interviewed (success rate of 91.3%). Fieldwork 1971/2, 6.9% rated as definite or assumed dementia, another 1.4% possibles; 41% had psychological symptoms, the majority of which were not recorded by their GP.

Broe *et al* (1976). Sample of 808 elderly people living at home in northern Glasgow and Kilsyth. Three surveys amalgamated: two stratified by age to give equal numbers under and over 75. Refusal rate 20–30%. Dates of fieldwork not given. Criteria for senile dementia closely followed Slater & Roth (1969). Interviewed at home by a doctor; most doubtful cases examined by a neurologist: 1.6% of those aged 65–74, and 7.2% of those over 75, rated as suffering from severe or moderate dementia.

Gilmore (1977). Study of 300 people of 65-plus, living at home. Three (1%) rated as suffering from severe dementia, defined as "incapable of coherent and sustained speech, of indicating their needs or of responding to commands". Another 13 (4.3%) rated moderate: "some recent loss of memory, obvious difficulty in concentration, fluctuating disorientation for time, place and event". Another 12 (4%) rated as mild dementia, with variability in concentration, resembling a state of preoccupation, in combination with some recent memory loss of trivial information with occasional "personal and geographical" orientation lapses.

Bergmann *et al* (1979). Sample of 800 on lists of two GPs in Newcastle. Diagnosis of dementia based on (i) use of psychogeriatric assessment schedule to diagnose probable organic psychiatric disorder and (ii) interview with psychiatrist using Geriatric Mental State schedule (Copeland *et al*, 1976). Another psychiatrist arbitrated in disputed cases: 25 subjects reckoned to be suffering from dementia.

Gurland *et al* (1983). Sample of 489 people aged 65 or more on GP lists and living at home in London were approached: 93 could not be contacted or refused to be interviewed; 396 were interviewed—a response rate of 81%. Assessment by Comprehensive Assessment and Referral Evaluation (CARE), a semi-structured interview developed by the team, and administered by a psychiatrist with a special interest in psychogeriatrics. A 22-item scale of dementia was constructed, composed of errors in memory, orientation and learning; in reporting age, date and significant events; in learning the interviewer's name; and in recalling the names of famous persons. A score of 6 or more on this scale was considered to be particularly likely to be indicative of a severe clinical disorder. Ten subjects (2.5%), all female, eight aged over 80, reached this level. Similar techniques have produced higher rates in New York (in the same report) and in Hobart (Kay *et al* 1985).

Clarke *et al* (1984, 1986). The entire population (1203 people) over the age of 75 living in and around Melton Mowbray, Leicestershire, were approached in 1981: 95% were interviewed. Refusals were not thought to have major health problems. Dementia was defined as a score of 7 or less on the Information Orientation Sub-test of the Cognitive Assessment Scale taken from Pattie and Gilleard (1979); 4.5% came into this category and another 3.5% were unclassifiable.

Maule *et al* (1984). Random sample of 487 people aged 62–90 drawn from GP's lists (i.e. including those who had been in long-stay institutions for less than 2 years) in North Edinburgh interviewed by a psychiatrist in 1968–1969. Psychiatric assessment based on answers to a semi-structured questionnaire and from the behaviour, mood and attitude of the subject at the time. Chronic Brain Syndrome defined as the permanent and irreversible decline of all mental abilities; moderate or severe cases were those in which clinical dementia was present and included senile dementia, arteriosclerotic dementia and other cerebral organic syndromes. Only three cases of severe CBS were recorded (0.6%) but in 14 cases (2.9%) the impairment was classed as clinical dementia and was severe enough to interfere with self-care.

Table I, drawing on the above studies and those based on surveys conducted outside the United Kingdom, shows an overall rate for dementia (of all levels of severity) which ranges from 24.6% to 2.5%. Despite some interesting consistencies (e.g. the clustering of the four Scottish studies) the range of estimates make this information of little use; it is more practical to concentrate only on those surveys which produce a more restricted definition of dementia.

TABLE I
Studies of the overall prevalence of dementia

Author	*Date of publication*	*Country*	*Age range*	*Sample size*	*Overall rate (%)*
Sternberg & Gawrilova	1978	USSR	60+	1020	24.6
Weissman *et al*	1985	USA	65+	2588	16.1
T. Helgason	1973	Iceland	74–76	2462	11.9
Cooper & Sosna	1983	W. Germany	65+	519	11.4
Gilmore	1977	Scotland	65+	300	9.3
Kay *et al*	1970	England	65+	758	8.8
Maule *et al*	1984	Scotland	62–90	487	8.6
Gruer	1975	Scotland	65+	762	8.3
Broe *et al*	1976	Scotland	65+	808	8.1
Campbell *et al*	1983	New Zealand	65+	559	7.7
Weyerer	1983	W. Germany	65+	295	7.5
Gurland *et al*	1983	USA	65+	445	5.8
Shibayama *et al*	1986	Japan	65+	3106	5.8
Nilsson	1983	Sweden	70	404	5.2
Bollerup	1975	Denmark	70	619	5.0
Shinfuku *et al*	1984	Japan	65+	9218	4.5
Clarke *et al*	1986	England	75+	1203	4.5
Persson	1980	Sweden	70	392	4.3
Bergmann *et al*	1979	England	65+	800	3.1
Gurland *et al*	1983	England	65+	396	2.5

TABLE II
Studies of the prevalence of severe (and severe + moderate) dementia

Author	*Date of publication*	*Location*	*Age range*	*Sample size*	*Population sampled*	*% Severe dementia*	*% Severe + moderate dementia*
Weyerer	1983	Upper Bavaria	65+	295	Whole community	7.5	—
Kay *et al*	1964	Newcastle	65+	758	At home	6.2	—
Cooper & Sosna	1983	Mannheim	65+	519	Areas of city	6.0	—
Gurland *et al*	1983	New York	65+	445	At home	5.8	—
Shinfuku *et al*	1984	Tokyo	65+	9218	Various samples	4.5	—
Clarke *et al*	1984	Melton Mowbray	75+	1203	Whole community	4.5	—
Broe *et al*	1976	Glasgow & Kilsyth	65+	808	At home	3.8	—
T. Helgason	1973	Iceland	74–76	2642	Whole community	3.6	—
Sternberg & Gawrilova	1978	USSR	60+	1020	N/A	3.6	—
Weissman *et al*	1985	USA	65+	2588	At home	3.4	—
Bergmann *et al*	1979	Newcastle	65+	800	GP lists	3.1	—
Gurland *et al*	1983	London	65+	396	At home	2.5	—
Campbell *et al*	1983	Gisborne	65+	559	Whole community	2.4	7.7
Bollerup	1975	Copenhagen	70	619	Areas of city	1.6	—
Persson	1980	Gothenberg	70	392	Whole community	1.5	—
Gilmore	1977	Scotland	65+	300	N/A	1.0	5.3
Nilsson	1983	Gothenberg	70	404	Whole community	1.0	—
Maule *et al*	1984	Edinburgh	62–90	487	GP lists	0.6	2.9

Table II includes only those surveys which include a measure of severe dementia, with or without a further measure of moderate dementia; or which include only one measure of dementia. It also incorporates the sampling technique used. Weyerer's study in Bavaria is the only one to exceed the figure of Kay *et al* of 6.2% for severe dementia alone. Eight of the studies produce a figure of less than half of that found by Kay *et al*. The recent study in Hobart (Kay *et al* 1985) also reports low figures for severe dementia among its sample of over-70's, using a variety of different measuring techniques.

Studies of Incidence

Very few studies of incidence have been carried out due to the considerable practical problems of such research.

Mortimer *et al* (1981) quote the Newcastle studies and other work, suggesting the annual incidence figure for senile and arteriosclerotic dementia may be of the order of 1.4% of over-65s. Kay & Bergmann (1980) quote four other studies which show very much lower rates for chronic brain syndrome. This may reflect the technique of enquiry more than anything else: three of the four low-incidence studies are derived from case registers, whereas the Newcastle study is based on interviews.

One Swedish study is particularly notable. Hagnell *et al* (1981) studied the total population of Lundby (2550 people) between 1947 and 1972, finding that over that time 'age psychosis' appeared to be decreasing, for elderly men and for women over the age of 80. This the authors attribute to the improved security and material circumstances of elderly people in Sweden. Bergmann & Jacoby (1983) suggest the causes might include improved physical health, better nutrition and the control of high blood pressure by hypotensive drugs, reducing the incidence of stroke.

Meanwhile, estimates based on studies in the 1960s have suggested that about one in five people will end their lives suffering from dementia (Mortimer *et al*, 1981); almost 30% of 85-year-olds will dement (Sluss *et al*, 1981). Research designed to test comprehensively ideas on the onset and incidence of dementia would clearly involve frequent and prolonged contact with a large sample, and there appears little prospect of such work ever being conducted..

What does the diagnosis of dementia in old age mean?

The range of techniques used to establish the presence of dementia in the quoted prevalence studies is clearly very considerable, and a contributory factor to the great range of answers that they produce.

Gardner (1984) provides a guide to the clinical assessment of dementia. Henderson (1983) gives the Royal College of Physicians' 1980 definition, and points out that the rubric of dementia applies to 100 different diagnoses in which higher brain function is impaired; only two principal categories (Alzheimer type and multi-infarct dementia) are recognised in the elderly. Pitt (1982) considers there may be a third category of dementia sufferers, whose problem is essentially one of forgetfulness. Roth (1985) has an extended discussion of the problems involved in diagnosing and classifying dementia, and Kay *et al* (1985) describe the use of a variety of techniques on one relatively small sample. They note that much of the construction of the testing tools rests upon clinical observation, and feel that their validity is still to be investigated.

Cooper & Bickel (1984) quote four studies where the rate of confirmation of the diagnosis varies from 74% to 90%. Hare (1978), on the basis of a study of 200 admissions to a psychogeriatric unit, found that severe memory loss was not equivalent to dementia: too much emphasis on memory loss led to overdiagnosis. She considered that psychological tests based on parietal signs were good predictors. Other studies quoted by Gurland & Cross (1982) suggest that within psychiatric settings, dementia may be overdiagnosed.

The problem of diagnosis of mild dementia is especially great. It is probable that persons with mild dementia constitute a heterogeneous group: those with low IQ, those with early dementia who subsequently deteriorate, and those whose dementia remains a a mild level (Kay & Bergmann, 1980).

Intervention can make some impact on the level of dementia: mental stimulation has been shown to improve intellectual performance, but for the improvement to be maintained, the stimulation must be consistently applied (Charatan, 1984).

Factors Influencing Prevalence

Sampling technique

Most of the UK studies have used either GP lists or surveys of people living at home to gather their sample. The first technique excludes only those people in long-stay hospitals; the second those in all kinds of institutions. Both approaches produce a wide scatter of results.

Studies using GP lists include Clarke *et al* (1986) who report 4.5% of over 75s; Bergmann *et al* (1979), who give an undifferentiated overall figure of 3.1%, and Maule *et al* (1984), who give 8.6% for overall prevalence, but 0.6% for severe dementia only.

Five surveys (Kay *et al*, 1970; Broe *et al*, 1976; Gilmore, 1977; Gurland *et al*, 1983; Clarke *et al*, 1984) are based on samples of people living at home. Their results range from 1.6% (Clarke *et al*, 1984) to 9.3% (Gilmore, 1977) for overall prevalence, and from 1.0% (Gilmore, 1977) to 6.2% (Kay *et al*, 1970) for severe dementia alone.

Age

There is clear evidence from numerous studies that dementia is age-related (Kay *et al*, 1970; Helgason, 1977; Campbell *et al*, 1983; Henderson, 1983; Maule *et al*, 1984), although Henderson & Huppert (1984) point out that in some earlier studies prevalence falls abruptly in the over-85s. Whether or not mild dementias proceed to more severe conditions is unclear. Mortimer (1983), also on the basis of early studies, considers that the incidence rate may level off at around age 75; evidence from the oldest age-groups is too slender to judge if the trend continues. He quotes autopsy studies which suggest that over the age of 80 there may be a reduced risk of developing the brain lesions characteristic of Alzheimer's Disease.

Sex

There is considerable evidence (Weyerer, 1983; Cooper, 1984; Maule *et al*, 1984) that even controlling for the greater longevity of women, more elderly women become mentally ill than elderly men.

Dementia alone is less clear-cut. Some evidence (Broe *et al* 1976; Helgason 1977; Campbell *et al* 1983; Bergmann 1985) shows little or no difference between the sexes. Other surveys (Gurland *et al* 1983; Clarke *et al* 1984, 1986; Maule *et al* 1984; Weissman *et al* 1985) find more dementia among females. However, Scandinavian investigators (Bollerup, 1975; Persson, 1980) find an excess of males. Perhaps their evidence is influenced by the effects of alcoholism, which so dominates the psychiatric scene in those countries.

Social class

Cooper (1984) notes an inverse association between mental disorder and physical disability with occupational class among elderly people. The excess of mental illness is accounted for solely by organic conditions. The rate of mental illness was especially high among people living in poor material conditions.

Very little evidence is available specifically on dementia. Kay *et al* (1964, 1970) found no association between dementia and social class, but few other writers have considered the subject. Gurland (1981) and Weissman *et al* (1985) found highest rates among those with least education. The difficulty of diagnosing accurately dementia among those of low intelligence or poor education has been noted (Gurland, 1981; Henderson, 1983; Henderson & Huppert, 1984). Murphy (1982) notes more major social difficulties, poor physical health and depression among working-class than middle-class elderly people in her East London sample.

Location

Most surveys have been held in urban locations. None has been held with an exclusively rural sample; three (Gruer, 1975; Weyerer, 1983; Clarke *et al*, 1984, 1986) have taken place in areas comprising a mixture of small town and rural areas, and these have produced very different findings. Gurland *et al* (1983) produce very different rates for New York and London, using identical methodologies in the two cities. Other research (e.g. Srole *et al* 1961) has found a high rate of mental disorders among New Yorkers generally.

How long do dementia patients live?

Dementia appears to be associated with premature death, although the relationship is not clear-cut. Significantly more of the samples of Kay *et al* (1970) and Maule *et al* (1984) had died within 4 or 5 years of the initial diagnosis. The more severe cases in the latter sample were all dead within 7 years, although the milder cases had a better record of survival. Gurland & Cross (1982), in contrast, quote other published studies which show that persons with mild dementia have an increased rate of mortality. A study of elderly twins found survival associated with the preservation of competent performance in completing psychological tests (Jarvik & Falek, 1963). Several studies (Larsson *et al*, 1963; Heston *et al*, 1981; Whitehead, 1981; Henderson & Kay, 1984) show increased mortality is associated with early onset of dementia, but not with onset over 80. In a review of longitudinal studies, Christie (1985) found that survival time among dementia patients appears to be lengthening. Other research yields little in the way of firm conclusions.

Studies of survival, using contact with services as a measure of onset, are of course notoriously unsuitable for estimating survival rates among elderly people with dementia. They may reflect nothing more than a service's efficiency in tracking down and preserving patients, and be misleading over the question of the influence of the condition *per se*. Gruenberg (1978) found that for every person over 65 in a Mental Hospital in New York State, there were 4.5 people of the same age who were at least as mentally disabled but who were not in hospital. The ratio increased with age: some severely mentally disabled old people probably die without ever being hospitalised.

Gilleard (1984) discusses the question of the onset of the condition at some length, pointing out that the criteria laid down by researchers to determine onset are often vague and rely on relatives' recall. Levels of disagreement over 2 years are not uncommon. He considers the average survival time in hospital is about 2 years for dementia patients; he finds no difference in mortality rates between demented elderly people at home and in institutions, concluding:

> . . . the expected survival of elderly individuals developing dementia does not greatly differ from that of elderly individuals not developing dementia, unless the onset of mental decline occurs before the age of 75. It is probable that supporters who care for an elderly relative beginning to show signs of mental deterioration may expect, therefore, six to eight years of coping, with ever-increasing problems. Such extended caregiving may involve a gradually increasing burden for family members that may extend beyond their limits to maintain care over such a prolonged period (1984).

How many dementia cases?

The study by Kay *et al* (1970) suggests a figure of 6.2% of all over-65s for the prevalence of dementia. However, more recent work indicates this figure may be too high. Mortimer *et al* (1981) propose a figure of 4.15%, based on the median figure for eight surveys. However, the most recent research suggests that this figure may itself be too high. Three recent UK studies which apply the most rigorous criteria in identifying 'a case' (Bergmann *et al*, 1979; Gurland *et al*, 1983; Maule *et al*, 1984) have very similar rates if the 'clinical dementia' found by Maule *et al* is added to 'clinical brain syndrome'. The mean rate for dementia for these studies is 2.83% and for their summed samples is 2.91% (see Table II).

Applying these flat rates to increases in national population, drawing on mid-year estimates for 1983 and the population projection for 2001 (OPCS, 1985), gives an increase of 4.8% of cases over that period.

However, the changing age structure of the elderly population means that a flat-rate estimate for dementia based on the number of over-65s is of little use. Age- and sex-stratified figures derived from Kay *et al* (1970) and two other studies in the 1960s have been calculated (Kay & Bergmann, 1980). Applied to national figures, these show a very similar increase (4.7%) to the flat-rate figures.

In view of the lower figures suggested by more recent research, a simple rule-of-thumb is suggested: 1% of 65–74s, 10% of 75 and over. Applying these rates yields lower figures than those of Kay & Bergmann, but a much larger increase (17.2%) between 1983 and 2001. In local areas containing an above-average number of elderly people the increase will be even larger: population projections for Bath Health District, for example, indicate an increase of the order of 33.6%, and an even larger increase may be calculated for health districts already containing a very large proportion of elderly people.

References

Adolfsson, R. (1981) Prevalence of dementia disorders in institutionalised Swedish old people. *Acta Psychiatrica Scandinavica*, **63**, 225–244.

Arie, T. & Jolley, D. (1982) Making services work: Organisation and style of psychogeriatric services. In *The Psychiatry of Late Life*, (eds R. Levy & F. Post). Oxford: Blackwell.

Bergmann, K. (1985) Epidemiological aspects of dementia and considerations in planning services. *Danish Medical Bulletin*, **32** (Suppl. 1), 84–91.

—— & Jacoby, R. (1983) The limitations and possibilities of community care for the elderly demented, in D.H.S.S., *Elderly People in The Community: Their Service Needs* London: HMSO.

——, Proctor, S. & Prudham, D. (1979) Symptom profiles in hospital and community resident elderly persons with dementia. In Bayer-Symposium 7, *Brain Function in Old Age*, Heidelberg: Springer-Verlag.

Blazer, D. G. (1980) The epidemiology of mental illness in late life. In *Handbook of Geriatric Psychiatry*, (eds E. W. Busse & D. G. Blazer). New York: Van Nostrand Rheinhold.

Bollerup, T. R. (1975) Prevalence of mental illness among 70-year-olds domiciled in nine Copenhagen suburbs. *Acta Psychiatrica Scandinavica*, **51**, 327–339.

Broe, G. A., Akhtar, A. J., Andrews, G. R., Caird, F. I., Gilmore, A. J. J. & McLennan, W. J. (1976) Neurological diagnosis in the elderly at home. *Journal of Neurology, Neurosurgery & Psychiatry*, **39**, 362–366.

Campbell, A. J., McCosh, L. M., Reinken, J. & Allan, B. C. (1983) Dementia in old age and the need for services. *Age & Ageing*, **12**, 11–16.

Charatan, F. B. (1984) Mental stimulation and deprivation as risk factors in senility. In *Risk Factors in Senility*, (ed. H. Rothschild). New York: Oxford University Press.

Christie, A. B. (1985) Survival in dementia: a review. In *Recent Advances in Psychogeriatrics 1* (ed. T. Arie) Edinburgh: Churchill Livingstone.

Clarke, M., Clarke, S., Odell, A. & Jagger, C. (1984) The elderly at home: Health and social status. *Health Trends*, **16**, 3–7.

——, Lowry, R. & Clarke, S. (1986) Cognitive impairment in the elderly: a community survey. *Age & Ageing*, **15**, 278–284.

Cooper, B. (1984) Home & away: The disposition of mentally ill old people in an urban population. *Social Psychiatry*, **19**, 187–196.

—— & Bickel, H. (1984) Population screening and the early detection of dementing disorders in old age: A review. *Psychological Medicine*, **14**, 81–95.

—— & Sosna, U. (1983) Psychische Erkrankung in der Alteberolberung, Eine epidemiologische feltstudie in Mannheim. *Nervenarzt*, **54**, 239–249.

Copeland, J. R. M., Kelleher, M. J., Kellett, J. M., Gourlay, A. J., Gurland, B. J., Fleiss, J. L. & Sharpe, L. (1976) A semi-structured clincal interview for the assessment of diagnosis and mental state in the elderly: The Geriatric Mental State Schedule. I. Development and reliability. *Psychological Medicine*, **6**, 439–449.

Davis, D. R. (1984) *Introduction to Psychopathology*, 4th edn. Oxford: Oxford University Press.

Diesfeldt, H. F. A. (1986) Duration of survival in dementia. *Acta Psychiatrica Scandinavica*, **73**, 366–371.

Eastwood, P. & Corbin, S. (1985) Epidemiology of mental disorders in old age. In *Recent Advances in Psychogeriatrics 1* (ed. T. Arie) Edinburgh: Churchill Livingstone.

Gardner, A. (1984) The assessment of dementia. In *Current Themes in Psychiatry*, Vol. 3. (eds R. N. Gaind *et al*) London: Spectrum.

Gilleard, C. J. (1984) *Living With Dementia*, London: Croom Helm.

Gilmore, A. J. J. (1977) Brain failure at home. *Age & Ageing*, 6 (suppl.), 56–60.

Gruenberg, E. M. (1978) Epidemiology of senile dementia. In *Neurological Epidemiology: Principles and Clinical Applications*, (ed. B. S. Schoenberg). New York: Raven Press.

Gruer, R. (1975) *Needs of the Elderly in the Scottish Borders*, Edinburgh: Scottish Home & Health Department.

Gurland, B. (1980) The assessment of the mental status of older adults. In *Handbook of Mental Health & Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs: Prentice-Hall.

—— (1981) The borderlands of dementia, In (eds N. Miller and G. Cohen) Clinical Aspects of Alzheimer's Disease. *Ageing*, 15, 61–80.

—— & Cross, P. S. (1982) The epidemiology of psychopathology in old age, in Psychiatric Clinics of North America. *Aging*, 5 (1), 11–26.

——, Copeland, J., Kuriansky, J., Kelleher, M., Sharpe, L. & Dean, L. L. (1983) *The Mind and Mood of Aging: Mental Health Problems of the Community Elderly in New York and London*, London: Croom Helm.

Hagnell, O., Lanke, J., Horsman, B. & Ojesjo, L. (1981) Does the incidence of age psychosis increase? *Neuropsychobiology*, 7, 201–211.

Hare, M. (1978) Clinical check-list for diagnosis of dementia. *British Medical Journal*, 266–268.

Health Advisory Service (1982) *The Rising Tide: Developing Services for Mental Illness in Old Age*, Sutherland House, Sutton, Surrey, SM2 5AN.

Helgason, L. (1977) Psychiatric services and mental illness in Iceland. *Acta Psychiatrica Scandinavica*, (suppl.) 268.

Helgason, T. (1973) Epidemiology of mental disorder in Iceland: A geriatric follow-up. *Excerpta Medica International Congress Series*, 274, 350–357.

Henderson, A. S. (1983) The coming epidemic of dementia, *Australian and New Zealand Journal of Psychiatry*, 17, 117–127.

—— & Huppert, F. A. (1984) The problem of mild dementia. *Psychological Medicine*, 14, 5–11.

—— & Kay, D. W. K. (1984) The Epidemiology of Mental Disorders in old age. In *Handbook of Studies on Psychiatry & Old Age*, (eds D. W. Kay & G. D. Burrows). Amsterdam: Elsevier.

Heston, L. L., Mastri, A. R., Anderson, E. & White, J. (1981). Dementia of the Alzheimer Type: Clinical genetics, natural history and associated conditions. *Archives of General Psychiatry*, 38, 1085–1090.

Jarvik, L. F. & Falek, A. (1963) Intellectual stability and survival in the aged. *Journal of Gerontology*, 18, 173–179.

Jorm, A. F. & Henderson, A. S. (1985) Possible improvements to the diagnostic criteria for dementia in DSM-III. *British Journal of Psychiatry*, 147, 394–399.

Kay, D. W. K. & Bergmann, K. (1980) Epidemiology of mental disorders among the aged in the community. In *Handbook of Mental Health and Ageing*, (eds J. E. Birren & R. B. Sloane). Englewood Cliffs: Prentice Hall.

——, Beamship, P. & Roth, M. (1964) Old age mental disorders in Newcastle on Tyne. Part I, a Study of prevalence. Part II. A study of possible social and medical causes. *British Journal of Psychiatry*, 110, 146–158; 668–682.

——, Foster, E. M., McKechnie, A. A. & Roth, M. (1970) Mental illness and hospital usage in the elderly: A random sample followed up. *Comprehensive Psychiatry*, 11, 26–35.

——, Henderson, A. S., Scott, R., Wilson, J., Rickwood, D. & Grayson, D. A. (1985) Dementia and depression among elderly living in the Hobart community: the effect of diagnostic criteria on the prevalence rates. *Psychological Medicine*, 15, 771–788.

Larsson, T., Sjogren, T. & Jacobson, G. (1963) Senile dementia: A clinical, socio-medical and genetic study. *Acta Psychiatrica Scandinavica*, (suppl.) 167, 1–259.

Mahendra, B. (1984) *Dementia: A Survey of the Syndrome of Dementia*, Lancaster: MTP Press.

Maule, M., Milne, J. S. & Williamson, J. (1984) Mental illness and physical health in older people. *Age & Ageing*, 13, 349–356.

McCarthy, M. (1982) *Epidemiology and Policies for Health Planning*, London: King's Fund.

Miller, E. (1984) Epidemiology. In *Dementia: A Clinical Approach*, (ed. J. M. S. Pearce). Oxford: Blackwell.

Molsa, P. K., Martila, A. R. J. & Rinne, U. K. (1982) Epidemiology of dementia in a Finnish population. *Acta Neurologica Scandinavica*, 65, 541–552.

Mortimer, J. A. (1983) Alzheimer's disease and senile dementia: Prevalence and incidence. In *Alzheimer's Disease*, (ed. B. Reisberg). Glencoe: Free Press.

——, Schuman, L. M. & French, L. R. (1981) Epidemiology of dementing illness. In *The Epidemiology of Dementia*, (eds J. A. Mortimer & L. M. Schuman). New York: Oxford University Press.

Murphy, E. (1982) The social origins of depression in old age. *British Journal of Psychiatry*, 141, 135–142.

Nilsson, L. V. (1983) Prevalence of mental disorders in a 70-year-old urban sample: A cohort comparison. *Journal of Clinical & Experimental Gerontology*, 5 (2), 101–120.

Norman, A. (1982) *Mental Illness in Old Age: Meeting the Challenge*, London: Centre for Policy on Ageing.

Office of Health Economics (1979) *Dementia in Old Age*. London: OHE.

Office of Population Censuses (1985) *Social Trends* 15.

Parsons, P. L. (1965) Mental health of Swansea's old folk. *British Journal of Preventive & Social Medicine*, 19, 43.

Pattie, A. H. & Gilleard, C. J. (1979) *Manual of the Clifton Assessment Procedures for the Elderly*, Kent: Hodder & Stoughton.

Persson, G. (1980) Prevalence of mental disorders in a 70-year-old urban population. *Acta Psychiatrica Scandinavica*, 62, 119–139.

Pitt, B. (1982) *Psychogeriatrics: An Introduction to the Psychiatry of Old Age*, 2nd edn. Edinburgh: Churchill Livingstone.

Roth, M. (1985) Some strategies for tackling the problems of senile dementia and related disorders within the next decade. *Danish Medical Bulletin*, 32 (Suppl 1), 92–111.

—— & Mountjoy, C. Q. (1980) Mental Health Services for the Elderly Living in the Community: a UK perspective. *International Journal of Mental Health*, 8, 6–35.

Shibayama, H., Kasahara, Y., Kobayashi, H. *et al* (1986) Prevalence of dementia in a Japanese elderly population. *Acta Psychiatrica Scandinavica*, 74, 144–151.

Shinfuku, N., Sugita, T. & Shingai, N. (1984) Presenile and senile dementia in Japan. *Asian Medical Journal* 27, 393–399.

Slater, E. & Roth, M. (1969) *Clinical Psychiatry*, 3rd ed. London: Balliere, Tindall & Cassell.

Sluss, T. K., Gruenberg, E. M. & Kramer, M. (1981) The use of longitudinal studies in the investigation of risk factors for senile dementia—Alzheimer type. In *The Epidemiology of Dementia*, (eds J. A. Mortimer & L. M. Schuman). New York: Oxford University Press.

Srole, L., Langner, T. S., Michael, S. T., Opler, M. K. & Rennie, T. A. C. (1961) *Mental Health in the Metropolis*, New York: McGraw-Hill.

Sternberg, E. & Gawrilova, S. (1978) Uber klinisch-epidemiologische untersuchungen in der Sowjetischen Altenspsychiatrie. *Nervenarzt*, 49, 347–353.

WEISSMAN, M. M., MYERS, J. K., TISCHLER, G. L., HOLZER, C. E. III, LEAF, P. J., ORVASCHEL, H. & BRODY, J. A. (1985) Psychiatric disorders (DSM-III) and cognitive impairment among the elderly in a U.S. urban community. *Acta Psychiatrica Scandinavica*, 71, 366–379.

WEYERER, S. (1983) Mental disorders among the elderly: True prevalence and use of medical services. *Archives of Gerontology & Geriatrics*, 2 11–22.

WHITEHEAD, A. (1981) Age and mortality in dementia. *British Journal of Psychiatry*, **139**, 249.

WILKIN, D., DURIE, A., WADE, G., JOLLEY, D. & STOUT, I. (1984) *Specialist Services for the Elderly: A Study of Referrals in Geriatric, Psychiatric and Social Services, Research Report No. 5*, Manchester: Psychogeriatric Unit, University Hospital of South Manchester.

WILLIAMSON, J., STOKOE, I. H., GRAY, S., FISHER, M., SMITH, A., MCGHEE, A. & STEPHENSON, E. (1964) Old people at home: Their unreported needs. *Lancet*, i, 1117–1120.

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The Present State Examination Change Rating Scale

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The development and use of a new psychiatric symptom change scale based on the Present State Examination (PSE) – the PSE Change Rating Scale – is described. The advantages of the PSE are retained (the extensive glossary, the diagnostic facility and the standardised interview technique) while improving its sensitivity to change and decreasing its administration time. The scale has been designed to prevent feedback of results and 'halo' effects: it has proved highly reliable in use across raters with different backgrounds and experience. The scale has been used to monitor drug effects on clinical state across a variety of diagnoses and examples of its use are given.

One of the problems affecting research on the biological concomitants of psychotic illness has been to elucidate the causes of change in clinical state. A common approach has been to test patients initially when they are not receiving medication and to repeat the test some time later after treatment has been instituted. Changes in response to the test between the two occasions could then be due either to the natural history of the symptoms or to an effect of the medication itself. In order to decide between these two alternatives it is necessary to be able to measure sensitively changes in clinical state.

Ideally, a psychiatric clinical rating instrument should:

1. (1) generate data which provide the basis for clinical classification;
2. (2) be sensitive to change in symptoms;
3. (3) provide clear and mutually exclusive definitions of the symptoms;
4. (4) utilise a standardised interview;
5. (5) be acceptable to interviewers and subjects alike.

Manchanda & Hirsch (1986) surveyed five psychiatric journals over 3 years to establish which rating scales were in common use for schizophrenia. Of the six scales in most common use only four were applicable for all psychiatric diagnoses and for both acute and chronic disorders. These were: the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962), the Comprehensive Psychopathological Rating Scale (CPRS) (Åsberg *et al*, 1978), the In-patient Multidimensional Psychiatric Scale (IMPS) (Lorr & Klett, 1967) and the Present State Examination (PSE) (Wing *et al*, 1974). They found that the BPRS is able to satisfy points 2 and 5 above but does not give data for a clinical classification, provide clear definitions or use a standardised interview. The same criticism is true for the IMPS whereas the CPRS satisfies points 2, 3 and 5. The PSE, while satisfying points 1, 3, 4 and 5, was found to be somewhat insensitive to change with, for most symptoms, a 3-point scale and, for some, a 2-point scale compared, for instance, to the 7-point scale for the BPRS. Three points seem to provide too narrow a scale for the sensitivity to change required. This reflects the fact that the PSE was constructed as an instrument for assessing the patients' current psychopathology, and was not primarily designed as a change measure. However, its suitability for measuring change is an empirical question to be studied.

This was the starting point for our attempt to modify the PSE in order to increase its sensitivity to change. A further consideration was the length of the interview. In a floridly ill, psychotic patient, the PSE generally takes 45–60 min to administer. To repeat this procedure at regular intervals on a large number of patients would clearly exhaust the interviewer, might well alienate many of the patients, and would be difficult to fit in with the demands of a clinical job. Therefore, we were also concerned to decrease the administration time of repeated interviews.

Method

The PSE Change Rating is administered in the following manner. Initially, a PSE (9th edition) is administered to the patient by a trained interviewer according to the method described in Wing *et al* (1974). The symptoms that are not rated positively on this interview are discarded, (along with symptoms rated 8 or 9) and the subsequent ratings are made only on items that are rated positively. *The interviewer retains the PSE form throughout the rating period and uses it as the baseline for the ratings.* The words the patient employed to describe the symptoms initially are used as probes in subsequent interviews.

The subsequent ratings are made using a booklet made up of slips of paper. Each slip has only one of the symptoms

Symptom ratings compared to admission

0	Completely remitted
1	Markedly improved
2	Moderately improved
3	Minimally improved
4	Unchanged
5	Minimally worsened
6	Moderately worsened
7	Markedly worsened

FIG. 1. Symptom change scale used for the PSE change rating.

rated positively for that patient typed on it and the slips of paper are in random order. The exception is behavioural observations (PSE items 108–139) which are presented at the end of the booklet when the rater will have observed the patient for the length of the interview. The rater is asked to rate each symptom separately using the obligatory questions and probes from the PSE schedule on a scale 7—markedly worsened to 0—completely remitted (see Fig. 1 for complete scale) *compared to the state of the patient at the initial PSE.*

The rater works through the booklet in the order given without returning to any symptoms. This eliminates the possibility of 'halo' effects (Hilgard *et al*, 1971)—the tendency to rate all related symptoms as better or worse together. When the booklet is completed it is returned to the secretariat so that 'carry-over' effects from one occasion of rating to the next are minimised i.e. the raters do not see their ratings again. Any new symptoms that appear (raters are asked to use general probes) are written on blank slips provided at the end of the booklet and are then included in the next rating booklet. When a symptom has been absent (score of 0—completely remitted) for 3 weeks it is omitted from the booklet.

Certain of the symptoms are not suitable for the increased frequency of assessment associated with a change score and these have been dropped or modified. They comprise symptoms 26 (primacy of anxiety or depression), 27 (diurnal variation of mood), 39 (pre-menstrual exacerbation), 93–97 (general ratings of delusions and hallucinations), 97–107 (organic psychiatric symptoms). Questions that specify a time period for the occurrence of the symptoms need to be changed to the time period since the last rating. The question for symptom 34 (loss of weight due to anorexia) has been modified to take appetite only into account.

In general the ratings, compared to the initial interview, take into account the frequency, severity and, in the case of psychotic symptoms, conviction (insight) and delusional elaboration. Although a symptom needs to reach a certain level of intensity or conviction to be rated as present on the initial PSE it would need to be totally absent to rate 0—completely remitted—on the change rating. This would often mean that a change rating of 1—markedly improved—would not be severe enough to be rated as present if a full PSE were done. Similarly a psychotic symptom, such as delusion, may no longer be present but, if the patient still believes it *was* happening, the symptom is not completely remitted and should be rated as 2—moderately improved, or 1—markedly improved, depending on the strength of conviction.

Examples of repeated ratings carried out on acute admission psychiatric patients

Five examples of ratings carried out on patients admitted to the research ward are given below. They represent a spectrum of diagnosis from acute-onset schizophrenia to prolonged neurotic depression. All patients had been unmedicated for at least 4 weeks prior to admission. In presenting the change ratings graphically each PSE item can be charted separately. In practice, we have found it most useful to present all psychotic symptoms combined, all neurotic symptoms combined, and then an amalgam of all symptoms (which can be seen in Fig. 2). This gives an overall picture of change.

Case A

This 23-year-old woman had suffered three previous psychotic episodes, each of which began abruptly and resolved within a week or so. She had been prescribed lithium, but was not taking it regularly. The present episode began a week before admission while the patient was holidaying abroad. She experienced auditory hallucinations and delusions of control, and exhibited histrionic and uninhibited behaviour. She was treated with haloperidol up to 6 mg twice daily. Her psychotic symptoms resolved within 2 weeks (see Fig. 2A).

Case B

This 35-year-old woman was admitted after a long period of gradual social decline. On examination she showed self-neglect, irrelevant speech and inappropriate laughter. She complained that people interfered with her thinking and put thoughts into her head. She also heard voices calling her. It was considered that she had been suffering from untreated schizophrenia for several years and she was prescribed oral haloperidol up to a dose of 24 mg twice daily. She showed some improvement and was then changed to injections of haloperidol decanoate, 300 mg every 4 weeks. Her clinical condition stabilised, but she developed uncontrollable akathisia. For this reason her medication was altered to fluphenazine decanoate, 100 mg weekly. Unexpectedly, her symptoms showed a further substantial improvement, which first became evident from the PSE Change Ratings (see Fig. 2B). She was eventually discharged to live with a foster family.

Case C

This 52-year-old man had experienced 17 previous admissions for manic-depressive psychosis. On this occasion he had been sleeping rough for several days and then spent 3 days with the Simon Community prior to admission. On examination he was elated, jocular, irreverent and overactive. He showed flight of ideas, pressure of speech and expressed grandiose delusions about writing a best-seller. He was treated with haloperidol up to a dose of 15 mg twice daily. Lithium was added after the second week, following which all his symptoms rapidly resolved (Fig. 2C).

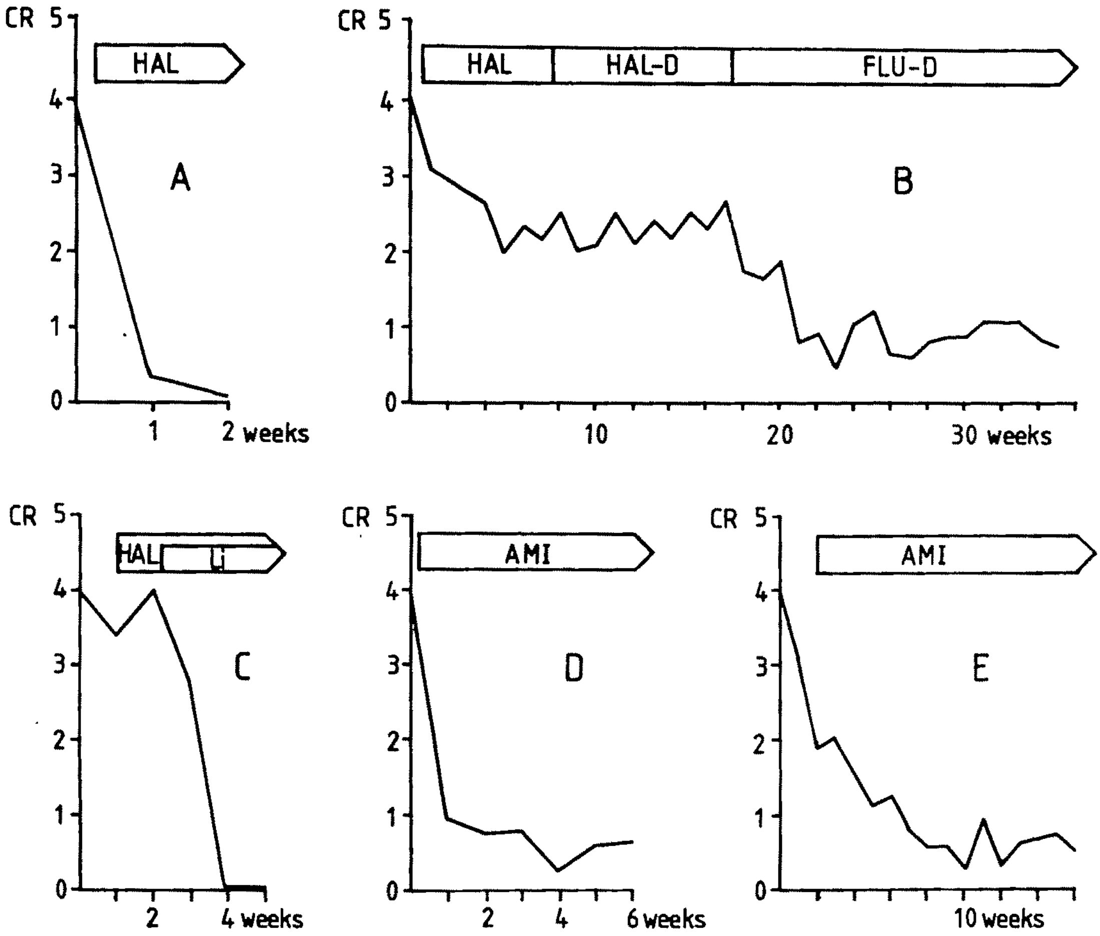

FIG. 2 Illustrations of the symptom change scores over time of cases A–E described in the text. The abscissa is the time in weeks from admission (week 0) and the ordinate is the mean clinical rating for all symptoms (CR). Medication is represented by the names above the graphs. HAL = Haloperidol (oral); HAL-D = Haloperidol Deconate (depot injection); FLU-D = Fluphenazine Deconate (depot injection); LI = Lithium carbonate (oral); AMI = Amitriptyline hydrochloride (oral).

Case D

This 44-year-old man was admitted following an attempt to hang himself. He was divorced and had not worked for 4 years. He had been living in a hostel for 3 months prior to admission. On examination he was depressed and tearful and expressed feelings of severe guilt. He heard people in the street talking about him. He also heard the sound of knives being sharpened at the hostel and believed that he was going to be killed. He was treated with amitriptyline, 150 mg nocte and the psychotic features resolved rapidly (see Fig. 2D), although he was left with persistent feelings of inadequacy.

Case E

This 56-year-old man was admitted with a prolonged bereavement reaction to his mother's death a year previously. He had lived with his parents all his life and never married. Following his mother's death he had moved into a flat of his own, but was completely isolated. He had been drinking excessively and gave a long history of compulsive gambling. He was treated with amitriptyline, 150 mg nocte and showed considerable improvement in his depressive symptoms (see Fig. 2E). However, he remained a very dependent person and continued gambling.

Experiment 1: Inter-rater reliability of the PSE Change Rating Scale

As with any instrument it is necessary to test the reliability of the rating procedure by comparing ratings made on the same patient by different, independent raters. In administering the PSE Change Rating Scale, as with the PSE itself,

TABLE I
Psychiatric diagnosis, sex and age of the patients rated in Experiment 1

Patient no	*Sex*	*Age*	*CATEGO diagnosis*	*Clinical diagnosis*
1	M	21	S+	Schizophrenia
2	F	34	S+	Schizophrenia
3	M	42	S+	Schizophrenia
4	F	39	S+	Schizophrenia
5	M	35	P+	Schizophrenia
6	F	41	P+	Schizophrenia
7	F	33	P+	Schizophrenia
8	F	25	P+	Manic psychosis
9	F	62	M+	Manic psychosis
10	M	29	D+	Depressive psychosis
11	M	44	D+	Depressive psychosis
12	M	37	D+	Depressive psychosis
13	M	41	N+	Depressive neurosis
14	M	26	N+	Depressive neurosis

there are two independent skills involved. One is the actual interview technique and the other is the rating of the patient's replies and behaviour. As the interview technique was identical to that used with the PSE and all raters had been trained to the same standard for administering the interview, it was not thought necessary to check the reliability of this aspect of the procedure. It was decided, therefore, to have one rater interview the patient in the presence of three other raters and for all four to rate the patient independently and simultaneously. This allowed the inter-rater reliability to be calculated for the symptom change ratings.

Subjects

All subjects were psychiatric patients recently admitted to research beds on an acute admissions ward. None of the patients had received medication for at least 2 weeks prior to admission. Patients with a history of drug or alcohol dependence were excluded, as were those over 65 years of age and those suffering from dementia. Apart from these exclusions, the patients were 14 consecutive admissions, who gave consent to the procedures. Information about the patient sample is shown in Table I.

All patients were started on drug regimes after the initial diagnostic PSE and before the first measure of clinical change was made. All the psychotic patients except 2 and 11 were given haloperidol in standard doses, rising weekly until the patient was judged to be well. Patients 8 and 12 also received lithium, patients 10 and 12 received amitriptyline, patient 8 received chlorpromazine and patient 12 had a course of ECT. Patient 2 received only fluphenazine deconate and patients 11, 13 and 14 received only amitriptyline.

Raters

The raters were from varied backgrounds and with differing amounts of clinical experience. Three of the raters remained constant throughout the study but part way through the study one rater (the psychiatric registrar) was changed so that half the patients were rated by one registrar and the other half by the other registrar. Two of the raters were psychiatrists — one a consultant psychiatrist (A) working in psychiatric research and 17 years experience in using the PSE, the other a psychiatric registrar (B) with a few months experience with the PSE. The other two raters were both clinical psychologists — one a senior research psychologist (C) with 4 years' experience of the PSE and the other a basic grade psychologist (D) with 6 months' experience of the PSE. Neither of the two clinical psychologists was involved in the treatment of the patients and they only saw the patients at initial test and to rate them at weekly intervals. The two psychiatrists were involved in the daily care of the patients.

Procedure

Each patient was assessed using the 9th edition of the PSE within 48 hours of admission and before drug treatment was initiated. The initial PSE was carried out by one of the raters in the presence of the remaining three raters who rated the interview independently. At the end of the interview the raters came to a consensus on the symptoms that were present and these were the symptoms used for the ratings the following week. Ratings took place at weekly intervals following the initial PSE. Again, one rater interviewed and rated the patient, while three of the raters rated the patient. All four 'symptom booklets' contained the symptoms in the same random order, with the behavioural observations (PSE items 108–140) at the end. Occasionally, a rater was unable to attend a session and then the session was audio taped for her/him to rate as soon as possible after the interview. This resulted, occasionally, in lost data when behavioural items (other than those rated from speech) were rated. Each patient was rated for 4 weeks or until discharge or other factors made weekly testing impossible. All booklets were handed to a research assistant at the end of the interview. No rater saw the ratings from another rater until the end of the experiment, nor did they discuss their own ratings with each other.

Results

Four of the 14 patients were rated for only 2 weeks and two for 3 weeks. The remaining 8 patients were rated for 4 weeks. Two of the patients had no psychotic symptoms, one patient had no neurotic symptoms and one patient had no behavioural symptoms. The inter-rater reliabilities of the ratings were measured using the Intra-class Correlation Coefficient (unbiased estimator method) (Winer, 1971) and significance tested by the *F* statistic (Bartko & Carpenter, 1976). The results are shown in Table II.

The coefficients range from 0.75 to 0.99, with all but one of the 28 coefficients exceeding 0.8. All the *F* values in the table are significant beyond the 1% level.

This shows an extremely high degree of reliability between raters for these measures. The lowest reliabilities (although still statistically significant) are for the behavioural items. This result is consistent with the findings from studies of the PSE itself (summarised in Wing *et al*, 1974) which also

TABLE II

Intra-class correlation coefficients of total symptom ratings and grouped symptom ratings for the four raters (r_4') in Experiment 1

	Week 1			*Week 2*			*Week 3*			*Week 4*		
Mean score	r_4'	*n*	*F*	r_4'	*n*	*F*	r_4'	*n*	*F*	r_4'	*n*	*F*
All symptoms	0.98	14	46.8	0.98	14	42.8	0.98	10	46.5	0.97	8	42.3
All psychotic symptoms[1]	0.98	12	62.8	0.97	12	36.3	0.97	8	34.9	0.87	6	8.8
All neurotic symptoms[2]	0.99	13	136.1	0.98	13	55.9	0.99	10	78.7	0.98	8	69.7
All behavioural symptoms[3]	0.75	11	4.2	0.91	11	11.3	0.84	7	6.8	0.91	6	12.3
All delusions[4]	0.96	12	73.1	0.98	12	46.0	0.97	8	42.1	0.87	6	8.7
All hallucinations[5]	0.98	9	43.2	0.94	9	17.1	0.83	6	6.6	0.93	3	19.7

1. Includes possible ratings of PSE symptoms 55–64, 66–81, 83–92
2. Includes possible ratings of PSE symptoms 4–25, 27–38, 40–54
3. Includes possible ratings of PSE symptoms 108–139
4. PSE symptoms rated in this group of patients 59, 71–81, 86–88, 91, 92
5. PSE symptoms rated in this group of patients 60–63, 68, 70.

found that the reliabilities for behavioural items were lower than for symptoms. Individual symptoms (where these symptoms were sufficiently common to occur in at least four patients) were also subjected to analysis by the Intra-Class Correlation Coefficient and the results are shown in Table III.

Again, the coefficients are high and range from 1.00 (complete accord) down to 0.70. All the *F* values are significant beyond the 1% probability level.

Pearson Product Moment Correlation Coefficients (Ferguson, 1976) were also calculated on the data to compare pairs of raters across the 4 weeks and with all the symptom subgroupings shown in Table III. The smallest range of *r* values was from 0.27 to 0.98 for delusions. However, the majority (76%) of the correlations were above 0.80. Individual raters also followed this pattern—of the *r* values associated with each rater approximately 75% were above 0.80 (A = 74%; B = 79%; C = 80% and D = 73%). It is evident, that high levels of reliability were achieved, even by raters who had relatively little clinical experience.

Experiment 2: A comparison of the PSE Change Rating Scale with the standard PSE scoring method

The results of the previous two sections show clearly that the PSE Change Rating Scale measures clinical change in psychiatric patients and that it has high inter-rater reliability across symptom groupings. However, the introduction of

TABLE III

Intra-class correlation coefficients of individual symptom ratings for the four raters (r_4') in Experiment 1

	Week 1			*Week 2*			*Week 3*			*Week 4*		
Score	r_4'	*n*	*F*	r_4'	*n*	*F*	r_4'	*n*	*F*	r_4'	*n*	*F*
Symptom 20 (poor concentration)	0.85	7	7.5	0.97	7	39.4	0.99	5	82.0	0.89	3	11.8
Symptom 23 (depressed mood)	0.98	7	45.9	0.95	7	21.6	0.99	4	139.7	1.00	4	∞
Symptom 62[1] (voices talking about the pt.)	0.97	4	35.4	0.70	4	4.0	0.92	3	15.1	—	—	—
Symptom 72 (delusions of reference)	0.99	8	94.0	0.98	9	45.1	0.90	5	8.5	0.85	5	8.5
Symptom 131 (pressure of speech)	0.75	5	4.7	0.95	6	20.9	0.89	4	10.6	0.94	3	21.0

1. Insufficient data were available for performing the test at the 4th week for admission.

TABLE IV
Psychiatric diagnosis, sex and age of additional patients rated in Experiment 2

Patient no.	*Sex*	*Age*	*CATEGO diagnosis*	*Clinical diagnosis*
15	M	31	S+	Schizophrenia
16	M	24	S+	Schizophrenia
17	M	20	S+	Schizophrenia
18	F	25	M+	Manic psychosis
19	F	32	D+	Depressive psychosis
20	F	21	N+	Neurotic depression
21	F	28	N+	Neurotic depression

the 0–7 scale might be an unnecessary complication (Kazdin, 1977) and the relative nature of the remission/relapse ratings might make cross-comparisons of clinical severity difficult. The existing scoring scheme for the PSE (0, 1, 2) is one way of overcoming these possible problems. Thus, it was planned to introduce the existing PSE scoring scheme in parallel with the PSE Rating Scale. In this experiment each patient, on each occasion, was rated by the same rater on the two different rating schemes.

Subjects

The criteria for selection of patients were the same as for experiment 1 and 13 of the patients in Experiment 1 were included in this experiment (patient 9 was not rated). A further seven patients were included making a group of 20 patients. Information about these seven patients is shown in Table IV. Patients 16, 17 and 18 received haloperidol, patient 19 received amitriptyline, patient 15 received diazepam, patient 20 received thioridazine, and patient 21 received dothiepin hydrochloride, and chlorpromazine.

Procedure

The patients were rated by the psychiatric registrars using the symptom booklets in the manner described above but also assigning a score on the existing PSE scale. Each patient was rated weekly—eight by one registrar and 12 by the other registrar. As in Experiment 1 each booklet was given in by the rater as soon as it had been completed.

Results

It became apparent during the course of this experiment that the raters experienced a great deal of difficulty in using the existing PSE scale to reflect the change that they observed. This was seen in the large percentage of symptoms that were not rated on this scale. Of the 90 rating sessions in only 40% were all symptoms on the existing PSE scale rated. In 15.5% of sessions more than 75% but fewer than 100% of symptoms were scored. In a further 15.5% of sessions between 50% and 75% of symptoms were rated and in 29% fewer than 50% of symptoms were rated.

The major problems occurred with those symptoms of the PSE in which the difference between a score of 1 and a score of 2 is a qualitative rather than a quantitative one. That is when an improvement in the patient would not necessarily be associated with a change score from 2 to 1.

In particular for the Schneiderian first-rank symptoms 56–58 and auditory hallucinations 60–64 and visual hallucinations 66 and 67 ratings of 1 and 2 indicate quite different symptoms. For example symptom 57 rates 1 for thought echo and 2 for thought commentary. Symptom 61 rates 1 for hearing single words, 2 for hearing voices based on depressed mood and 3 for voices based on elated mood.

The raters, although well aware of the criteria for rating symptoms on the PSE, found it extremely frustrating being unable to reflect the changes they saw in the symptoms (which is what they had been asked to do) so, instead of marking a symptom absent (by PSE criteria) when it was still present (by PSECRS criteria) they did not rate the symptom by PSE criteria. They also commented on the fact that, when a symptom was originally rated 2 on the PSE criteria there was no way of registering a worsening of the symptom.

Discussion

The difficulty of the raters in using the standard PSE to assess clinical change over short periods of time reflects the fact that its primary purpose was to standardise the cross-sectional examination of the mental state. The clinical examples given demonstrate that its modification has rendered it sensitive to change. The PSE Change Rating Scale was constructed *using psychometric principles* to reduce any possibility of 'halo effects' to a minimum and to avoid the possibility of 'carry-over' effects from one occasion of testing to the next. We have found the inter-rater reliability to be extremely high overall, regardless of the length of clinical experience of the rater. The lowest reliabilities were found for the behavioural items, as with the standard PSE.

A disadvantage of this scale is that, by restricting questioning to symptoms that were present at the original interview, new symptoms might be missed. In practice, with a newly admitted acute psychiatric population, new symptoms seem to be rare. However, this certainly would be a problem with a more chronic population. Supplementary questions could be devised for these populations based on symptoms known to have been present in the past (from notes) or on observation from ward staff. Indeed, this latter source is also available for acute populations and was made use of for probes in this study.

We have found it useful to analyse the data generated by this new instrument as psychotic symptoms, neurotic symptoms, and total symptoms. However, the scores may be displayed for any group of symptoms of interest, or even for individual items.

The assessment of symptom change over short periods of time is essential for the evaluation of any psychiatric treatment, whether pharmacological or social in nature. We have shown the modified PSE to be sensitive to change, easy to use by trained raters, and reliable across diagnostic groups.

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References

Åsberg, M., Perris, C., Schalling, D. & Sedvall, G. (1978) The CPRS — Development and applications of a psychiatric rating scale. *Acta Psychiatrica Scandinavica* (Suppl.) 271, Copenhagen: Munksgaard.

Bartko, J. J. & Carpenter, W. T. (1976) On the methods and theory of reliability. *Journal of Nervous & Mental Diseases*, **163**, 307–317.

Ferguson, G. A. (1976) *Statistical Analysis in Psychology and Education*, 4th ed. New York: McGraw-Hill Book Company.

Hilgard, E. R., Atkinson, R. C. & Atkinson, R. L. (1971) *Introduction to Psychology*, 5th ed. New York: Harcourt, Brace, Jovanovich.

Kazdin, A. E. (1977) Artefact, bias and complexity of assessment: The ABCs of reliability. *Journal of Applied Behaviour Analysis*, **10**, 141–150.

Lorr, M. & Klett, C. J. (1967) *Inpatient Multidimensional Psychiatric Scale: IMPS* (revised manual) Palo Alto, California: Consulting Psychologists Press.

Manchanda, R. & Hirsch, S. R. (1986) Psychopharmacological Research in schizophrenia. 1. Review of Rating Scales. In *The Psychopharmacology and Treatment of Schizophrenia*. (eds P. B. Bradley & S. R. Hirsch). New York, Oxford: Oxford University Press.

Overall, J. E. & Gorham, D. R. (1962) The Brief Psychiatric Rating Scale, *Psychological Reports*, **10**, 799–812.

Winer, B. J. (1971) *Statistical Principles in Experimental Design*, 2nd ed. Kogakusha, Tokyo: McGraw-Hill.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*, Cambridge: Cambridge University Press.

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CSF Neuropeptides in Euthymic Bipolar Patients and Controls

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Levels of vasopressin, somatostatin, neurotensin, vasoactive intestinal peptide, corticotrophin-releasing factor and adrenocorticotrophin in CSF were determined in lithium-treated and unmedicated euthymic bipolar patients and controls, in a search for a trait marker in affective disorder. No group differences in levels of these peptides were found. Highly significant positive correlations were found among these peptides (with the exception of neurotensin), suggesting that their presence in CSF is functionally significant, as opposed to the result of random diffusion from the interstitial space of the brain.

Bipolar affective (manic-depressive) illness is a cyclic mood disorder of unknown aetiology, in which genetic factors play an important role in pathogenesis (for review, see Berrettini *et al*, 1984). Manic-depressives characteristically have cyclic episodes of markedly sad or euphoric mood, associated with other symptoms, but are well between episodes of mood disturbance. Theoretically, the underlying genetic factors, which increase one's vulnerability to develop affective illness, should be detectable independently of mood state. The study of well-state (euthymic) patients avoids the difficulty of separating state-related abnormalities (associated with acutely ill manic or depressed patients) from trait abnormalities (which may be genetic vulnerability markers). A strategy of studying euthymic patients has been delineated previously (Rieder & Gershon, 1978).

Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis has been observed in actively ill manic-depressive patients (Sachar *et al*, 1973; Greden *et al*, 1983). Both the abnormal dexamethasone suppression test results (Greden *et al*, 1983) and the hypercortisolaemia (Sachar *et al*, 1973), seen among actively ill manic-depressive patients, are thought to disappear after remission of the depression or mania (Greden *et al*, 1983). Abnormalities of pituitary adrenocorticotrophin (ACTH) response to corticotrophin-releasing factor (CRF) may accompany the dysregulation of cortisol in these patients (Gold *et al*, 1984). These observations led to the conclusion that there is some state-dependent dysfunction of the HPA axis in some patients with affective illness.

The HPA axis is regulated in part by hypothalamic neurons which release CRF, ACTH, vasopressin (AVP), somatostatin (SRIF), vasoactive intestinal peptide (VIP) or other neuropeptide transmitters and/or hormones (Axelrod & Reisine, 1984). For these five peptides, much evidence suggests that brain is the source of the CSF levels, which are regulated independently of the plasma levels (Wood, 1982; Post *et al*, 1982). In the light of the evidence that the HPA axis is regulated abnormally in the actively ill manic-depressive patient, we reasoned that evidence of a state-independent (genetic) abnormality might be found by examination of some HPA-axis-related peptides in CSF. While plasma evidence of HPA axis dysregulation may not be evident in the euthymic patients, CSF levels of HPA-axis-related peptides may be abnormal, representing a biological genetic vulnerability marker. Abnormalities in CSF levels of HPA-axis-related peptides have been reported among acutely ill patients. Significantly lower CSF levels of CRF (Nemeroff *et al*, 1984), VIP (Gjerris *et al*, 1984), AVP (Gold *et al*, in press; Gjerris *et al*, 1985) and SRIF (Gerner & Yamada, 1982; Rubinow *et al*, 1983) were found among actively depressed subjects, suggesting that CSF levels of these peptides should be evaluated as possible trait markers for vulnerability to affective illness.

Recently, it has been reported that VIP is contained and secreted with acetylcholine from some cholinergic neurons (Lundberg *et al*, 1980). VIP may be released into CSF after cholinergic stimuli (Kaji *et al*, 1983). Neurotensin (NT) has been reported to stimulate cholinergic function in guinea-pig enteric neurons (Yau *et al*, 1983); this stimulation is thought to be secondary to increased presynaptic release of acetylcholine and is blocked by SRIF. These observations (that NT and VIP may be modulators of cholinergic function) prompted us to measure CSF levels of these neuropeptides in euthymic bipolar patients to determine whether the reported cholinergic supersensitivity in affective disorder (Sitaram *et al*, 1980; Nadi *et al*, 1984) is associated with abnormalities in the CSF levels of peptidergic modulators of cholinergic function.

Method

Subjects

All individuals participating in this study were out-patients at the National Institute of Mental Health, Bethesda, Maryland. All provided informed consent. The bipolar patients and normal subjects (as well as the clinical details of the lumbar puncture) have been previously described (Berrettini *et al*, 1985*b*). Briefly, euthymic bipolar patients (who were treated solely with lithium for at least 1 month prior to study) and normal volunteers underwent a lumbar puncture as an out-patient procedure after 2 h of bedrest. In addition, 15 of the lithium-treated patients discontinued lithium for at least 2 weeks to provide a second CSF sample in the unmedicated state. Thus, for these patients, paired CSF samples, on and off lithium, were obtained.

Peptide radioimmunoassay

CSF concentrations of immunoreactive human CRF were measured as previously described (Roy *et al*, in press), except that unextracted CSF was used.

VIP radioimmunoassay kits were obtained from Immunoclear Corporation (Stillwater, Minnesota). VIP was determined in duplicate 0.450-ml aliquots of CSF. Recovery of added standard was 90%. Final volume was 0.650 ml. The limit of detection was 4 pg/tube.

A volume of 0.200 ml of unextracted CSF was used to make each AVP determination, as previously described (Zerbe *et al*, 1982).

SRIF was determined on 1.00 ml of lyophilised CSF, as described previously (Patel & Reichlin, 1979; Rubinow *et al*, 1983), and levels were corrected by indexing to chloride concentration.

NT radioimmunoassay kits were purchased from Immunoclear Corporation (Stillwater, Minnesota). NT was determined in duplicate 0.400-ml samples of CSF collected from a 1-ml fraction to which 0.020 ml of concentrated perchloric acid was added. Samples were adjusted to neutral pH with 10-M NaOH before assay. Recovery of added standard was 90%, and the limit of detection was 35 pg/ml.

For the ACTH radioimmunoassay, Sep-Pak cartridges were washed with 10 ml of methanol followed by 10 ml of distilled water. Aliquots of 0.6 ml of CSF were applied to the cartridges which were then washed with 10 ml of distilled water. Immunoreactive ACTH was eluted with a mixture of TEAF buffer (1% formic acid, pH 3.2 with addition of triethylamine) and acetonitrile (4 : 6, v/v). The eluent was lyophilised and the residue taken up in assay buffer (0.63-M Na_2HPO_4 and 0.13-M Na_2EDTA, pH 7.4), containing 0.02% Na_2NO_2, 0.1% triton X-100 and 250 KIU of aprotinin/ml. The recovery of ACTH by this procedure is 85–95%. The antibody was obtained from IgG Corporation (Nashville, Tennessee, ACTH-1). Both standard and trace were $ACTH_{1-39}$. ACTH labelled with ^{125}I was prepared using the chloramine-T method (Greenwood *et al*, 1963). The specific activity was approximately 450 Ci/g. The methods of the radioimmunassay have been previously described (Orth, 1979). The detection limit is 3–5 pg/ml.

Analysis of data

A general linear models procedure was used to assess the effects of age, height, weight and sex on CSF peptide levels. ACTH levels in CSF were corrected for a sex effect. Males ($n=33$, $\bar{x}=22.2\pm5.7$ pg/ml) had significantly higher ($P<0.005$) values than did females ($n=34$, $\bar{x}=18.4\pm4.7$ pg/ml). No other peptide was altered significantly by age, sex, height or weight.

Group *t*-tests were performed to compare euthymic bipolar patients and controls. Paired *t*-tests were used to study the effect of lithium, using those patients from whom paired CSF samples were obtained. Pearson correlation coefficients were calculated for the three separate groups of data (and on all individuals combined) to examine the relationships between these CSF neuropeptides. Z transformation of the correlation coefficients was done to evaluate possible significant differences in correlation coefficients between groups.

All ACTH, CRF, VIP and NT levels were measured in one assay. The coefficient of variation within an assay was less than 15% for all assays. SRIF and AVP levels were measured in two separate assays.

Results

The levels of the six neuropeptides are shown in Table I. No group differences (*t*-tests) or effects of lithium (paired *t*-test) were found, except a trend ($P=0.05$) for CSF VIP to be lower during lithium treatment.

As shown in Table II, a number of significant positive correlations were found among AVP, ACTH, CRF, SRIF and VIP. The correlation coefficients were similar for the three subject groups, with the exception of the correlation coefficient for CRF and VIP (see Table II). The strongly positive correlation between CRF and VIP in controls was not present among the bipolar patients, suggesting that the patients may have had a dysfunction of whatever regulatory mechanisms determine the levels of these two peptides in CSF. No significant correlations (defined as $P\leqslant0.05$) were found between CSF NT levels and any other peptide (data not shown).

Discussion

The most interesting finding in this study is the significant correlations among CSF neuropeptides. This deserves a comment on the origins of CSF neuropeptides.

Some hypothalamic neurons (that contain SRIF) have nerve endings at the wall of the third ventricle (Pollay M., 1979; Knigge *et al*, 1980). Several investigators have hypothesised that neuropeptides may be released into CSF from such periventricular neurons, to be transported to distant CNS sites of action (Pollay, 1979; Knigge *et al*, 1980; Post *et al*, 1982). As indicated in the review by Jackson (1981), peptides may gain access to CSF via diffusion from CNS interstitial fluid, from the periphery at sites

TABLE I

CSF neuropeptide levels[1] in euthymic bipolar patients and controls

Peptide	*Controls*	*Lithium-treated bipolar patients*	*Unmedicated bipolar patients*
Neurotensin	58.6±13 (25)	65.2±6.8 (19)	56.0±13 (11)
Vasopressin	4.4±1.0 (30)	4.6±1.0 (25)	4.6±1.7 (14)
VIP	18.0±7.4 (32)	15.7±6.0 (21)	20.0±6.5 (13)
ACTH	22.6±5.8 (29)	21.4±5.8 (25)	22.3±5.4 (13)
SRIF	36.3±16.4 (31)	32.3±15.6 (27)	39.8±15.1 (16)
CRF	33.0±11.9 (31)	29.8±12.9 (26)	31.8±13.7 (15)

1. Values are mean (pg/ml)±standard deviation. The number of observations is given in parentheses.

where the blood–brain barrier does not exist or by transport from the tanycyte median eminence, as well as by direct release from neurons into CSF. However, Wood's (1982) review states that peripheral sources of CSF peptides are unlikely. Thus, it seems more probable that peptides reach CSF either through random diffusion or direct neuronal release.

Do the observed significant correlations favour direct release or random diffusions as the point of entry of neuropeptides into CSF? These correlations do not provide strong evidence favouring either hypothesis. However, these significant correlations are more congruous with the hypothesis that the concentrations of CRF, SRIF, AVP, VIP and ACTH in CSF are not the result of random simple diffusion from the interstitial space of the neurons of origin, because differences in number and activity of the various peptidergic neurons and their distance from the ventricular space would affect the process of random diffusion, and one would be less likely to observe significant correlations between numerous peptides (Berrettini *et al*, 1985*a*).

One possible explanation for the correlations between SRIF, AVP, VIP, CRF and ACTH in CSF may be that they are released from neurons into CSF in response to a stimulus. From their sites of release, they may be transported to distant CNS sites where each of them may mediate some aspect of the response to that stimulus. What evidence exists for stimulus-induced changes in these CSF peptides? Koob & Britton (1984) have shown that random inescapable footshock causes a doubling in rat CSF CRF, a response which is attenuated by diazepam. CSF, ACTH and β-endorphin levels increase several-fold following periaqueductal grey (but not internal capsule) self-stimulation for analgesia (Hosobuchi & Bloom, 1983). These studies suggest that certain stimuli can increase CSF levels of these peptides. Shared mechanisms of release into CSF may account for these correlations.

Other possible explanations for these correlations include shared catabolism by peptidases in CSF and shared transport out of CSF, but no evidence exists to support either of these possibilities.

Rubinow *et al* (1985) have reported that carbamazepine, which is an effective acute and prophylactic treatment for manic-depressive illness (Okuma *et al*, 1973; Post *et al*, 1983), decreases CSF SRIF levels. Because carbamazepine seems to have a therapeutic effect in the treatment of manic-depressive illness which is similar to that of lithium, we hypothesised that lithium might have a similar effect on CSF SRIF. This was clearly not the case in our data. Our results support those of Rubinow *et al* (1983), who reported that recovered patients did not show significantly decreased CSF SRIF levels, as was found among the acutely depressed patients. Thus, the lower CSF SRIF level is a state-dependent marker for depression.

Given that lithium antagonises the effects of AVP (Forrest, 1979) at a post-receptor site in the kidney, we hypothesised that lithium might cause increased presynaptic release of AVP in the CNS, yielding a higher CSF AVP level. There was no effect of lithium on CSF AVP, but we cannot exclude the possibility that lithium may alter CNS AVP activity without affecting CSF AVP levels.

Recent data show that CSF ACTH and CSF CRF levels are not abnormal in acutely depressed patients (Davis *et al*, 1984; Roy *et al*, in press), although Nemeroff *et al* (1984) found higher CSF CRF in acutely depressed patients. Thus, although evidence for abnormal pituitary ACTH regulation in acutely depressed patients has been reported by Gold *et al* (1984), CRF or ACTH levels in CSF do not appear to be affected. These observations are consistent with the hypothesis that CSF levels of these HPA-axis-related peptides are regulated independently of HPA axis function.

CSF levels of VIP and NT do not provide evidence for a trait disturbance of cholinergic function in affective illness. The fact that the bipolar group does not show a significant positive correlation between

TABLE II
CSF neuropeptide correlations[1]

		ACTH	*VIP*	*AVP*	*SRIF*
Controls					
CRF	*r*	0.62	0.63	0.24	0.28
	P	0.0005	0.0002	0.19	0.14
	n	28	30	30	29
ACTH	*r*		0.56	0.39	0.37
	P		0.0024	0.050	0.060
	n		27	26	27
VIP	*r*			0.22	0.50
	P			0.24	0.0057
	n			29	29
AVP	*r*				0.34
	P				0.07
	n				28
Lithium-treated euthymic bipolar patients					
CRF	*r*	0.41	0.075**	0.42	0.17
	P	0.045	0.75	0.040	0.41
	n	24	20	24	26
ACTH	*r*		0.42	0.57	0.028
	P		0.064	0.0038	0.89
	n		20	24	25
VIP	*r*			0.58	0.27
	P			0.0071	0.24
	n			*20*	*21*
AVP	*r*				0.11
	P				0.60
	n				25
Unmedicated euthymic bipolar patients					
CRF	*r*	0.38	0.18*	−0.056	0.58
	P	0.21	0.58	0.86	0.023
	n	12	12	12	12
ACTH	*r*	0.67	0.22	0.64	
	P		0.024	0.51	0.019
	n		11	11	13
VIP	*r*			0.58	0.36
	P			0.062	0.23
	n			11	12
	r				0.13
	P				0.63
	n				12

[1] *r* = correlation coefficient; *P* = probability; *n* = number of observations. Significant differences from control correlation: $*P<0.05$; $**P<0.01$.

CRF and VIP may reflect a disturbance of some regulatory factor that controls the release of CRF and VIP into CSF. Whether this is related to a trait disturbance of central cholinergic neurons in affective disorder must await further study. This connection seems possible in light of recent evidence that cholinergic agonists increase CSF VIP (Kaji *et al*, 1983).

Gjerris *et al* (1984) reported decreased CSF VIP levels in atypically depressed patients. They rated their patients atypical, using the Newcastle Rating Scale. While our patients were not rated with this scale, we believe that they are not atypical. Thus, our results in a euthymic population may be in agreement with those of Gjerris *et al* (1984), who found normal CSF VIP levels in their endogenously depressed patients.

The trend for lithium to reduce CSF VIP levels must be interpreted with caution, as it is of borderline significance. We have reported elsewhere similar results in plasma and an effect of lithium to increase the affinity of a lymphocyte VIP receptor for its ligand (Berrettini *et al*, 1985*b*). Whether this effect of lithium on VIP function is related to the efficacy of that ion in affective illness must await further study. The levels reported here for CSF VIP are somewhat lower than previously reported by us (Berrettini *et al*, 1985*b*). This may represent slight changes in antibody specificity. (The antibody is from a commercial source.)

That CSF NT levels are not correlated with those of any other CSF peptide suggests that CSF NT levels are relatively independent of whatever factors generate the correlations between the CSF levels of CRF, ACTH, VIP, SRIF and AVP. This statement is limited by the difficulty in drawing conclusions about the absence of a significant correlation.

In summary, we find no evidence in bipolar affective illness for a trait disturbance of CSF, SRIF, ACTH, CRF, AVP, VIP or NT. It is unknown to what extent CSF levels of these neuropeptides reflect CNS activity of the peptidergic neurons in question. CSF levels of ACTH, CRF, SRIF, AVP and VIP are positively correlated with one another, suggesting that they share a common regulatory factor which determines CSF levels. Lastly, some evidence has been obtained suggesting that the functional relationship of CRF and VIP in CSF is disturbed in euthymic bipolar patients.

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References

AXELROD, J. & REISINE, T. D. (1984) Stress hormones: their interaction and regulation. *Science*, **224**, 452–459.

BERRETTINI, W. H., GOLDIN, L. R., NURNBERGER, J. I., JR, & GERSHON, E. S. (1984) Genetic factors in affective illness. *Journal of Psychiatric Research*, **18**, 329–350.

—— NURNBERGER, J. I., JR, GOLD, P. W., CHRETIEN, M., CHROUSOS, G. P., CHAN, J. S. D., GOLDIN, L. R. & GERSHON, E. S. (1985*a*) Neuropeptides in human cerebrospinal fluid. *Life Sciences*, **37**, 1265–1270.

——, ——, Hotvedt, P., Simmons—Alling, S. & Gershon, E. S. (1985*b*) VIP and bipolar affective illness: evidence for an effect of lithium. *Journal of Affective Disorders*, **8**, 55–63.

Davis, B. M., Davis, K. L., Mohs, R. C., Mathe, A. A., Vale, W. & Krieger, D. (1984) Abstracts of the Annual Meeting, American College of Neuropsychopharmacology, San Juan, Puerto Rico, 10–14, December, p. 26.

Forrest, J. N. (1979) Lithium-induced polyuria: cellular mechanisms and response to diuretics. In *Lithium: Controversies and Unresolved Issues* (eds T. B. Cooper, S. Gershon, N. S. Kline & M. Schou). Amsterdam: Excerpta Medica.

Gerner, R. H. & Yamada, T. (1982) Altered neuropeptide concentrations in CSF of psychiatric patients. *Brain Research*, **238**, 298–302.

Gjerris, A., Rafaelsen, O. J., Vendsborg, P., Fahrenkrug, J. & Rehfeld, J. F. (1984) VIP decreased in CSF in atypical depression. *Journal of Affective Disorders*, **7**, 325–337.

——, Hammer, M., Vendsborg, P., Christensen, N. J. & Rafaelsen, O. J. (1985) Cerebrospinal fluid vasopressin changes in depression. *British Journal of Psychiatry*, **147**, 696–701.

Gold, P. W., Chrousos, G., Kellner, C., Post, R., Roy, A., Avgerinos, P., Schulte, H., Oldfield, E. & Loriaux, D. L. (1984) Psychiatric implications of basic and clinical studies with corticotropin releasing factor. *American Journal of Psychiatry*, **141**, 619–627.

——, Ballenger, J. C., Robertson, G. L., Robinow, D. R., Goodwin, F. K. & Post, R. M. (in press) Plasma and CSF arginine vasopressin in primary affective disorder. *American Journal of Psychiatry.*

Greden, J. F., Gardner, R., King, D., Grunhaus, L., Carroll, B. J. & Kraunfol, Z. (1983) Dexamethasone suppression tests in antidepressant treatment of melancholia. *Archives of General Psychiatry*, **40**, 493–500.

Greenwood, F. C., Hunter, W. M. & Glover, J. S. (1963) The preparation of ^{131}I-labeled human growth hormone of high specific radioactivity. *Biochemical Journal*, **89**, 114–123.

Hosobuchi, Y. & Bloom, F. E. (1983) Analgesia induced by brain stimulation in man: its effect on release of beta-endorphin and ACTH into CSF. In *Neurobiology of Cerebrospinal Fluid* (ed. J. H. Wood) vol. 2. New York: Plenum Press.

Jackson, I. (1981) Neural peptides in the CSF. *Advances in Biochemical Psychopharmacology*, **32**, 337–355.

Kaji, H., Chihara, K., Minamitani, N., Kodarna, H., Yaraihara, N. & Fajita, T. (1983) Release of VIP into CSF of the fourth ventricle of the rat: involvement of a cholinergic mechanism. *Brian Research*, **269**, 303–310.

Knigge, K. M., Bennett-Clarke, C. & Burchanowski, B. (1980) Relationships of some releasing-hormone-producing neuron systems to the ventricles of the brain. In *Endocrine Functions of the Brain* (ed. M. Motta). New York: Raven Press.

Koob, G. F. & Britton, K. T. (1984) Stress produces an increase in CRF in rat CSF. Abstracts of the Annual Meeting of the American College of Neuropsychopharmacology, San Juan, Puerto Rico.

Lundberg, J. M., Anggard, A., Fahrenkrug, J., Hokfelt, T. & Mutt, V. (1980) VIP in cholinergic neurons of exocrine glands; functional significance of coexisting neurotransmitters for vasodilation and secretion. *Proceedings of the National Academy of Sciences of the United States of America*, **77**, 1651–1655.

Nadi, N. S., Nurnberger; J. I., Jr & Gershon, E. S. (1984) Muscarinic cholinergic receptors on skin fibroblasts in familial affective disorders. *New England Journal of Medicine*, **311**, 252–230.

Nemeroff, C. B., Widerlov, E., Bissette, G., Walleus, H., Karlsson, I., Eklund, K., Kilts, C. D., Loosen, P. T. and Vale, W. (1984) Elevated concentrations of CSF CRF-like immunoreactivity in depressed patients. *Science*, 266:1342–1344.

Okuma, T., Kishomoto, A., & Inoue, K. (1973) Anti-manic and prophylactic effects of carbamazepine on manic-depressive psychosis. *Folia Psychiatrica et Neurologica Japonica*, **27**, 283–297.

Orth, D. N. (1979) Adrenocorticotropin hormone. In *Methods of Hormone Radioimmunoassay* (ed. B. M. Jaffe & H. R. Beheman). New York: Academic Press.

Patel, Y. C. & Reichlin, S. (1979) Somatostatin. In: *Methods of Hormone Radioimmunoassay* (eds B. M. Jaffe & H. R. Beheman). New York: Academic Press.

Pollay, M. (1979) Recent developments in CSF physiology. *Contemporary Neurosurgery*, **1**, 1–6.

Post, R. M., Gold, P. W., Rubinow, D. R., Ballenger, J. C., Bunney, W. E., Jr & Goodwin, F. K. (1982) Peptides in CSF of neuropsychiatric patients: an approach to CNS peptide function. *Life Sciences*, **31**, 1–15.

——, Uhde, T. W., Ballenger, J. C. & Squillace, K. M. (1983) Prophylactic efficacy of carbamazepine in manic-depressive illness. *American Journal of Psychiatry*, **140**, 1602–1604.

Reider, R. O. & Gershon, E. S. (1978), Genetic strategies in biological psychiatry. *Archives of General Psychiatry*, 35:866–873.

Roy, A., Pickar, D., Paul, S., Doran, A., Chrousos, G. & Gold, P. W., (in press). CSF CRH in depressed patients and normal controls. *American Journal of Psychiatry.*

Rubinow, D. R., Gold, P. W., Post, R. M., Ballenger, J. C., Cowdry, R., Bollinger, J. & Reichlin, S. (1983) CSF somatostatin in affective illness. *Archives General of Psychiatry*, **40**, 409–412.

Rubinow, D. R., Post, R. M., Gold, P. W., Ballenger, J. C. & Reichlin, S. (1985) Effects of carbamazepine on CSF somatostatin. *Psychopharmacology* **85**, 210–213.

Sachar, E. J., Hellman, L., Roffwarg, H. P., Halpern, F. S., Fukushima, K. K. & Gallagher, T. F. (1973), Disrupted 24 hour patterns of cortisol secretion in psychotic depression. *Archives General of Psychiatry*, **28**, 19–24.

Sitaram, N., Nurnberger, J. I., Jr., Gershon, E. S., & Gillin, J. C. (1980) Faster cholinergic REM sleep induction in euthymic patients with primary affective illness. *Science*, **208**, 200–202.

Wood, J. H. (1982) Neuroendocrinology of CSF: peptides, steroids and other hormones. *Neurosurgery*, **11**, 293–305.

Yau, W. M., Verdun, P. R. & Youthe, M. L. (1983), Neurotensin: a modulator of enteric cholinergic neurons in the guinea pig small intestine. *European Journal of Pharmacology*, **95**, 253–258.

Zerbe, R. L., Feverstein, G., Meyer, D. K. & Kopin, I. J. (1982) Cardiovascular, sympathetic and renin-angiotensin system responses to hemorrhage in vasopressin-deficient rats. *Endocrinology*, **111**, 608–613.

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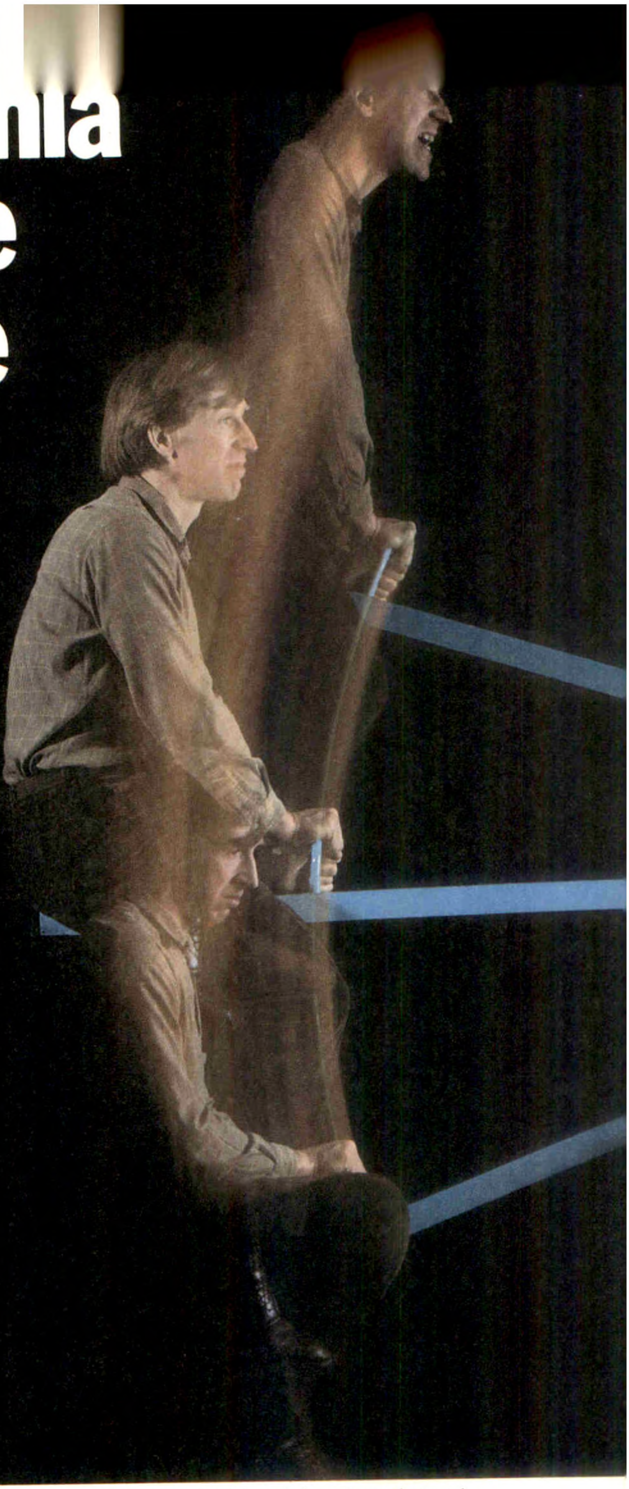
Schizophrenia therapy: the importance of balance



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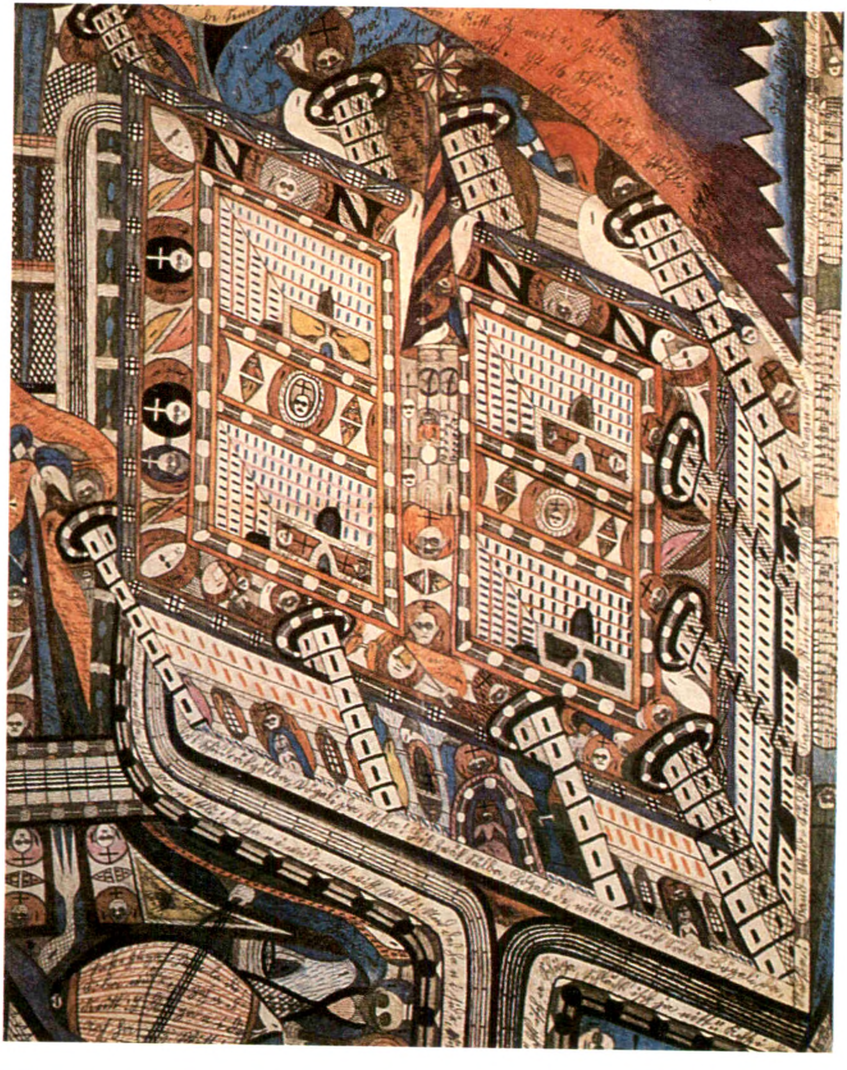
The picture below is one of a number that, we believe, give an insight into the schizophrenic mind and is reproduced with the kind permission of the Museum of Fine Art, Berne.



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The Psychological Effects of Service in British Antarctica:
A Study Using the General Health Questionnaire

J. BELL and P. H. GARTHWAITE

This study follows the psychological profile of a group of Antarctic explorers on a remote base in British Antarctica. Each month for 1 year the 60-item General Health Questionnaire (GHQ) was administered to the 12-man complement and the results compared to those of a control group. Three men resigned and left, 4 months later, at the end of winter. Their scoring showed marked differences from the others, including the highest scores in the severe depression factor scale. The GHQ was accepted by the men despite the intrusive threat it offered in such a small community; there was also a seasonal variation in scoring, with higher scores in the winter, and no evidence of long-term psychological sequelae in those who stayed.

The present Antarctic treaty lapses in 1991 and, as this date approaches, international interest in the continent is gathering momentum. Several studies report the occurrence of psychological disturbances in the Antarctic (Mullin & Connery, 1959; Strange & Klein, 1973; de Monchaux *et al*, 1979; Bell, 1984). That such morbidity should occur is not surprising: the Antarctic is a harsh, alien environment that is both isolated and dangerous. It is likely that as populations there increase, so will the numbers of psychological problems.

This paper reports a study that followed the psychological profile of a small group of workers in the Antarctic over the course of a year. The study was conducted at Rothera Station, a scientific base in British Antarctica, where one of the authors (J.B.) served as medical officer. In the study, the instrument used to quantify people's psychological state was the General Health Questionnaire (GHQ) designed by Goldberg (1972). This questionnaire has been used and validated in a variety of cultures and settings, including Jamaica (Harding, 1976), Australia (Worsley *et al*, 1978), Spain (Munoz *et al*, 1978) and Yugoslavia (Radovanovic & Eric, 1983), but to the writers' knowledge it has not previously been used in Antarctica.

The study started in April 1983, at the beginning of the Antarctic winter, and continued until March 1984. The GHQ was administered monthly to the 12 base members who were spending the winter on Rothera base and who were all volunteers. Three of them resigned prematurely in mid-winter and left the Antarctic, 7 months after the study started, when the base was relieved. They were replaced in the study by three members of the relief party.

The study's main aim was to examine the psychological changes that occur in men during their stay in Antarctica and also to examine the usefulness of the GHQ in an Antarctic setting. We paid particular attention to the three men who resigned prematurely, two of whom were diagnosed as psychologically disturbed. We found that their psychological states, as measured by GHQ scores, showed some marked differences from those of the people who did not resign. The seasons have a dominating influence on Antarctic life and so we also compared winter and summer GHQ scores. We found them to be generally higher in winter, when there was a greater incidence of sleep disturbance and general illness. However, if one excludes from consideration those people who were anxious to leave the Antarctic, our study then gives no indication that spending the winter there has any residual effect on a person's mental health in the following summer.

As part of the study, the GHQ was also administered to a control group of 21 men living in a conventional urban setting. Their scores were compared with those of the people in the Antarctic who did not resign but, perhaps surprisingly, few differences between these two groups were found.

It is clearly to an individual's detriment to remain in the Antarctic when suffering psychological morbidity. Not only are facilities for treating him on a small remote base inadequate, but the Antarctic itself is often the root cause of the psychological problems, and leaving often appears to serve as both treatment and cure. The rigorous climatic conditions of the Antarctic preclude evacuation during the winter. Therefore, one long-term aim of psychological studies in the Antarctic must be to develop methods of detecting very early signs of psychological disturbance and identify precursor factors of psychological morbidity. As questionnaires such as the GHQ are so straightforward to administer, their usefulness for predicting psychological morbidity is of obvious interest. Our study tentatively suggests that the GHQ may be of some value in this area.

Method

Rothera Base

Rothera Station is the main earth sciences base of the British Antarctic Survey. It lies just inside the Antarctic circle on Adelaide Island, off the west coast of the Antarctic peninsula, about 900 miles due south of Cape Horn. The year there divides into two distinct phases—winter and summer. The winter phase lasts about 7 months, beginning with the departure of the aeroplanes and relief ship at the end of March. Winter isolates the party of 12 men on the base and brings progressively shorter days, colder temperatures and more violent storms. The base is warm and comfortable, but there are essential tasks such as gathering ice-blocks for water, feeding the dogs and shifting fuel drums, which must be carried out regardless of the weather. Each man chooses his own pace of life, which varies from a busy routine of hobbies and work for some, to long periods alternating between the bunk and the bar for others. Generally, the group maintained good relationships with one another, although minor groupings did form and there were occasional signs of stress between individuals. (A full analysis of the social structure of the base is beyond the scope of this paper.)

The summer phase starts in late October when aeroplanes arrive, ferrying in men and equipment at spring relief. The days are long and the weather often perfect for many weeks. The base complement swells to over 40 and it is a time of busy activity, supporting scientific missions and summer bases scattered over the Antarctic peninsula. There is a consistently high pressure of work that only ends in March, when the ship arrives, autumn relief takes place and the winter phase starts again.

General Health Questionnaire

The standard 60-item version of the GHQ was used. The relationship between all 60 questions and factor scales is unclear from earlier studies. As suggested by Goldberg (1978), we discarded 30 of them when examining different symptoms and used the remainder to form six factor scales of five questions each: general illness, somatic symptoms, sleep disturbance, social dysfunction, anxiety and dysphoria, and severe depression. The scales proved to be of particular use in illuminating the different scoring patterns of individuals. Responses to the GHQ were evaluated using the Likert scoring system (a progressive scale that gives each column a score of 0, 1, 2 or 3).

The Antarctic group

During the study, the GHQ was administered to subjects at the beginning of each month. This was done early in the morning, immediately after other physiological and medical data had been gathered as part of a medical research programme. The setting for answering the questionnaire was made as consultative as possible. Subjects filled in the form at their own pace, alone and undisturbed. None of them said they found the routine time-consuming, stressful or overly intrusive. Some subjects were absent from base for parts of the summer and did not answer the GHQ every month; thus, there were 10 missing records from a total of 144. The ages of the 12 participants over the winter period ranged from 23 to 38, and averaged 28.6 years. The three subjects who resigned were aged 22, 26 and 32 and were replaced by men aged 34, 33 and 37 years.

The control group

To allow a comparison between the Antarctic group and men in urban surroundings, a control group of 21 men was drawn from staff at the medical school of Aberdeen. They were chosen to be of similar age to the Antarctic group (their ages ranged from 22 to 39) but the only other selection criterion was their willingness to participate in the study. Unlike the Antarctic group, who were all single, eight of them were married. The GHQ was administered individually to each of them once. They completed it in a setting that was quiet and consultative, although the location and time of day varied from person to person.

Clinical diagnosis

Two men were diagnosed as having a degree of psychological disorder that was of clinical significance. These were confirmed for their 'caseness' by submission of their case histories to four independent consultant psychiatrists in Aberdeen, who concurred with the diagnoses.

Statistical analysis

Unpaired t-tests were used to compare the Likert scores of different groups of people and paired t-tests used to compare the scores of a single group of people over two different time periods. The three people who resigned early did not form a homogeneous group, so their scores were examined individually. For each factor scale their scores were compared to the scores of the nine people who were with them in the Antarctic and did not resign. As a measure of the differences between an individual's score and the mean score of this group, we quote the number of standard deviations (s.d.s) by which the individual's score exceeds the mean. (Standard deviations were estimated from the scores of non-resigners.) If scores were normally distributed, then a difference of 2.43 s.d.s would equate to a significance level of $P=0.05$, 3.54 s.d.s to $P=0.01$ and 5.31 s.d.s to $P=0.001$. However, scores did not follow normal distributions precisely, so we used these values only as a guide (we will not quote significance levels when discussing individuals) and are cautious in the conclusions we draw.

TABLE I

Monthly Likert scores on the 60-item GHQ

Subject	*Apr*	*May*	*Jun*	*Jul*	*Aug*	*Sep*	*Oct*	*Nov*	*Dec*	*Jan*	*Feb*	*Mar*
A	**59**	**67**	**65**	**85**	**43**	**82**	**88**					
B	**73**	**60**	**49**	**58**	**46**	36	32					
C	26	24	21	25	23	22	22					
D	34	38	36	**41**	**47**	26	33	34	38	38	39	**45**
E	30	33	32	**67**	30	32	30	24	25	11	**54**	**42**
F	26	35	**41**	36	33	37	25	29	29	—	31	39
G	21	14	13	29	**46**	**44**	**46**	15	18	19	12	16
H	27	24	**54**	**52**	**42**	33	**47**	28	17	—	**59**	27
I	29	24	30	30	24	23	21	22	17	19	21	18
J	22	20	**69**	24	23	23	12	13	19	—	12	16
K	25	23	23	22	22	25	31	25	22	—	—	22
L	25	20	20	20	21	20	24	19	—	20	15	—
M								26	—	38	—	21
N								—	27	38	23	29
O								20	—	29	30	14

Scores above or equal to the threshold level of 40 are given in **bold type**.

Results

Monthly Likert scores on the 60-item GHQ are given for each individual in Table I. Goldberg (1972) suggested a cutting score of 40 and above as suggestive of psychological disturbance. This seemed a reasonable threshold to use in the Antarctic as, if one ignored isolated high scores, then no false positives were found and only one false negative. Scores above or equal to the threshold level of 40 are given in bold type in the table. Of the three men who resigned early, two of them (A and B) had many monthly scores above the threshold. A's were above it every month, while B's only fell below it in his last 2 months on the base, after his resignation had been accepted and he knew he would soon be leaving the Antarctic. Their high scores in April, at the start of winter, preceded their decisions to resign by 3 months. Of the other men, most had one or more scores above the threshold during the last two-thirds of the winter (June–October). Otherwise, the only time scores went above the threshold was in February and March when D, E and H, who were all scheduled to leave the Antarctic at the end of March, had at least one high score.

People resigning early

The men who resigned early (subjects A, B and C) participated in only the first 7 months of the study. Table II gives the average score over this period for each person on the six factor scales. The three resigners had the three highest scores on the severe depression scale ($P<0.005$). However, on the other scales their scores did not show the same degree of homogeneity, so we examined their scores individually.

Subject A was diagnosed as having an anxiety-depressive state which gradually developed over the winter. He was still receiving psychological treatment 1 year after

TABLE II

Average Likert scores over the winter period by factor scale

	Factor scale					
Subject	*General illness*	*Somatic symptoms*	*Sleep disturbance*	*Social dysfunction*	*Anxiety and dysphoria*	*Severe depression*
A	7.86	0.71	1.29	6.29	10.29	3.14
B	7.29	2.86	5.14	4.86	4.14	1.29
C	2.43	0.15	0	5.00	0.29	1.71
D	4.71	1.29	2.43	4.86	0.86	0
E	4.71	0	3.86	5.43	0.86	0
F	2.71	0.29	2.71	5.14	1.71	0.43
G	3.43	0	3.86	4.14	1.29	0
H	6.43	0.71	7.57	4.57	1.00	0
I	2.57	1.00	2.57	4.57	0	0
J	3.43	0.14	3.29	3.00	0.71	0.71
K	2.29	0.29	1.43	5.00	0	0
L	2.57	0	0.86	4.86	0	0

leaving the Antarctic. On four of the six factor scales, his average score was higher than that of anyone else. It was exceptionally high on the anxiety and dysphoria scale (15.7 s.d.s above the average of the nine winterers who did not resign) and on the severe depression scale (11.5 s.d.s above average). The other scales where his average score was the highest were general illness (3.1 s.d.s above average) and social dysfunction (2.3 s.d.s above average).

Subject B was not clinically disturbed but was clearly unhappy being in the Antarctic. He had particularly high scores on the scales of anxiety and dysphoria and somatic symptoms (5.6 and 5.1 s.d.s above average respectively). On the latter scale, his average was more than twice as large as anyone else's. His score was also noticeably above average on the scale severe depression (4.4 s.d.s above average).

Subject C was diagnosed as suffering a depressive neurosis with associated personality disorder and adjustment reaction. He was always somewhat distrustful of the questionnaire and the motives behind its administration. His state was chronic rather than acute (personal observation) and did not change much over the winter. His scores tended to be lower than average rather than higher; for example, he was the only person to show consistently no signs of sleep disturbances. It is possible he was not altogether honest in his answers to the questionnaire. Tennant (1977) found that false negatives with the GHQ were often defensive individuals who used denial as a means of coping. On the other hand, the chronicity of his state may have been the basis of his low scores: the limitations of the GHQ in chronic disorders have been recognised in other studies (Finlay-Jones & Murphy, 1979; Goodchild & Duncan-Jones, 1985). The only scale on which he did score highly was for severe depression (6.1 s.d.s above average).

Winter *vs* summer

We refer to the nine men who participated in the study over both the winter and summer as the 'winterers' group. For every factor scale their overall average was lower in the summer than in the winter. The proportional reduction was particularly large for the sleep disturbance scale where the average score in summer was less than half the value in winter. Paired *t*-tests showed there was some evidence that individuals' average scores were lower in summer than winter on the scales general illness ($t = 2.48$, $P < 0.05$) and sleep disturbances ($t = 2.70$, $P < 0.05$). On the other four scales, the differences between summer and winter scores were not significant ($t < 1.63$, $P > 0.10$), but the total scores over the full 60-item questionnaire were significantly lower in summer ($t = 3.05$, $P < 0.02$).

Residual effect of winter on subsequent scores in summer

The base personnel in the Antarctic over the summer consisted of the group of nine winterers and another group of three people who arrived at the start of summer. The scores over the summer of these two groups were compared but no evidence was found of a difference between them on any factor scale (unpaired *t*-tests were used to make these comparisons; the largest *t*-value found was 0.84, $P > 0.1$). Hence, there is no suggestion that wintering in the Antarctic has a residual effect on those who are content to stay.

Comparison of the control group

If one excludes the three people who resigned, then over the winter the average scores of the people in Antarctica were lower than those of the control group on half the factor scales and, over the summer, their average scores were lower than those of the control group on all factor scales apart from social dysfunction. However, the differences were all small and none was statistically significant ($t < 1.98$, $P > 0.05$).

Discussion

In our study, we found distinct differences between the three men who resigned prematurely and the men who did not. We first summarise our findings on the latter, who formed a more homogeneous group. In general, they had low scores in both summer and winter on the factor scales—somatic symptoms, anxiety and dysphoria, and severe depression. Their scores were much higher on the other factor scales—social dysfunction, general illness and sleep disturbance, and on the last two of these there is a detectable seasonal difference, with higher scores in winter. The difference is particularly marked on the sleep disturbance scale, where the winter average score was twice that of summer. However, over the summer, there was no apparent difference between the men who arrived in the Antarctic at the start of summer and those who had spent the winter there, suggesting that the Antarctic winter had no lasting effect on them. A comparison of the scores over the summer with those of a control group supports this view: on five of the six factor scales the group in the Antarctic had lower average scores than the control group, although the differences were small. Over the winter, their scores tended to be only slightly higher than the control group, although most had scores on the 60-item GHQ that were above the threshold of 39 in 1 or 2 months. Despite these occasional high scores, it seems that there is no evidence of long-term psychological disturbance in the initially well-adjusted.

These findings are essentially in agreement with those of other studies. Mullin (1960) found no evidence of adverse change in individuals with experience of the Antarctic; Law (1960) found that men were generally positive about their experience, variously stating that it had made them more tolerant, better controlled, more adaptable and more confident. It is worth noting that these studies examined people who had returned (or were on the point of returning) from the Antarctic, while our study suggests that most people are psychologically well for almost all the time they are there. Several people have also found a greater incidence of psychological disturbance over winter—the so-called 'wintering-over' syndrome (Strange & Klein, 1973). Other studies do not comment on the precise symptoms found in our factor scales but the prevalence of sleep disturbance is well documented (Lewis & Masterson, 1957; Natani & Shurley, 1973). We found that subjects were troubled by sleep disturbance in winter, but less so in summer.

These findings do not apply to the three people who resigned prematurely. Turning our attention to them, we find that their psychological health was disturbed by being in the Antarctic. One was clearly unhappy being there and the other two were clinically diagnosed as psychologically ill. One of these latter was still receiving psychiatric help a year after leaving the Antarctic. (Fortunately, this is a comparatively rare occurrence; other studies indicate that any harmful effects from living in the Antarctic usually disappear on return to a more normal environment [Taylor, 1978]). Whether their psychological difficulties were exaggerated by the 'wintering-over syndrome' is a matter for conjecture, but the fact that they resigned in winter rather than in summer is perhaps what one might expect.

The Likert scores of the three people who resigned show clear differences from those of the others. They have the three highest scores on the severe depression factor scale, as well as all the high scores on the somatic symptoms and anxiety and dysphoria scales. Thus, there is the suggestion that a high score on one or more of these scales is indicative that a person is suffering psychologically from being in the Antarctic. In contrast, high scores on the scales —general illness, social dysfunction or sleep disturbance appear to carry no such portent. Other studies have reported that depression (Strange & Klein, 1973) and somatic symptoms (Palmai, 1963) are a common problem in Antarctica. Our findings suggest that these symptoms (and also anxiety and dysphoria) are confined to a relatively small proportion of the people there and do not affect the majority.

The GHQ was not designed for use in the Antarctic but appears to have performed very well in that setting. It is true that only two of the three people who resigned had high scores on the 60-item GHQ, but they both had scores that were almost always well above the threshold of 39. Moreover, in the first month of the study, which was also the first month of winter, their scores of 59 and 73 were well above the threshold, while the next highest score of 34 was below it. This suggests that the GHQ might be useful for predicting at the onset of winter which people are likely to suffer psychologically during its course. In making predictions, the questions on the GHQ should perhaps not be scored equally. Our earlier findings suggest more emphasis should be placed on the factor scales—somatic symptoms, anxiety and dysphoria and, in particular, severe depression.

The study shows that questionnaires such as the GHQ can be used to follow the psychological profile of men while they are in the Antarctic. People proved willing to answer it and were prepared to do so repeatedly. This latter is important, since many individuals seem occasionally to give isolated high scores that have no long-term significance. In the main, people seem to have given honest answers to the questionnaire and these appear to reflect their psychological state. The number of people examined in the study is small but this will inevitably be the case with studies in the Antarctic. For progress to be made in understanding the psychological problems that occur there, other similar studies need to be conducted in an organised fashion and their results pooled to yield a more complete picture than a single study.

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References

BELL, J. A. (1984) The detection of psychological changes in antarctic explorers. *Scottish Medical Journal*, 30, 57.

DE MONCHAUX, C., DAVIS, A. & EDHOLM, O. G. (1979) Psychological studies in the Antarctic. *British Antarctic Survey Bulletin*, 48, 93–97.

FINLAY-JONES, R. A. & MURPHY, E. (1979) Severity of psychiatric disorder and the 30-item Gneral Health Questionnaire. *British Journal of Psychiatry*, 134, 609–616.

GOLDBERG, D. (1972) *The Detection of Psychiatric Illness by Questionnaire* (Maudsley Monographs) London: Oxford University Press.

—— (1978) *Manual of the General Health Questionnaire*. Windsor: NFER-Nelson.

GOODCHILD, M. E. & DUNCAN-JONES, P. (1985) Chronicity and the General Health Questionnaire. *British Journal of Psychiatry*, 146, 55–61.

HARDING, T. W. (1976) Validating a method of psychiatric case identification in Jamaica. *Bulletin of the World Health Organisation*, 54, 225–231.

LAW, P. (1960) Personality problems in Antarctica. *Medical Journal of Australia*, 1, 273–280.

LEWIS, H. E. & MASTERSON, J. P. (1957) Sleep and wakefulness in the Arctic. *Lancet*, 1, 1262–1266.

MULLIN, C. S. (1960) Some psychological aspects of living in Antarctica. *American Journal of Psychiatry*, 117, 323–325.

—— & CONNERY, H. J. M. (1959) Psychological study at an Antarctic IGY station. *United States Armed Forces Medical Journal*, 10, 323–325.

MUNOZ, P. E., VASQUEX, J. L., PASTRANA, E., RODRIGUEZ, F. & ONECA, C. (1978) Study of the validity of Goldberg's 60 item GHQ in its Spanish version. *Social Psychiatry*, 13, 99–104.

NATANI, K. & SHURLEY, J. T. (1973) Sleep research in the Antarctic: why? In *Polar Human Biology* (eds O. G. Edholm & E. K. E. Gunderson). London: Heineman.

PALMAI, G. (1963) Psychological observations on an isolated group in Antarctica. *British Journal of Psychiatry*, 109, 364–370.

RADOVANOVIC, Z. & ERIC, L. (1983) Validity of the General Health Questionnaire in a Yugoslav student population. *Psychological Medicine*, 13, 205–207.

STRANGE, R. E. & KLEIN, W. J. (1973) Emotional and social adjustment of recent US winter-over parties in isolated Antarctic stations. In *Polar Human Biology* (eds O. G. Edholm & E. K. E. Gunderson). London: Heineman.

TAYLOR, A. J. W. (1978) Summary of psychological effects of antarctic service. *New Zealand Antarctic Record*, 1, 31–34.

TENNANT, C. (1977) The General Health Questionnaire: a valid index of psychological impairment in Australian populations. *Medical Journal of Australia*, 2, 392–394.

WORSLEY, A., WALTERS, W. A. W. & WOOD, E. C. (1978) Responses of Australian patients with gynaecological disorders to the General Health Questionnaire: a factor analytic study. *Psychological Medicine*, 8, 131–138.

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(Accepted 8 April 1986)

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Influence of Emotional Distress among Supporters on the Outcome of Psychogeriatric Day Care

C. J. GILLEARD

From a sample of consecutive referrals for psychogeriatric day care, the influence of the supporters' emotional distress and self-reported strain, together with the number of problems presented by the patient, was examined in relation to outcome, 6–7 months after initial attendance. A higher level of problems and the failure to achieve any reduction in reported distress were both found to contribute to the breakdown of community care and to the subsequent institutionalisation of the patient. Day hospital care was associated with a significant reduction in emotional distress for the majority of relatives, although for relatives whose distress was not alleviated by their dependents' attendance, admission to long-term care did produce such a reduction.

One of the most frequently cited goals of psychogeriatric day hospitals is the relief of strain amongst relatives caring for the elderly patient (Greene & Timbury, 1979; Peace, 1982; Sands & Suzuki, 1983). The high level of emotional distress and strain experienced by supporting relatives of patients newly referred to psychogeriatric day care has been described in an earlier paper (Gilleard *et al*, 1984*a*). We found that much of this distress could be accounted directly for by the problems experienced by the supporter in caring for their mentally infirm relative. The present study was undertaken to examine the significance of the supporters' strain and distress in determining the outcome of psychogeriatric day care, and in particular the breakdown of the assisted community care afforded by day hospitals. The principal hypothesis to be tested is that the level of strain experienced by supporters contributes directly to the breakdown of community care, and that day hospital services are effective in keeping the elderly person at home when they serve to reduce that distress. Some support already exists for this hypothesis. Levin *et al* (1983) have demonstrated a significant relationship between distress levels in supporters of confused elderly people and subsequent institutionalisation of the dependents.

However, since supporters' strain and distress are closely linked to the nature and number of behaviour problems presented by their dependent (Greene *et al*, 1982; Gilleard *et al*, 1984*a*), it may be the problems themselves which principally determine the supporters' ability to continue to care. In this case, day hospital services will only be effective in keeping the elderly person at home when they serve to reduce the frequency of the problems presented to the supporter at home. Distress and strain will only appear to be influential in determining outcome in so far as they are reflecting the problems currently faced by supporters.

Retrospective studies (e.g. Isaacs, 1971) which identify family strain as a cause of institutional care are less helpful in answering such questions, since they cannot easily unravel the interdependent influences of strain and stressors. It seems desirable to examine the process of breakdown in care through prospective studies, where the relative influences of strains and stressors can be linked to future outcomes. The prospective study of community referrals for psychogeriatric day care from which the present data were obtained offered such an opportunity.

Method

Sample

The present sample was formed by 129 community referrals for psychogeriatric day hospital care to four such units in the Lothian region of Scotland. Each of the supporters was interviewed at the point when they had been offered day care for their relative, and again 3 and 6–7 months later. Details of these patient–supporter pairs have been described elsewhere (Gilleard *et al*, 1984*b*): briefly, all but 18 had a clinical diagnosis of dementia, all were living in the community and were looked after by a relative who either lived with them ($n = 90$) or visited at least three times per week ($n = 39$).

Measures

The interview and questionnaires were also described in the previous paper (Gilleard *et al*, 1984*b*). The chief measures analysed here were the 28-item Problem Checklist—covering the reported frequency of such behaviour problems as incontinence, mobility, disturbed behaviour, excessive demands, impairments in self-care and forgetfulness; the 30-item General Health Questionnaire (GHQ) (Goldberg, 1978) as an index of emotional distress; and a 13-item measure of strain derived from Machin's (1980) Strain Scale, modified to a three- rather than a five-point response format. The high internal reliability of the Problem Checklist total score has been reported in a separate study (Eagles & Gilleard, 1984), while the internal reliability and external validity of the Strain Scale have been documented by Machin. The minor modifications made to this scale (*vide* Appendix) do not appear to have reduced its internal consistency (internal reliability alpha coefficient = 0.87).

Outcome categories

To test the hypothesis, three categories of outcome were examined: those who attended day hospital consistently over the 6–7 month follow-up; those who ceased attendance before the end of the follow-up, but continued to be looked after at home by their relative; and those who attended for at least 3 months, but were admitted to long-term institutional care before the end of the follow-up period. Excluded from this analysis were those whose dependent died ($n = 13$), whose dependent entered institutional care less than 3 months after starting at the day hospital ($n = 19$), whose dependent never attended day hospital ($n = 4$), and an additional eight supporters who could not be interviewed at the 3-month follow-up.

Of the remaining 86 supporters eligible for this study, 36 had their dependent still attending day hospital (complete data, $n = 29$), 23 were looking after their dependent at home, but the patient had stopped attending (complete data, $n = 20$), and 26 had had their dependent admitted for long-term care (complete data, $n = 18$). The 19 supporters with some missing data had in most cases failed to respond to some of the items in the questionnaires.

Results

Table I presents the original scores of the total 'potential' sample, together with those from the sample with complete 3-month follow-up data. None of the means of this latter group deviated by more than 1 s.e. from those established for the original 'potential' sample, and it seems reasonable to conclude that the 'complete data' sample is not a significantly biased subsample.

The next stage in the analysis was to examine the differences between the three outcome groups in the level of problems faced, and in the strain and distress experienced during the first 3 months of day hospital attendance. Three univariate repeated measures analyses of variance were run using the SPSS MANOVA programme (SPSS, 1983). The means and standard deviations on both occasions are presented in Table II, together with the F values obtained for the group, occasion and group × occasion effects. No significant differences in the within-cell variances were observed.

The largest group differences were observed on the Problem Checklist scores. Those patients who were eventually admitted to long-term care presented a significantly higher number of problems throughout the first 3 months of day hospital attendance than did those who remained in the community. However, no group showed any evidence of a significant increase in the problems reported by their relatives. The results for the Strain Scale showed a similar, but less pronounced pattern. The supporters' GHQ scores showed a different pattern, with no outcome group differences but a highly significant decline over the first 3 months. It appears that day hospital care does reduce emotional distress amongst supporters, while having no obvious impact on the problems presented by the patients. The groups × occasions interaction term is not at a conventional level of significance ($P = 0.06$), but there is a clear suggestion that those supporters whose dependent will later be admitted for long-term care do not experience any reduction in their distress, unlike those whose dependent continues to attend day care for at least a further 3 or 4 months, whose GHQ score almost halved at the 3-month follow-up.

Since problems correlate closely with both measures of strain and distress—in this sample, the overall correlations for initial scores were 0.64 (Problem Checklist and Strain Scale) and 0.56 (Problem Checklist and GHQ)—a further analysis was conducted to examine the outcome group differences in the strain and distress scales, partialling out the influence of the problems reported by the supporters on both occasions. Two repeated measures analyses of covariance were performed on the GHQ and Strain Scale scores. The results of this analysis indicated that GHQ

TABLE I

Comparison of the initial questionnaire scores for the total 'eligible' sample and the sample with complete follow-up scores: means and standard deviations

	Original sample (n = 86)			*Complete data sample (n = 67)*		
Group	*Strain*	*GHQ*	*Problem*	*Strain*	*GHQ*	*Problem*
Attenders	13.2 (3.9)	11.6 (7.3)	18.7 (8.5)	13.7 (3.9)	13.1 (7.7)	17.9 (9.3)
Discharge	10.6 (5.5)	8.7 (9.1)	12.1 (10.7)	10.7 (5.4)	8.8 (9.4)	13.9 (10.9)
Institutionalised	12.8 (5.9)	11.1 (8.0)	24.5 (8.6)	13.7 (4.7)	11.2 (7.6)	24.7 (8.8)

TABLE II
Initial and 3-month scores on GHQ, Strain Scale and Problem Checklist by eventual outcome group

	Initial		*3 months*		*F values*		
	mean	*(s.d.)*	*mean*	*(s.d.)*	*Group (G)* *d.f.* = *2, 64*	*Occasion (O)* *d.f.* = *1, 64*	*G×O* *d.f.* = *2, 64*
Problems Checklist							
Attenders	17.9	(9.3)	19.1	(11.7)	7.8**	1.3	0.7
Discharged	13.8	(10.9)	13.5	(9.6)			
In care	24.7	(8.8)	27.3	(8.8)			
Strain Scale							
Attenders	13.7	(3.9)	13.2	(5.2)	9.2*	0.0	0.6
Discharged	10.7	(5.4)	10.6	(6.8)			
In care	13.7	(4.7)	14.8	(4.4)			
GHQ							
Attenders	13.2	(7.7)	7.1	(6.9)	0.9	15.5**	3.0
Discharged	8.8	(9.4)	6.6	(8.9)			
In care	11.2	(7.6)	10.1	(8.6)			

$^*P<0.05$; $^{**}P<0.01$.

scores showed an overall significant decline during the first 3 months ($F=19.7$; d.f. = 1, 63: $P<0.01$), with a now statistically significant interaction according to later outcome ($F=3.2$; d.f. = 2.63, $P<0.05$). There were no overall differences in GHQ scores by outcome group ($F=1.0$; d.f. = 2, 63; NS). As a result of partialling out the influence of the covariate, total problem score, no significant differences were evident between the three outcome groups on their adjusted scores on either initial or later interview point on the Strain Scale.

Finally, we examined changes over the full study period in the supporters' levels of distress, measured by the GHQ,

TABLE III
Changes in mean GHQ scores over the whole study period according to outcome circumstances and daycare attendance

	Initial *mean (s.d.)*	*3 months* *mean (s.d.)*	*6–7 months* *mean (s.d.)*
Attended throughout the study ($n=31$)	12.6 (7.7)	6.4 (6.9)	5.9 (8.2)
Discharged to own home after attendance ($n=17$)	7.6 (7.8)	4.9 (7.2)	5.2 (5.8)
Admitted to long-stay care after more than 3 months' attendance ($n=17$)	13.3 (6.4)	11.0 (8.3)	4.7 (6.4)
Admitted to long-stay care less than 3 months' attendance ($n=16$)	13.6 (8.1)	6.8 (8.7)	4.5 (7.3)

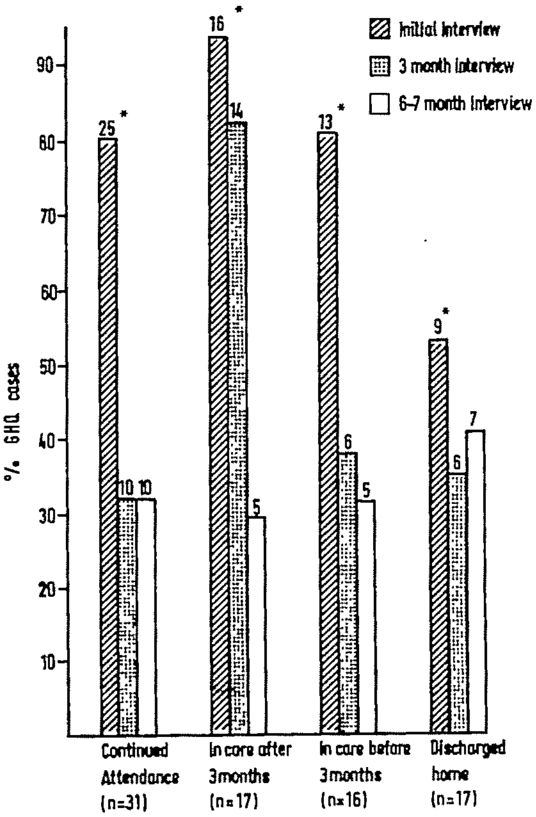

FIG. 1 Changes in relatives' GHQ caseness following day care and/or institutional admission of the patients. Numbers marked with an asterisk indicate the number of relatives scoring 5 and over on the GHQ (i.e. probable 'cases').

for all four outcome groups, including those ($n = 19$) whose dependent had been institutionalised. Since Strain Scale and Problem Checklist scores could only be obtained from supporters whose dependent was still at home, it was not appropriate to examine these variables over the full study period. Table III presents the means and standard deviations for each group on each occasion. Because some supporters had data missing from their final interview, the numbers in some groups are somewhat smaller than those in Table II, while other groups contain more subjects, since complete Problem Checklist and Strain Scale scores were not necessary for inclusion of subjects.

The results indicate significant differences in the development and remission of emotional distress amongst the supporters, depending upon the circumstances of their dependent. The main effect of outcome did not discriminate amongst the groups ($F = 1.28$; d.f. = 3, 77; NS), suggesting that the average level of supporters' distress was unrelated to the outcome situation of the patients. The occasions effect was significant ($F = 31.9$; d.f. = 2, 76, $P < 0.001$), with a large and significant overall decline during the first 3 months ($F = 62.4$; d.f. = 1, 77; $P < 0.001$) and a rather lower rate of decline over the last 3–4 months, though still significant ($F = 4.4$; d.f. = 1, 77; $P < 0.05$). A significant interaction term was observed ($F = 2.5$; d.f. = 6, 154; $P < 0.05$), although this was primarily due to the differential change during the first 3 months ($F = 3.3$; d.f. = 3, 77; $P < 0.05$), and not over the whole 6–7 months ($F = 1.6$; d.f. = 3, 77; NS).

If scores on the GHQ are treated as dichotomous rather than continuous variables, this effect can be seen even more clearly (Fig. 1). For this purpose, Goldberg's cut-off score on the GHQ of 5 and over was used to indicate 'probable caseness'. This sample of 81 supporters had, at the initial interview, an overall 'case' rate on the GHQ of 75%: this compares closely with 73.5% in the total ($n = 129$) sample. In every outcome group of supporters, apart from those whose dependent was later to be admitted to long-term care, the proportion of GHQ-defined cases dropped to less than 38% (i.e. halved) after the first 3 months; yet, amongst this latter group, the proportion of cases had hardly dropped at all—from 94 to 82%. By the time of the final follow-up, less than one-third of the relatives of patients still attending day care and a similar proportion of relatives whose dependents were in long-term care were still scoring as cases on the GHQ.

Discussion

While day hospital attendance produced no short-term reductions in the number of problems faced by the supporting relatives, it does seem to have significantly reduced their reported level of distress. Yet the 'strains' of caring remained unchanged. This differential response on the GHQ and Strain Scales may be understandable if the Strain Scale is seen as measuring the stressful experiences of caring, while the GHQ reflects the distress reaction within the carer. Thus, the former questionnaire asks "are you worried or depressed by, or unable to cope with your relative?", while the latter asks "are you worried, or depressed, or not able to cope?" The Strain Scale may therefore be an insensitive instrument to measure changes in the emotional effect of caring for an elderly mentally infirm relative.

Day hospital care and the relief it affords alleviated the more reactive emotional distress of the supporters; however, by not changing the problems presented by their dependents, the experience of caring may remain as much a strain as ever. The significance of reduced levels of distress appears to be that it permits carers to continue giving care, while the converse—a failure to reduce the supporter's distress—seems to predict the breakdown of care and resulting institutionalisation. This effect is quite independent of the level of problems faced by supporters. Of course, the level of problems itself has been demonstrated to influence outcome: those patients who present the most problems to their supporter are more likely to be institutionalised. But there is no evidence that an increase in the number of problems leads to the breakdown of care and subsequent institutionalisation.

Such findings offer considerable optimism concerning the potential of psychogeriatric day hospitals to contribute to the community care of the elderly mentally infirm. If institutionalisation was preventable only by decreasing the number of problems presented by the patient at home, then, to be effective in sustaining family care, day hospitals would have to both prevent the development of new and reduce the frequency of existing behavioural deficits and disturbances in a primarily dementing patient group—a task that is clearly daunting. That a reduction in distress, even in the absence of change in problems presented by the patient, is predictive of sustained community care suggests a much more attainable goal. Research directed at identifying those elements of care services most effective in reducing supporters' distress seems well worth pursuing, not only for the inherent value it offers the individual families, but also because it seems likely to lead to a reduction in the demand for the limited institutional resources available for the elderly mentally infirm, without adding to the burden on the family.

The findings which consider all four outcome groups also indicate that institutionalisation of the elderly mentally infirm does produce a significant reduction in level of emotional distress amongst care-giving relatives, but this effect is no greater than that achieved for more than two-thirds of those relatives whose dependent attended day hospital services for 3 months. In addition to investigations the means of reducing distress, it can be argued that we also need to know more about those whose distress day care fails to relieve, and whether or not for this group any real alternative exists to their relatives' admission into long-term care.

Acknowledgement

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Appendix

Strain Scale

Responses to each item are: 'a great deal of the time', 'sometimes' and 'never'. Scoring for item 9 is reversed.

(1) Do you fear accidents or dangers concerning the elderly person (e.g. fire, gas, falling over etc.)?
(2) Do you ever feel embarrassed by the elderly person in any way?
(3) Is your sleep ever interrupted by the elderly person?
(4) How often do you feel it is difficult to cope with the situation you are in and in particular with the elderly person?
(5) Do you ever get depressed about the situations?
(6) How much do you worry about the elderly person?
(7) Has your household routine been upset in caring for the elderly relative?
(8) Do you feel frustrated with your situation?
(9) Do you get any pleasure from caring for the elderly person?
(10) Do the problems of caring prevent you from getting away on holiday?
(11) Has your standard of living been affected in any way due to the necessity of caring for your elderly relative?
(12) Would you say that your health had suffered from looking after your relative?
(13) Do you find the demand for companionship and attention from the elderly person gets too much for you?

References

Eagles, J. M. & Gilleard, C. J. (1984) The functions and effectiveness of a day hospital for the demented elderly. *Health Bulletin (Edinburgh)*, **42**, 87–91.

Gilleard, C. J., Belford, H., Gilleard, E., Gledhill, K. & Whittick, J. E. (1984*a*) Emotional distress amongst the supporters of the elderly infirm. *British Journal of Psychiatry*, **145**, 172–177.

——, Gilleard, E. & Whittick, J. E. (1984*b*) Impact of psychogeriatric day hospital care on the patient's family. *British Journal of Psychiatry*, **145**, 487–492.

Greene, J. G. & Timbury, G. C. (1979) A geriatric psychiatry day hospital service: a five year review. *Age and Ageing*, **8**, 49–53.

——, Smith, R., Gardiner, M. & Timbury, G. C. (1982) Measuring behavioural disturbance of elderly demented patients in the community and its effects on relatives: a factor analytic study. *Age and Ageing*, **11**, 121–126.

Goldberg, D. P. (1978) *Manual for the General Health Questionnaire*. Windsor: NFER-Nelson.

Issacs, B. (1971) Geriatric patients: do their families care? *British Medical Journal*, **iv**, 282–286.

Levin, E., Sinclair, I. & Gorbach, P. (1983) *The Supporter of Confused Elderly Persons at Home. Final Report to DHSS* vol. 1. London: National Institute of Social Work Research Unit.

Machin, E. (1980) A survey of the behaviour of the elderly and their supporters at home. MSc Thesis, Department of Psychology, University of Birmingham.

Peace, S. M. (1982) Review of day hospital provision in psychogeriatrics. *Health Trends*, **14**, 92–95.

Sands, D. & Suzuki, T. (1983) Adult daycare for Alzheimer's patients and their families. *The Gerontologist*, **23**, 21–23.

SPSS (1983) *SPSS 'X'. User's Guide*. New York: McGraw Hill.

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Plasma Levels of β-Endorphin in Depressed Patients
with and without Pain

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We studied 27 patients who complained of pain, for which no organic basis could be found, and 11 patients without pain. All patients were depressed according to clinical diagnosis and Beck Depression Inventory (BDI) scores. Plasma β-endorphin levels were found not to differ between the two groups or across other variables. The patients who complained of pain had significantly higher scores on the BDI.

Many patients complain of pain while suffering from depression (Merskey & Spear, 1967). However, investigations have demonstrated that some patients with affective disorders have diminished sensitivity to experimental pain stimuli (Davis, 1983). Psychological explanations were popular in the past for this association of altered pain sensitivity and affective illness but in recent years hypotheses based on biochemical research have been proposed. For example, Garvey *et al* (1983) showed that a group of patients who suffered from headaches during depressive episodes were younger, had more somatic complaints and experienced more hypersomnia than other depressives. They postulate that this association between depressive headaches and hypersomnia and somatic complaints might involve serotonin abnormalities. Since their discovery in 1975, much research has focussed on the endogenous opiates and their involvement in pain and mood disturbances.

Almay *et al* (1978) showed that in patients with pain, CSF β-endorphin levels correlated with patients' self-ratings of depression. Recently Atkinson *et al* (1983) studied plasma measures of β-endorphin in Chronic Pain Syndrome and psychiatric subjects. These authors found that the Chronic Pain patients demonstrated elevated concentrations of plasma β-endorphin immunoreactivity.

While it is possible that the endogenous opiates play a role in pain perception, it is still not clear if such a role can be identified with respect to affective illness. Some work has been successful in relating endorphins to the various other biological phenomena which are thought to be associated with depressive illness. In particular, studies have demonstrated a relationship with HPA (hypothalamic-pituitary-adrenal) axis activity. Rubinow *et al* (1981) found that CSF opioid activity related significantly to mean urinary-free cortisol in depressed patients but did not do so in controls. Cohen *et al* (1984) observed a relationship between levels of plasma β-endorphin imunoreactivity and levels of plasma cortisol in patients with major depression but not in those with minor depression or in controls. It has been shown that β-endorphin and ACTH are stored in the same secretory granules of the pituitary and are released together in animals during stress (Rossier *et al*, 1980). Mains *et al* (1977) showed that β-endorphin shared the same precursor molecule (pro-opiocortin) with ACTH. Plasma ACTH suppression after dexamethasone is accompanied by plasma opioid suppression (Kriegar & Liotta, 1979).

In spite of the emerging literature showing involvement of the HPA axis in depressive illness, research has not demonstrated a clear abnormality of endorphin measures in depression. Initial studies on CSF performed by Terenius *et al* (1977) showed elevated CSF opiate activity in depression; Naber *et al* (1981) could not replicate this and similarly Gerner & Sharp (1982) found no CSF endorphin abnormality in depressed patients. Plasma studies have also produced contrasting results. Brambilla *et al* (1981) found plasma β-endorphin levels which were significantly higher in patients with depression of both primary and secondary types. Risch (1982) also found significantly higher plasma β-endorphin immunoreactivity in a group of depressed patients meeting the Research Design Criteria (RDC) for Major Depressive Disorder or Schizo-affective Disorder–Depressed, than in a group of matched controls. Alexopoulos *et al* (1983) found no difference from normals. In a study by Cohen *et al* (1984), the plasma β-endorphin immunoreactivity was significantly lower in patients with non-major depression than in those with major depression or in normal subjects.

In the present study, the β-endorphin immunoreactivity was measured in a group of patients suffering from either Endogenous Depression (ICD

296.1) or Neurotic Depression (300.4). Also included were three patients who had a different ICD classification but were significantly depressed. Depressed mood was measured using the Beck Depression Inventory (Beck *et al*, 1961). Patients were divided into two groups: those complaining of pain and depression and those who were depressed but without pain.

Method

Subjects

Thirty-eight patients were studied. They were either in-patients or out-patients of the Professorial Psychiatric Unit at the Cork Regional Hospital. Twenty-seven patients were suffering from pain for which no organic basis could be found by appropriate investigation; they were also judged to be depressed by the referring psychiatrist. These patients complained mainly of headaches (17 patients), pains in the face and neck (2), abdomen (4), legs (2) and others (3). Their psychiatric diagnoses were as follows: neurotic depression (22) and endogenous depression (5).

A smaller group of 11 patients who stated that they had no pain were referred to the study. Their psychiatric diagnoses were as follows: neurotic depression (5), endogenous depression (3), personality disorder (1), anxiety state (1) and anorexia (1).

Twenty-two patients were receiving antidepressant medication at the time of the study.

Materials

On referral each patient was administered the BDI. Those who complained of pain were also administered a Visual Analogue Scale for pain (Ohnhaus & Adler, 1975). Blood was taken from patients on this occasion for plasma β-endorphin estimation. All samples were taken between 10–11.30 am. Blood (10 ml) was mixed with lithium heparin in a polypropylene tube which was stored on ice (no more than 15 min) until plasma was obtained by centrifugation at 3000 rpm for 10 min at 4°C. Samples were then frozen at −20°C.

β-Endorphin in plasma was measured by the RIA procedure marketed by the Immunonuclear Corporation. The specificity of the assay for β-endorphin was assured by removing cross-reacting β-lipotropin by affinity chromatography using a sepharose anti-β-endorphin complex. Only the elution peak containing β-endorphin was further processed. The radioimmunoassay involved overnight incubation of extracted samples with rabbit anti-β-endorphin and ^{125}I β-endorphin and phase separation was accomplished using goat anti-rabbit precipitating complex.

Statistics

For comparison of unpaired measurements as applied to unequal group sizes, the Mann Whitney U Test was used. Spearman's Rank Correlation Coefficient was used to assess correlation between two variables.

TABLE I

Patients with pain

E^1	n^2	*Sex*	*Age*	*Diagnosis (ICD.9)*	*Pain*	*Beck*^{3}	*T*
<5.0	11	M5	Md56	300.4=10	Md50	Md27	N6
		F6	R20–70	296.1=1	R24–70	R12–42	T5
5.0–20.0	14	M5	Md53	300.4=11	Md51	Md36	N6
		F9	R30–71	296.1=3	R15–100	R17–53	T8
30.0	1	F1	54	296.1	100	15	T1
72.0	1	F1	43	300.4	48	32	N1

1. Beta-endorphin concentration (pmol/litre)
2. Number of patients
3. On treatment with antidepressants

Md. Median value
R. range of values

TABLE II

Patients without pain

E^1	n^2	*Sex*	*Age*	*Diagnosis (ICD.9)*	*Beck*	T^3
<5.0	4	M2	Md43	300.4=2	Md20	T4
		F2	R16–51	300.0=1 301.5=1	R16–27	N0
5.0–20.0	7	M2	Md42	300.4=4	Md20	T4
		F5	R19–64	296.1=2 307.1=1	R12–31	N3

1. β-endorphin concentration (pmol/litre)
2. Number of patients
3. On treatment with antidepressants

Md.Median value
R.Range of values

Results

See Table I and Table II.

Median β-endorphin level for group with pain = 6.0 pmol/litre. Median β-endorphin level for group without pain = 6.0 pmol/litre.

The results were analysed to see if there was any relationship between the demographic variables, sex and age, and endorphin levels, pain rating or BDI score. No statistically significant difference was found ($P>0.05$). Comparing those who were receiving antidepressant medication with those who were not, there was no significant difference in pain rating, endorphin level or BDI score ($P>0.05$). In those patients who complained of pain, no connection between pain scores and β-endorphin levels was found. When compared to the group of patients without pain, no significant difference in endorphin levels was found. However, a statistically significant difference in BDI scores was detected ($P<0.05$).

When all the BDI scores were compared with β-endorphin levels no significant correlation was found. When divided into diagnostic categories, i.e. Endogenous Depression *vs* Neurotic Depression and other Neuroses, no significant difference in β-endorphin levels was found.

Two of our patients had high outlying plasma β-endorphin values. One was an in-patient and we subsequently studied

TABLE III
Effect of ECT on plasma β-endorphin

	β-Endorphin concentration (pmol/litre)		
	1st ECT Treatment	*2nd ECT Treatment*	*3rd ECT Treatment*
Before ECT	24	21	27
10 min after end of convulsion	>80	>80	60
4 h after convulsion	43	36	23

her plasma β-endorphin level while she had ECT (see Table III). The other patient was requested to have further studies but declined.

Discussion

Cohen *et al* (1984) found that plasma β-endorphin immunoreactivity was significantly lower in patients with non-major depression than in those with major depression or in normal subjects. In contrast to this, in our study of depressed patients without any diagnosed physical lesion, we found that plasma β-endorphin showed no difference in the subtypes of depression. However, our study group consisted predominantly of neurotic patients. Another difference was that in Cohen's study β-endorphin was not separated from β-lipotropin and the ratio between them was not determined. Brambilla *et al* (1981) found in their study of chronic schizophrenics that β-lipotropin and β-endorphin levels did not always increase in parallel and suggest that measuring both together might be misleading. Atkinson *et al* (1983), in a study of β-endorphin/β-lipotropin-like immunoreactivity in chronic pain syndrome and psychiatric subjects, found that pain patients had significantly higher β-endorphin/β-lipotropin-like immunoreactivity than psychiatric patients even though there was no significant difference in severity of depressive symptomatology. They state that "β-endorphin may reflect pain, stress or mood disturbance as a state or trait marker". Our results indicate no difference between depressed patients reporting pain and those without pain with regard to β-endorphin levels. There are some recognised limitations in using the Visual Analogue Scale to assess the difference in pain state between patients. In our study, β-endorphin level did not correlate with degree of pain, as measured on this scale. In a recent report, Willer *et al* (1984) present data which suggest that there is little correlation between plasma β-endorphin concentration and pain control in humans. The two patients who had high outlying values in our study (*viz* 72 and 30 pmol/litre) both complained of pain which is further evidence that plasma β-endorphin concentration and pain state are poorly correlated. We did find that the pain group scored higher on the BDI.

Much effort and research has been concentrated on β-endorphin over the past 10 years. A possible role has been elaborated for endorphins in psychiatric illness and in pain perception and in their association. If the state of β-endorphin metabolism is to be used as a viable state or trait marker for depressive illness it can be assumed that plasma would be the body fluid used. While presently it is accepted that CSF studies give a better indication of central β-endorphin metabolism (Woolf & Wall, 1983) there are serious limitations of an ethical nature to this line of research. Measures of β-endorphin may help to illuminate the role of HPA axis activity in depressive illness. However, our own study would indicate that a unitary measure of endorphin activity alone may not be sufficient to distinguish between subgroups of patients. In particular, we do not believe that β-endorphin levels can distinguish between those depressed patients with pain and those without pain.

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References

Alexopoulos, C. E., Inturrisi, C. E., Lipman, R., Frances, R., Haycox, J., Dougherty, J. H. & Rossier, J. (1983) Plasma immunoreactive beta-endorphin levels in depression. *Archives of General Psychiatry*, 40, 181–183.

Almay, B. G. L., Johanssen, F., Von Knorring, L., Terenius, L. & Wahlstrom, A. (1978) Endorphins in chronic pain: 1. Differences in CSF endorphin levels between organic and psychogenic pain syndrome. *Pain*, 5, 153–162.

Atkinson, J. M. Kremer, E. F., Risch, S. C., Morgan, C. D., Azad, R. F., Ehlers, L. L. & Bloom, F. E. (1983) Plasma measures of beta-endorphin/beta-lipotropin-like immunoreactivity in chronic pain syndrome and psychiatric subjects. *Psychiatry Research*, 9, 319–327.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. E. & Erbaugh, J. K. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

Brambilla, F., Genazzani, A. R., Facchinetti, F., Parrini, D., Petraglia, F., Sacchetti, E., Scarone, S., Guastalla, A. & D'Antona, N (1981) Beta-endorphin and beta-lipotropin plasma levels in chronic schizophrenia, primary affective disorders and secondary affective disorders. *Psychoendocrinology*, 6, 321–330.

Cohen, M. R., Pickar, D., Extein, I., Gold, M. S. & Sweeney, D. R. (1984) Plasma cortisol and beta-endorphin immunoreactivity in nonmajor and major depression. *American Journal of Psychiatry*, 141, 628–632.

Davis, G. C. (1983) Endorphins and pain. *Psychiatric Clinics of North America*, 6, 473–487.

GARVEY, M. J., SCHAFFER, C. B. & TUASON, V. B. (1983) Relationship of headaches to depression. *British Journal of Psychiatry*, **143**, 544–547.

GERNER, R. H. & SHARP, B. (1982) CSF beta-endorphin immunoreactivity in normal, schizophrenic, depressed, manic and anorexic subjects. *Brain Research*, **237**, 244–247.

KRIEGAR, D. T. & LIOTTA, A. S. (1979) Pituitary hormones in brain. Where, how and why? *Science*, **205**, 366–372.

MAINS, R. E., EIPPER, B. A. & LING, N. (1977) Common precursor to corticotropins and endorphins. *Proceedings of the National Academy of Science USA*, **74**, 3014–3018.

MERSKEY, H. & SPEAR, F. G. (1967) *Pain, Psychological and Psychiatric Aspects*. London: Bailliere, Tindall & Cassell.

NABER, D., PICKAR, D., POST, R. M., VAN KAMMEN, D. P., WATERS, R. N., BALLENGER, J. C., GOODWIN, F. K. & BUNNEY, W. E. (1981) Endogenous opioid activity and beta-endorphin immunoreactivity in CSF of psychiatric patients and normal volunteers. *American Journal of Psychiatry*, **138**, 1457–1462.

OHNHAUS, E. E. & ADLER, R. (1975) Methodological problems in the measurement of pain: a comparison between the verbal rating scale and the visual analogue scale. *Pain*, **4**, 375–378.

RISCH, S. C. (1982) Beta-endorphin hypersecretion in depression: possible cholinergic mechanisms. *Biological Psychiatry*, **17**, 1071–1079.

ROSSIER, J., FRENCH, E., GUILLERIN, P. *et al* (1980) On the mechanisms of the simultaneous release of immunoreactive beta-endorphin, ACTH and prolactin by stress. In *Neural Peptides and Neuronal Communications* (eds E. Costa & M. Trabuchi). New York: Raven Press.

RUBINOW, D. R., POST, R. M., PICKAR, D. *et al* (1981) Relationship between urinary-free cortisol and CSF opioid binding activity in depressed patients and normal volunteers. *Psychiatry Research*, **5**, 87–93.

TERENIUS, L., WAHLSTROM, A. & AGREN, J. (1977) Naloxone treatment in depression: clinical observations and effects on CSF and endorphins and monoamine metabolites. *Psychopharmacology*, **54**, 31–33.

WILLER, J. C., SHERY-SHU, L., BERTAGNA, X. & GIRARD, F. (1984) Pituitary beta endorphin not involved in pain control in some patho-physiological conditions. *Lancet*, **ii**, 295–296.

WOOLF, C. J. & WALL, P. D. (1983) Endogenous opioid peptides and pain mechanisms: a complex relationship. *Nature*, **306**, 739–740.

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Reliability of the Psychosocial Axes of DSM-III in an Adolescent Population

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Three psychosocial variables were rated independently by two clinicians for 140 adolescent referrals to a psychiatric unit. Overall, intraclass correlations for DSM-III Axis IV, Axis V and a psychosocial adversity index (PSAI) were respectively 0.44, 0.58 and 0.68. The main source of unreliability is probably the operational definitions, rather than their application by clinicians; indices similar to the PSAI may be preferable.

Over the last decade, there has been a revival of interest in issues surrounding psychiatric diagnosis and classification: In the US, this resulted in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders*: DSM-III (American Psychiatric Association, 1980), while some European countries have produced a unified system of recording clinical data (AMDP) (Joint WHO/ADAMHA Project, 1983). Preliminary work is now being done to prepare the way for the mental disorders section of the tenth edition of the International Classification of Diseases (ICD-10). The most fundamental decision for ICD-10 is whether it is to be a multi-category single axis system like ICD-9, or a multi-axial system like DSM-III and (presumably) its successors. Against this background, it is surprising how little empirical work has been carried out on one of the most novel aspects of DSM-III—the psychosocial axes: Axis IV (severity of stressors) and Axis V (level of adaptive functioning).

The inclusion of separate axes for the recording of clinical ratings of psychosocial factors is a departure from previous practice which is at least as unusual as some of the other aspects of DSM-III that have received much more critical attention. Though there is agreement that psychosocial stress and premorbid functioning are particularly important variables, which may have aetiological or prognostic value (Rutter & Shaffer, 1980), it does not necessarily follow that their formal definition and incorporation as separate axes in each diagnosis is the best way to ensure that this information will be considered. Nor does it ensure that the data will be recorded, since 19% of Axis IV ratings and 11% of Axis V ratings were 'unspecified' in the 1111 multiaxial DSM-III diagnoses reported by Mezzich *et al* (1982).

In any case, the agreement about the importance of psychosocial factors does not extend to their operational definitions in DSM-III; Rutter & Shaffer (1980) indicated some of the problems with the current definitions, and Kendell (1983) predicted that 'radical alterations' would be needed if Axes IV and V were to survive. The widespread acceptance of DSM-III (Spitzer, 1984) has encouraged many specialty groups to develop supplementary axes relevant to their own needs, and some of these may be of sufficient general importance to be considered for inclusion in future multiaxial systems. Given that clinicians do not have unlimited time to record all the information that might be coded as part of a psychiatric diagnosis, the role of supplementary axes in general, and Axes IV and V in particular, will probably be a more contentious issue in future than it was prior to DSM-III. In the process, psychosocial factors might be relegated to a level of secondary information which it is valuable to have, but not necessarily in a standard format for all cases. In the attempt to produce operational definitions which would be applicable to all patients and usable by any clinician, the creators of Axes IV and V may have been obliged to over-simplify the concepts. It may therefore be better to recognise the many difficulties that exist in assessing psychosocial factors (Rutter *et al*, 1975; Cleary, 1981; Paykel, 1983), and leave their assessment to individual clinicians, rather than diverting clinical and research effort into inadequate measures of important variables. On the other hand, it would be unwise to abandon the experiment with these Axes before their usefulness has fully been studied.

In the absence of simple and widely accepted measures of psychosocial stress and adaptive functioning, the recorded opinion of an experienced clinician is by no means a bad choice, provided it is not arbitrarily constrained by premature attempts at operationalisation. In practice, if Axes IV and V survive at all, it is likely to be in some such form, if only as a compromise between the information one would like to have, and what busy clinicians have time to assess and record. This paper attempts to answer some of the questions which arise within that context.

The first question is whether or not judges show reasonable agreement in their ratings on Axes IV and V for adolescents. The data on children and adolescents of the DSM-III field trials (Spitzer & Forman, 1979; Williams & Spitzer, 1980) suggest that this is not the case (intraclass reliability coefficients [ICRC] for Axes IV and V were respectively 0.59 and 0.52), but the methodological problems of those trials (Rutter & Shaffer, 1980) leave open the possibility that greater reliability might be obtained in other circumstances. Hence, the initial aim of this present work was to test the level of inter-rater agreement obtainable over a period of time between two judges with considerable prior experience of DSM-III, and using a standard set of recorded information. Unfortunately, the most similar study on the reliability of DSM-III in adolescents (Strober *et al*, 1981) used a draft which did not include these axes, and the case-history study of Russell *et al* (1979) also used a draft of DSM-III in which the psychosocial axes were not in their final form, so that their average figures of 63% agreement for 'Axis IV' (presence or absence of 28 stressors) and 64% agreement for 'Axis V' (a four-point scale of *present* impairment of adaptive functioning) have no direct bearing on these axes as they appear in the final version of DSM-III. Nevertheless, these findings agree in suggesting that assessment of psychosocial factors in young patients is not very reliable.

This raises an issue which is often passed over in reliability studies—the operational definition of 'reliability' itself. Even if there were no other differences between the DSM-III field trials and the study of Russell *et al* (1979), it would be impossible to compare them directly because they use different measures: intraclass correlations in the former, and percentage agreement in the latter. Mitchell (1979) showed that different definitions of reliability measure different *kinds* of reliability and we have therefore calculated a variety of measures for our data.

Another aim of the study was to explore beyond the confines of the present definitions of Axes IV and V, in the hope of finding ways in which such clinical ratings might be improved: the most direct way is to have an alternative measure. Since Axis IV has received most criticism, a modified version of the adversity index described by Shaffer *et al* (1975) was included for comparison.

Method

All adolescents referred to Rivendell Adolescent Unit for assessment from March 1983 to February 1984 were considered. A case was selected only if all of the following data were available: (1) information from the referring agency; (2) Rivendell Questionnaire, covering demographic, medical, developmental and school data (copy available on request); (3) Child Behavior Checklist for ages 4–16 (Achenbach & Edelbrock, 1979), completed by the parents; (4) Youth Self-Report for ages 11–18, completed by the adolescent (Achenbach & Edelbrock, 1983); (5) a 38-item questionnaire focusing on behaviours relevant to the assessment of attention-deficit disorders, completed by the parents (copy available on request); (6) Parental Bonding Instrument (Parker *et al*, 1979), completed by the adolescent; and (7) assessment report based on interview/s with the family and the adolescent by a psychiatrist or clinical psychologist and a social worker.

When information was complete—usually 2 or 3 weeks after initial assessment—the same two clinicians (J.M.R. and J.M.P.), with 3 years' prior experience in the use of DSM-III, made independent diagnoses on the five DSM-III axes and assigned a rating on the Psychosocial Adversity Index (PSAI). Once the independent DSM-III diagnoses were made, the cases in which there was disagreement on Axis I (clinical psychiatric diagnosis) were reviewed by the same two clinicians, and a diagnosis made by consensus. For the purposes of some analyses, these consensus diagnoses were grouped into three broad categories: (1) 'conduct', which includes conduct disorders, attention-deficit disorders and adjustment disorders with disturbance of conduct; (2) 'emotional', comprising affective disorders, adjustment disorders with depressed mood, anxiety disorders, and adjustment disorders with anxious mood; (3) 'other', including all other diagnoses. For similar reasons, cases in which one of the raters had participated in the assessment interview (seen) were distinguished from those where neither judge had been involved (not seen), there were no cases in which both raters had interviewed the family. Diagnoses on Axis II (personality disorders and specific developmental disorders) and Axis III (physical disorders) are not considered here.

The rating of severity of psychosocial stressors (Axis IV) was made according to the procedures in the DSM-III manual. The key features of that rating (on a seven-point scale from 1 (no apparent stressor) to 7 (catastrophic) are that judgement should be based on the hypothetical stress which an 'average' adolescent in similar circumstances and with similar sociocultural values would experience from the particular psychosocial stressor(s); that stressors occurring in the 12 months prior to the onset of the current disorder should be considered; and that ratings "should reflect the summed influence of all the psychosocial stressors". These requirements are difficult to apply, but no attempt was made to define them further, since our aim was to use DSM-III as it stands.

Ratings on Axis V are supposed to reflect the highest level of adaptive functioning during the previous year, taking into consideration the patient's performance at work (or school), at home and in the use of leisure time. Ratings may range from 1 (superior) to 7 (grossly impaired).

The PSAI attempts to measure the more chronic, enduring stressors or 'adversity'. This instrument is a simplified version of a scale developed by Shaffer *et al* (1975), in which patients are given one point for each of the following items: (a) broken home or chronic marital disharmony, (b) contact with two or more social agencies, (c) psychiatric disorder or chronic physical illness in mother, (d) psychiatric disorder or chronic physical illness in father, (e) four or more siblings; and the number of points is summed to obtain a rating of adversity between 0 and 5.

For all three measures, five indices of inter-rater agreement were calculated: ICC(2,1) and ICC(3,1) are related intra-class correlations calculated according to the procedures in Shrout & Fleiss (1979). ICC(2,1) differs from ICC(3,1) in including the variance associated with additive biases of judges as part of the error variance, and is usually lower. The other correlational measure calculated is the ordinary product moment correlation (PMC).

These measures share a common problem: they are not robust because they can be affected drastically by outliers—a few large discrepancies or agreements at extreme points of the range (Mosteller & Tukey, 1977). To compensate for this deficiency, two other measures of agreement were calculated: percentage exact agreement (PEA) and percentage close agreement (PCA). PEA is the proportion of cases, expressed as a percentage, in which both judges gave the same numerical ratings; PCA is similar, but includes the cases in which raters differed by a single scale point. PEA and PCA have many disadvantages (Bartko & Carpenter, 1976), but the robustness arising out of the fact that they simply count agreements and disagreements, irrespective of their magnitude, is useful (Paykel, 1983). Together, these measures are complementary. Where they agree, it is probable that all is well with the data; where they disagree, the pattern may give an indication as to the nature of the problem. For example, ICC(2,1) is sensitive to both additive and multiplicative biases of the raters; ICC(3,1) is sensitive to multiplicative biases only; and PMC is sensitive to neither, so that the relative values of these indices may be used to assess rater bias. Similarly, if all correlational measures are low, while PEA and PCA are fairly high, it is likely that there are outliers, or that there is a restriction of range in the ratings.

Apart from this statistical exploration of sources of unreliability, the reliability within various subsets of the data was also calculated to see if procedural or patient variables altered the reliability.

Results

The 140 adolescents who were included in the study represented 67% of all assessments during a period of 10 months. The only reason for exclusion of a case was incomplete data at the time the sample for this report was finalised: most of the omissions were because a particular nurse-administered questionnaire was not completed, due to the nurse concerned having been unavailable. With few exceptions, the remaining omissions resulted from the arbitrary reporting date in an ongoing study, which meant that the coverage during the early part of the period was complete, while in the latter part, more cases were not included because routine clerical tasks (typing of assessment reports, collection of case files etc.) had not been done. Though it is unlikely that this sampling procedure would be biased toward 'easy' cases, if there was any such bias, it should become apparent as a difference in reliability between the first and second half of the sample.

TABLE I
Inter-rater reliability for DSM-III Axis IV, Axis V and PSAI for the sample of 140 adolescents and two judges

Measure		*Axis IV*	*Axis V*	*PSAI*
ICC(2,1):	Estimate	0.44	0.58	0.68
	95% CI	(0.27–0.58)	(0.45–0.69)	(0.57–0.76)
ICC(3,1):	Estimate	0.46	0.58	0.68
	95% CI	(0.31–0.59)	(0.45–0.69)	(0.57–0.76)
PMC		0.50	0.58	0.68
PEA		41%	45%	54%
PCA		82%	89%	89%

The results for the total sample, all measures and all indices of agreement are given in Table I. The three correlational measures are identical for Axis V (0.58) and for PSAI (0.68); while the differences for Axis IV are small and negligible in relation to the sampling variability of the estimates as indexed by the associated 95% confidence intervals (95% CI). The percentage agreement figures provide a convenient interpretation of the variability of the ratings: for example, when rating Axis IV, the judges agreed exactly in 41% of the cases, disagreed by one scale-point on a further 41%, and by two or more scale-points on the remaining 18% of cases.

On all indices of agreement, Axis IV was rated less reliably than was Axis V or PSAI, while Axis V ratings were less reliable than PSAI on all indices except PCA, which was equal for both; the 95% CI for ICC(2,1) and ICC(3,1) lend statistical support to these differences. There was practically no overlap in the ranges for Axis IV and PSAI; while the point estimates of 0.58 for Axis V lay at the top of the 95% CIs surrounding the Axis IV estimates, and at the bottom of the 95% CIs surrounding the PSAI estimates.

The remaining results are based on subdivisions of the total sample according to two methodological variables—experience/practice (first 70 cases *vs* last 70 cases) and source of interview data (seen/not seen)—and according to three patient variables—age (12–13 *vs* 14+), sex and diagnostic group (conduct/emotional/other).

Since the sample is not large enough to support simultaneous application of all these dimensions, the procedure adopted for the exploratory analyses was to calculate point-estimates of ICC(2,1) separately for the data in any subdivision, and treat as chance variation any estimate lying within the 95% CI for the same variable in the overall analysis (Table I). This is an arbitrary decision rule, since the probability of type I and type II errors is indeterminate, but it serves the practical purpose of calling

TABLE II

Inter-rater reliability for two judges rating Axis IV, Axis V and PSAI for a sample of 140 adolescents subdivided by position in case sequence and type of interview data

	Seen	*Not seen*	*Total*
Number of cases			
First 70 cases	32	38	70
Last 70 cases	31	39	70
Total	63	77	140
Axis IV, ICC(2,1)			
First 70 cases	0.48	0.41	0.45
Last 70 cases	0.37	0.46	0.43
Total	0.43	0.43	0.44
(95% CI)			(0.27–0.58)
Axis V, ICC(2,1)			
First 70 cases	0.58	0.54	0.57
Last 70 cases	0.54	0.60	0.58
Total	0.58	0.56	0.58
(95% CI)			(0.45–0.69)
PSAI, ICC(2,1)			
First 70 cases	0.68	0.47	0.55
Last 70 cases	0.79	0.78	0.79
Total	0.73	0.64	0.68
(95% CI)			(0.57–0.76)

attention only to large differences. A problem of this approach is the possible restriction of range of the variables in question (Axis IV, Axis V and PSAI) within the relatively homogeneous subgroups produced by subdividing the sample on patient variables such as age, sex or, particularly, diagnosis. If patients genuinely do not vary or if, for whatever reason, the judges assign similar ratings to patients within such a subgroup, to report an intraclass correlation would be misleading. Following Lahey *et al* (1983), the between-patient variation within each subsample was first tested to see if there was a statistically reliable main effect for patients. For samples which failed this test, the ratings are described as 'unreliable'.

Table II shows the results of the analysis of the first half of the sample *vs* the second, and for the patients seen and not seen. For Axes IV and V, there is no suggestion that either of these variables alone or in combination have any effect on reliability. For PSAI, however, the picture is somewhat different—this was rated more reliably for the last 70 cases in the study. There is also the indication of an interaction, since the lower reliability for the first 70 cases is particularly marked for the 38 cases who were not interviewed by either rater. Unless it is assumed that PSAI was affected by sample selection when ratings for Axes IV and V were not, this suggests that the sample was representative of the referral population.

There were 18 girls in the group of 12–13-year-olds and 29 in the 14+-year-olds; the figures for boys were respectively 51 and 42. Although girls made up 34% of the total sample, they seem to be under-represented in the younger age group (26%), and over-represented in the older age group (41%); this difference just failed to be statistically significant, but is still important. The most unusual result of this analysis is the unreliability of Axis IV ratings for girls, which occurred largely because of a lack of variation in ratings, so that the patient effect for girls overall and in each age group was not statistically reliable. Reliability of Axis IV ratings for younger (0.48) and older boys (0.52) and for boys overall (0.50) were well within the range of sampling variation (0.27–0.58), as were the ratings for younger (0.49) and older patients overall (0.40). For Axis V, reliability was much the same for younger (0.63) and older boys (0.59), and younger (0.56) and older patients (0.57), but reliability for younger girls (0.36) was lower than that for older girls (0.51), and was also the only result to lie outside the 95% CI for Axis V (0.45–0.69). For PSAI, there is no indication that reliability is different for boys (0.67) and for girls (0.69) respectively, but reliability seems to be higher for younger patients (0.74 *vs* 0.63), though both values are within the 95% CI (0.57–0.76).

There were 17 girls in the 'conduct', 16 in the 'emotional' and 14 in the 'other' groups; the numbers for boys were 45, 23 and 25 respectively. As might have been expected from the previous results, Axis IV ratings were also unreliable for girls in each diagnostic group. The corresponding results for boys (conduct: 0.47; emotional: 0.56; other: 0.52) are well within the 95% CI (0.27–0.58), and so too are the overall results for each diagnostic group (0.44, 0.51 and 0.37 respectively); Axis V ratings for girls in each diagnostic group were also unreliable. With the exception of boys in the 'conduct' group (0.35) and that group as a whole (0.39), the remaining results lie within the 95% CI for this axis (0.45–0.69). For PSAI, sex differences were small within each diagnostic group (conduct: 0.62; emotional: 0.56; other: 0.83), and only one of the overall results falls inside the 95% CI (0.57–0.76).

Discussion

Comparison of the present results with those of the DSM-III field trials (Spitzer & Forman, 1979; Williams & Spitzer, 1980) is complicated by procedural differences and by some uncertainty about the reliability index used in those trials. The statistic used in them is probably comparable with ICC(2,1); it was described both as 'the kappa coefficient of reliability' and as 'the intraclass reliability coefficient' (ICRC) in the DSM-III manual and by Spitzer & Forman (1979). The statistic that best fits that double label is the 'weighted kappa' described by Fleiss & Cohen (1973), which, for moderately large samples and two judges, is essentially the same as ICC(2,1) in its calculation. For the overall sample in the present study, weighted kappa took the same numerical values as those for ICC(2,1) given in Table I. In what follows, therefore, it will be assumed that ICRC and ICC(2,1) have similar interpretations.

Within the field trial data themselves, there are some puzzling results, which require explanation before proceeding to comparisons with the present data; the main one is the difference in reliability between adult and child/adolescent patients. Reliability for the Phase II draft of the DSM-III was better for adults than for children/adolescents (Axis IV: 0.66 *vs* 0.59; Axis V; 0.80 *vs* 0.52); whereas reliability for the earlier Phase I draft was worse for adult than for younger patients (Axis IV: 0.58 *vs* 0.75; Axis V: 0.75 *vs* 0.77) (Williams & Spitzer, 1980). At face value, this suggests that the changes made to the definitions of Axes IV and V improved reliability for adults and substantially reduced reliability for children/adolescents. The result cannot reasonably be explained by a change in procedure between the Phase I and Phase II trials—the reduced proportion of jointly *vs* separately conducted interviews—since the change from a 60:40 ratio for Phase I to a 33:67 ratio in Phase II would have been expected to affect reliability for adult and younger patients in the same way; in any case, the differential reliability achieved with joint interview *vs* separate interview (Axis IV: 0.62 *vs* 0.58; Axis V: 0.80 *vs* 0.69) (Spitzer & Forman, 1979) is not large enough to explain the decline of reliability in younger patients. Nor can the results readily be explained by practice effects. It can be inferred by comparing Spitzer & Forman (1979) with Williams & Spitzer (1980) that most of the judges who contributed Phase II ratings for adults had previously contributed Phase I ratings, and since only some 15 'practice' cases were required before contributing ratings, it is possible that at least some of the improved reliability of the Phase II ratings might be due to increased familiarity with the general procedure (as found in the present data for PSAI). On the other hand, it seems that only about half the child/adolescent Phase II judges had previously contributed Phase I ratings, so that a smaller improvement might have been expected. The actual result of a substantial decline in reliability for younger patients is not consistent with this pattern.

In the absence of evidence on the statistical reliability of the ICRC estimates provided by Spitzer & Forman (1979) and Williams & Spitzer (1980), it is not worth while to pursue these differences further, but these analyses suggest that it is not possible to use the DSM-III field trial data to predict the reliability that might be expected in the present study. It would be simplistic to suppose that reliability for adolescents ought to lie somewhere between the adult and the child/adolescent figures, since there is no *a priori* reason for supposing that reliability increases with the age of the patient: it is quite possible that the lower reliability for a mixed adolescent/child sample was because of poor reliability for adolescents rather than children. As in the present data, reliability for different subgroups may be quite different, and overall reliability for a heterogeneous group provides no indication of specific effects of this kind. Against this background, the overall results obtained in the present study (Table I) and the detailed results for various subgroups (Table II) are best interpreted as independent evidence of the relatively poor reliability of Axes IV and V in adolescent patients.

There are other aspects of both protocols which also limit generalisation of their results. Reliability is more than just consistency: it is consistency achieved in the face of diversity. The more diverse the sample of patients and judges, the more variable the information input, the longer the span of time covered by the study (or, in brief, the more the study reflects the diversity of the real world), the more likely it is that the results obtained can be generalised safely to other patients, judges, records and times. Neither the DSM-III field trials nor the present study are adequate in that sense. The basic unit in all the DSM-III data is a tetrad of two colleagues from the same institution—and probably with similar biases—rating two nearly successive patients, after each has interviewed them and read all other relevant information. For comparison, the basic unit in our study might be said to be similar: two colleagues, two patients, but in some cases working from case file data, and in some cases with one of the clinicians having conducted the interview. Within that basic unit, therefore, one might have expected somewhat better reliability in the DSM-III data. However, there is considerable difference in the way that both studies extended this over the patient–rater universe. The field trials did so by drawing, more or less at random, another tetrad of two patients and two raters, while we did so by using the same raters for new patients.

At first glance, it might seem that the DSM-III data would thus be more general; it does, after all, sample many more rater pairs with the same (adolescent/child) number of patients. Though this is true, it is important to note that, no matter how many such samples are drawn, each rater only makes decisions about two patients (or in some cases four), judged within a short time-span. There is no scope for long-term variation in performance across many patients to have its effect, and, in fact, there is good reason to suppose that ratings of only two consecutive patients would tend to be comparative judgements (which would increase concordance of rank ordering), rather than judgements referred independently to a common yardstick. Anyone who has marked examination papers is familiar with this phenomenon. Conversely, the reliability figure for the present study does include long-run variation in performance over a period of 10 months. As far as patients are concerned, there is no good reason to suppose that either sample would be inadequate, but by sampling many patient/rater/institution combinations, the DSM-III trials gave better coverage of 'between institution/between colleagues' variation than our study did. Thus, the point of this analysis is to suggest that the data available from the field trials and the present study should be seen as indicating the reliability achievable under different kinds of restriction of diversity, and the results are not encouraging.

An unexpected finding of the present study is the very low reliability of the ratings in Axes IV and V for adolescent girls, which occurred because of a lack of variation in ratings. As noted by Lahey *et al* (1983), this may occur because the patients are truly similar or just because the judges consider them to be so. Since there is no reason to suppose that adolescent girls are very similar in their levels of psychosocial stress, an alternative explanation may be that male judges have difficulties in assessing the severity of psychosocial stress, as defined by DSM-III, for adolescent girls.

However, the most useful data are those for PSAI; not only is it the most reliable measure on all indices (Table I), but it is also the most constant throughout the various subdivisions except one. PSAI reliability was lower in the first half of the data and seemed to depend on the source of interview data, whereas, in the last 70 cases, reliability was higher and the same irrespective of who conducted the interview. The first result suggests that experience with even an 'objective' measure of this kind is necessary. The second is no surprise: PSAI relies on objective data for the most part and has an explicit rule for its summation, rather than the requirement that the rating should reflect (in some unspecified manner) 'the summed influence' of stressors.

The results of this work have some broad implications for other reliability studies in this area. There is little evidence that interview by the rater makes an important contribution to reliability if an adequate set of questionnaire data is available and an interview has been conducted and reported. On the other hand, there is some suggestion that the sex of the patient and the sex of the rater may be important; that reliability may improve with experience of a new measure (PSAI); that it is important to look at long-run reliability; and that partitioning the data and using multiple measures can give useful insights. Exploratory analyses of this kind provide only 'indications' (Tukey, 1977; Mosteller & Tukey, 1977) of effects, and do not have the same status as formal statistical inferences, but such indications can be useful. For example, any measure which is intended for general clinical use should be immune to minor variations of the protocol and irrelevant patient variables such as age and sex. The indications that Axis IV ratings were not 'robust' in this sense should be considered when planning alternative measures. Finally, some of the data suggest that the main source of unreliability is likely to be the operational definitions of Axes IV and V.

References

Achenbach, T. M. & Edelbrock, C. S. (1979) The Child Behaviour profile: II. Boys aged 12–16 and girls aged 6–11 and 12–16. *Journal of Consulting and Clinical Psychology*, 47, 223–233.

——, & —— (1983): *Manual for the Child Behavior Checklist and Revised Child Behavior Profile*. Vermont: T. M. Achenbach.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed. Washington, DC: APA.

Bartko, J. J. & Carpenter, W. T. (1976) On the methods and theory of reliability. *Journal of Nervous and Mental Disease*, 163, 307–317.

Cleary, P. J. (1981) Problems of internal consistency and scaling in life event schedules. *Journal of Psychosomatic Research*, 27, 341–352.

Fleiss, J. L. & Cohen, J. (1973) The equivalence of weighted kappa and the intraclass correlation coefficient as measures of reliability. *Educational and Psychological Measurement*, 33, 613–619.

Joint WHO/ADMHA Project (1983) Diagnosis and classification of mental disorders and alcohol- and drug-related problems: a research agenda for the 1980's. *Psychological Medicine*, 13, 907–921.

Kendell, R. E. (1983) DSM-III: A major advance in psychiatric nosology. In *International Perspectives on DSM-III* (eds R. L. Spitzer, J. B. W. Williams & A. E. Skodol). Washington, DC: American Psychiatric Press.

Lahey, M. A., Downey, R. G. & Saal, F. E. (1983) Intraclass correlations: There's more there than meets the eye. *Psychological Bulletin*, 93, 586–595.

Mezzich, J. E., Coffman, G. A. & Goodpastor, S. M. (1982) A format for DSM-III diagnostic formulation: Experience with 1,111 consecutive patients. *American Journal of Psychiatry*, 139, 591–602.

Mitchell, S. K. (1979) Interobserver agreement, reliability and generalizability of data collected in observational studies. *Psychological Bulletin*, 86, 376–390.

Mosteller, F. & Tukey, J. W. (1977) *Data Analysis and Regression: A Second Course in Statistics*. Reading, Mass.: Addison-Wesley.

Parker, G., Tupling, H. & Brown, L. B. (1979) A parental bonding instrument. *British Journal of Medical Psychology*, 52, 1–10.

Paykel, E. S. (1983) Methodological aspects of life event research. *Journal of Psychosomatic Research*, 27, 341–352.

Russell, A. T., Cantwell, D. P., Mattison, R. & Will, L. (1979) A comparison of DSM-II and DSM-III in the diagnosis of childhood psychiatric disorders. III: Multiaxial features. *Archives of General Psychiatry*, 36, 1223–1226.

Rutter, M. & Shaffer, D. (1980) DSM-III: A step forward or back in terms of the classification of child psychiatric disorders? *Journal of the American Academy of Child Psychiatry*, 19, 371–394.

——, —— & SHEPHERD, M. (1975) *A Multi-axial Classification of Child Psychiatric Disorders: an Evaluation of a Proposal*. Geneva: World Health Organization.

SHAFFER, D., CHADWICK, O. & RUTTER, M. (1975) Psychiatric outcome of localized head injury in children. In *Outcome of Severe Damage to the Central Nervous System* (ed. R. Porter & D. W. Fitzsimons). Ciba Foundation Symposium 34 (new series). Amsterdam: Excerpta Medica.

SHROUT, P. E. & FLEISS, J. L. (1979) Intraclass correlations: uses in assessing rater reliability. *Psychological Bulletin*, **86**, 420–428.

SPITZER, R. L. (1984) A debate on DSM-III: first rebuttal. *American Journal of Psychiatry*, **141**, 546–547.

—— & FORMAN, J. B. W. (1979) DSM-III field trials: II. Initial Experience with the multiaxial system. *American Journal of Psychiatry*, **136**, 818–820.

——, —— & NEE, J. (1979) DSM-III field trials: I. Initial interrater diagnostic reliability. *American Journal of Psychiatry*, **136**, 815–817.

STROBER, M., GREEN, J. & CARLSON, G. (1981) Reliability of psychiatric diagnosis in hospitalized adolescents. *Archives of General Psychiatry*, **38**, 141–145.

TUKEY, J. W. (1977) *Exploratory Data Analysis*. Reading, Mass.: Addison-Wesley.

WILLIAMS, J. B. W. & SPITZER, R. L. (1980) DSM-III field trials: Interrater reliability and list of project staff and participants. In *Diagnostic and Statistical Manual of Mental Disorders* 3rd ed. Washington, DC: American Psychiatric Press.

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Life Events and Mania
A Special Relationship?

A. AMBELAS

Fifty patients in their first manic episode were compared retrospectively with groups of (a) manic patients in other than first admissions and (b) acute surgical cases. They were then followed up for 3–8 years. First manic admissions were linked to life events far more frequently – 66% *vs* 20% and 8% respectively for the other groups. Within-group comparisons showed patients with life events were much younger. The link between life events and manic episodes appeared immediate and selective, a view further supported by the findings of the follow-up. Later episodes precipitated by life events seem to require smaller amounts of stress. The possible role of life events in relation to mania is discussed.

Anecdotal reports linking manic episodes to major life events have appeared over many years in the psychiatric literature of many countries and in many languages (Bonnafous-Serieux & Ey, 1938; Stern, 1944; Parkes, 1964; Borgna, 1964; Scotti & Cerletti-Scotti, 1969; Hedri, 1976; Rickarby, 1977; Hollender & Goldin, 1978). Attempts to quantify this association have been much more scarce. Of those few that exist, the earlier ones (Blankenburg, 1964; Tölle & Fritz, 1971) were plagued by methodological weaknesses. The recent surge of interest in life events has tended to neglect mania. Few papers have focused on the condition specifically. Dunner *et al* (1979) looked at life events in the context of researching bipolar affective illness. Two-thirds of their patients with a manic first episode reported preceding life events, and among these a great preponderance were male. Ambelas (1979) compared patients with manic episodes with a control group of acute surgical admissions and found life events preceding the manic episodes in 28% of his sample, five times more frequently than in the control group. Kennedy *et al* (1983) used manic patients as their own controls and observed a far higher frequency of life events in a period prior to the manic episode than during an equal subsequent period of time. Lastly, Glassner & Haldipur (1983) found life events common in late onset bipolar patients, but not in early onset ones, where the cut-off point for early onset was at 20 years of age!

In recent years, a number of papers have appeared on other forms of causable mania; Krauthammer & Klerman (1978) introduced 'secondary' mania, a variety where somatic illness or a chemical substance is implicated as a cause. 'Post-partum' mania appears to be recognised and diagnosed far more frequently than had been the case earlier (Brockington *et al*, 1978; Kadrmas *et al*, 1979; Dean & Kendell, 1981; Katona, 1983).

Aims of the study

(a) To add to the still meagre body of information on the subject.
(b) To confirm or refute observations suggestive of a greater frequency of life events prior to first manic episodes than prior to subsequent admissions.
(c) To identify differences between patients whose episodes were related to life events and those whose episodes were not preceded by life events.
(d) To examine the evidence contained in the data with reference to 'post-partum' mania and to 'secondary' mania.
(e) To look at patterns of further morbidity, while maintaining the focus on life events.
(f) Finally, to attempt to promote the understanding of the role of life events in the precipitation or causation of manic episodes.

Parts of the study

There are two distinct but inter-related parts in this work.

The incidence study

We identified and studied patients in their first manic episode. The purpose of this was to examine the connection between life events and the manic presentation of affective illness and not with manic-depressive illness as a whole. Therefore, patients who had earlier depressive presentations, but not manic ones, were included in the study.

The follow-up study

We examined the course of the illness of the patients in the incidence study over a period of 3–8 years, with a specific interest in further life events.

The incidence study

Method

Patients

The patients were identified from the health authority's records of discharges. During the period 1974–1981, 340 patients were admitted to Walsgrave Hospital, Coventry, with a diagnosis of mania. Their case notes were screened and 50 were found to have had their first manic episode during that period. This figure matched the expected incidence over 8 years for a catchment area of 500 000. The sample consisted of 24 males and 26 females with an age range of 16–76 and a mean age of 34.8 years. All patients had received mainstream education and none had attended a special school. Thirty-six were currently or had been married and 14 were single, mainly the younger ones.

Controls

Two control groups were used for comparisons:

(a) A group of 40 patients who had been admitted for manic episodes other than their first. They were extracted from the data of an earlier study by the present author, after the exclusion of those in the original sample who were then presenting with their first manic episode. This group did not differ from the index group in terms of sex distribution, but it did differ in terms of age with the mean age 46.3 years.

(b) Because there is evidence that life events tend to be reported more frequently by younger patients, it was necessary to have an age-matched control group. A group of acute surgical cases was used, matched for sex, age and education; although not matched individually in terms of marital status, these patients as a group did not differ significantly from the patients in terms of this parameter.

Diagnosis

A diagnosis of mania had on all occasions been made by the consultant psychiatrist responsible for the patient's care (the investigator did not have the clinical responsibility for any of those patients). The diagnoses were verified against the research criteria of Feighner *et al* (1972).

TABLE I
Mean scaling scores for 49 events

Rank in English sample	*Event*	*Mean*
1	Death of child	19.53
2	Death of husband/wife	19.14
3	Being sent to gaol	17.76
4	Death of close family member	17.65
5	Serious financial difficulties	17.58
6	Miscarriage or stillbirth	16.96
7	Court appearance for serious offence	16.94
8	Business failure	16.68
9	Marital separation due to arguments	16.57
10	Unwanted pregnancy	16.48
11	Divorced	16.29
12	Fired	15.93
13	Death of close friend	15.70
14	Serious illness of family member	15.52
15	Unemployed for 1 month	15.43
16	Serious personal physical illness	14.67
17	Involved in a lawsuit	14.47
18	Fail important exam or course	14.38
19	Losing or being robbed of personally valuable object	14.34
20	Demotion	14.33
21	Taking a large loan (more than half annual salary)	13.64
22	Break engagement	13.12
23	Child marries without approval	12.69
24	Separation from significant person (e.g. close friend)	12.01
25	Prepare for important exam	11.41
26	Marital separation not due to arguments	11.33
27	Move to another country	11.14
28	Son enlists or drafted in forces	10.82
29	New person moves into home (e.g. relative, lodger)	10.13
30	Retirement	10.05
31	Change in work hours or conditions (overtime, second job, new boss, reorganisation)	9.68
32	Cease going out with steady boyfriend/girlfriend (after at least 3 months)	9.36
33	Change in line of work	9.24
34	Move to another city	8.57
35	Marital reconciliation	7.96
36	Child leaves home (e.g. college)	7.85
37	Wife becomes pregnant	7.08
38	Birth of child (for mother)	7.03
39	Change school, college or university	6.79
40	Birth or adoption of child (for father)	6.49
41	Begin full- or part-time education	5.91
42	Promotion	5.89
43	Move within same city	5.50
44	Marriage	5.44
45	Finish full-time education	5.26
46	Become engaged	4.25
47	Child engaged	3.72
48	Wanted pregnancy	3.70
49	Child marries (with approval)	3.14

Events and interviews

The presence or absence of life events was recorded by the medical officers responsible. The events needed to comply to the principles described by Brown & Birley (1968). Furthermore, they had to be contained in a checklist of events derived from that used by Paykel *et al* (1976), shortened by the omission of such events that were most subjective in quality (Table I). For the purpose of quantification, events were given the scaled scores arrived at by Paykel *et al.*

Events were required to be independent of the illness and to have occurred prior to the onset of the manic episode. Judgement on their independence and timing was based on evidence given by an independent witness, usually a close relative. Fortunately, on all occasions such an interview was present on record. Particular attention was paid to events which could well be dependent, e.g. separations, engagement. On those occasions, the patients themselves were interviewed, inevitably at a later date, by the investigator. When doubts remained, patients were removed to the group negative for life events. The period between an event and the patient's admission was kept to 4 weeks. Dates of admission were used in preference to dates of onset, because they are harder data and because the delay between apparent onset and admission was usually very brief. The case notes of the surgical controls could not be expected to contain relevant information; the patients were interviewed by the researcher on the basis of the seven types of events suggested by Brown & Birley (1968), facilitated by the above checklist and always within 10 days from their admission.

Weaknesses of the method

(1) The use of case notes and the reliance on recording by the clinicians responsible introduced the likelihood of sporadic and biased recording. On the other hand, it also excluded investigator bias and the 'invitation to report'.

(2) The use of a shortened version of Paykel *et al* (1976) event list could result in some positive cases being excluded.

(3) Limiting the admission period to 4 weeks could also reduce further the number of positive cases.

Still, these flaws were more likely to result in false negatives rather than false positives.

Results

Comparisons with control groups

Of the 50 patients who had suffered their first manic episode during the period the study covered, 33 (66%) were found to have experienced life events preceding their admission. Even this high figure of positive cases is likely to be an underestimate resulting from the design of the research.

Among the 50 surgical controls, only four (8%) reported an event during the 4 weeks prior to admission. The difference between the groups is striking.

A group of 40 manic patients in a repeat admission included eight (20%) positive for recent life events: (manic first episodes *vs* controls: $\chi^2 = 33.6$, $P < 0.001$; manic first episodes *vs* manic repeat admissions: $\chi^2 = 17.4$, $P < 0.001$). We concluded that life events are more closely linked with first manic episodes than with repeat manic episodes and more so than with acute surgical treatment.

Comparisons between positive and negative groups

Sex. Among patients positive for life events, the ratio of males to females was 16:17. Among negative patients, the sexes were also equally represented. The study failed to confirm the observation by Dunner *et al* (1979) who found manic episodes preceded by life events more frequently in male patients.

Age. The mean age of patients positive for life events was 28 years. The mean age of negative patients was 48.1 years. Figure 1 presents the age distribution for the positive and negative groups. It can be seen that there is difference both in the position and the shape of the curves.

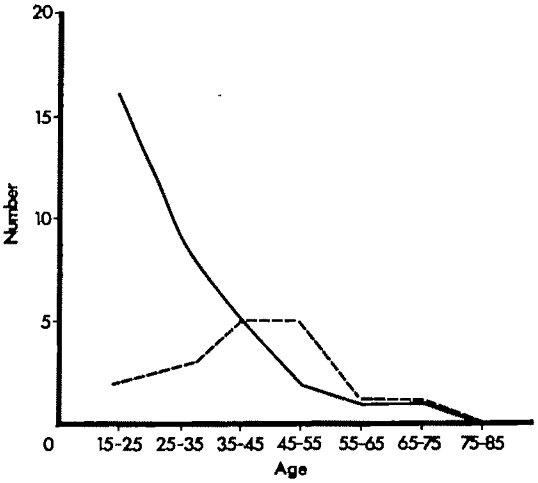

FIG. 1 Age at onset of first manic episode of patients positive (——) and negative (– – –) for life events.

Clinical picture. The groups were compared in terms of the frequency of the common symptoms of mania. No significant differences between positive and negative patients were found. However, there was a slight tendency for positive patients to present more paranoid features.

Previous depressive episodes. Because the first *manic* episode rather than the first manifestation of psychiatric illness was the basis for inclusion of patients to the study, some were expected to, and indeed did, have earlier episodes of depression. Among the 33 positive patients, six (18%) had such an episode, while among the 17 negative ones, nine (53%) had earlier depressive episodes. The difference is statistically significant ($\chi^2 = 4.90$, d.f. = 1, $P < 0.05$; Yates' Correction). This finding raised the question of whether the age differences between the groups were due to this factor alone. Exclusion of patients with earlier depressive episodes brought the mean age for positive patients down to 26.4 and for negative patients down to 43.5 years. Thus, the presence of previous depressive episodes could not account for the age difference.

Family history. Information about the family history, which was recorded in the notes, tended to be somewhat vague, especially with regard to the accuracy of diagnosis. We defined a positive family history as 'any mention of psychiatric problems in first and second degree relatives'. Evidence of positive psychiatric family history was found in eight (25%) of the positive and five (29%) of the negative patients. A definite diagnosis of mania in a first degree relative was found in only two positive and two negative patients. Thus, in the group positive for life events there was no evidence of familial loading which could account for their younger age as a result of greater genetic vulnerability.

Recovery after a depressive phase. Over half the positive and negative patients reached a stabilisation of their mood after a period of depression. The occurrence of a period of depression in such a high proportion of all manic patients may be an important observation.

The life events. Table II presents the life events which preceded the manic episodes in the positive cases. 'Deaths' included deaths of relatives and friends; 'separations' included divorces, breaks of engagement and terminations of steady relationships. The one case described as 'other' represents a combination of three different events from the list which all took place within the 4-week period under consideration.

Table III shows the frequency of occurrence of life events characterised as pleasant or unpleasant and entries or exits. It was considered inappropriate to characterise the birth of a child as either pleasant or unpleasant, since emotional uncertainty is very often present. This uncertainty is clearly shown by the fact that all childbirth-related events (wife pregnant, birth of child for mother, birth or adoption of child for father) in Paykel *et al*'s list showed some of the largest standard deviations from the mean scaling scores. Table III shows also that pleasant events were very rare before manic episodes. Only an engagement qualified for this group.

The four events among the surgical controls did not include any deaths or births. There was one separation and the other three were all related to employment.

TABLE II
Form of life events

	Event	*No.*	*%*
1	Deaths	8	24.24
2	Birth of child (for mother)	7	21.21
3	Birth of child (for father)	3	9.09
4	Personal health problems	3	9.09
5	Separations	5	15.15
6	Exams	3	9.09
7	Work related problems	2	6.06
8	Engagement	1	3.03
9	Other	1	3.03
	Total:	33	100%

TABLE III
Character of life events

	Events and stresses	*No.*	*%*	*Comments*
1	Pleasant	1	3.03	Births not included
2	Unpleasant	22	66.6	
3	Entries	10	30.30	
4	Exits	13	42.39	

In none of the subgroupings does the percentage total 100% since there are always events that cannot be classified.

Timing of events. Figure 2 shows the time delay between event and admission. The 4-week limit did not lead to the exclusion of any great number of cases. There were only two cases excluded on this basis—a birth 30 days before admission and a death 40 days before. The time relationship between events and manic admissions appeared close. An earlier finding of a delay in admission when the event was a bereavement (Ambelas, 1979) was not confirmed.

Mania and physical illness. In seven cases, there was a definite connection with poor physical health. On three occasions, the circumstances could be construed as definite events—two hysterectomies and one emergency admission for ruptured emphysema. In all three cases, these events occurred less than 10 days prior to the onset of mania. The other four patients had well established chronic illness (Stein-Leventhal syndrome, Addison's syndrome, hyper- and hypothyroidism) and were receiving active treatment. Thus, all four illnesses had a hormonal aspect. A positive family history of affective illness was not found in any of these cases.

'Post-partum' mania. The present study identified seven first manic episodes presenting in the puerperium. The onset was related closely in time to the birth, but this was the

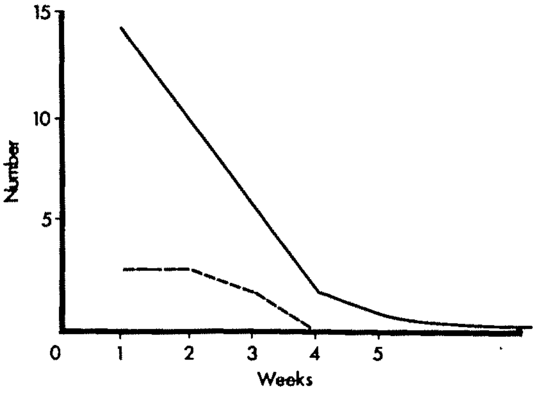

FIG. 2 Timing of life event prior to admission (——, all events; ——, event death).

case with all life events and the manic episodes they precipitated. The sample included three 'new fathers'.

The follow-up study

Method

The patients were followed for 3–8 years. The scope of this follow-up was limited and the focus was maintained exclusively on the occurrence of life events prior to further affective episodes. An early paper on reactive mania (Stern, 1944) suggested that, once a manic mechanism had been set up, further episodes could be precipitated by less severe events. Eventually a point might be reached when no event could be elicited. An attempt was made to look for evidence which would support this view.

Even though it would now have been possible to give the follow-up a prospective character, it was decided not to follow such a course, because it would destroy the comparability between first and other than first episodes. It was again left to the consultants responsible to diagnose, record events and interview relatives. The relevant data were then extracted from the case material. The same restrictions were applied and patients were interviewed only at a later date.

Results

The original cohort of 50 patients had a total of 59 readmissions. Of these, 43 were admissions of the 33 patients originally positive for life events and 16 were readmissions of the 17 negative ones. The difference in overall morbidity was not significant.

Among patients originally negative for life events, further admissions were predominantly manic (12/16). Of the 43 readmissions of positive patients, 29 were due to manic and 14 to depressive episodes.

Events were found prior to 20 readmissions, of which 18 were manic and only two depressive, while among the 23 admissions not precipitated by events, 11 were manic and 12 depressive. These differences are statistically significant ($\chi^2=8.67$, $P<0.01$). It is concluded that life events are perhaps selectively related to manic episodes.

Stress severity

In order to test the hypothesis that less severe stress precipitates further admissions, some means of quantification was needed. The scaled scores for life events determined by Paykel *et al* (1976) for English subjects were available (Table I). While there were large standard deviations for many events, suggesting significant differences in individual appreciation of stressfulness for a particular event, the alternative of getting individual scores from our patients was not chosen, because it was thought that patients would be likely to over-score events they had recently experienced.

From the list, each recorded event was allocated the mean scaling score corresponding to it. Scores for events related to first admissions and for those related to readmissions were averaged. The resulting means were 12.08 for events related to a first episode and 8.01 for events before readmissions. There was a clear difference and the finding supported the hypothesis that repeated manic episodes require less stress for their production.

Discussion

Two-thirds of patients admitted to hospital with their first manic episode reported life events that had preceded the onset of their illness. This association with life events was significantly higher than in subsequent admissions with mania, where life events were found in one-fifth of patients, and even more so than in the month before admission of acute surgical cases where less than one in ten had experienced life events. The different frequency of events before admission due to a first manic episode or as a surgical case appeared valid. The tendency of precipitating events to be less severe before further admissions may have led to underestimation of the association with later admission, as their severity falls below a recording threshold and their independence becomes more doubtful.

The age difference between patients who experienced preceding events and those who did not was a surprising finding. It could not be explained by genetic differences, or by a greater number of earlier depressive episodes in the group without events. The tendency of younger subjects to report more events must also be discarded as a sufficient explanation, since it clearly did not lead to the age-matched surgical controls reporting many events. We are left with the observation that younger subjects are more likely to have event-related mania and a possible speculation that life events may be bringing the manic presentation forward in time—by as much as 20 years.

Four cases were found where a diagnosis of secondary mania could be applied. A hormonal factor was implicated in each case. Interestingly, in Krauthammer & Klerman's (1978) original series, steroids were the agents most commonly thought responsible. It may well be that a relevant association exists and this should be sought at the interface of psychiatry and endocrinology.

In recent years, there has been an increasing recognition of manic disorder during the puerperium, with Dean & Kendell (1981) finding 12.7% of all the admissions in the 90 days following delivery to be manic, while in Katona's (1983) sample the proportion rose to 22%. Both studies point out a tendency for the mania to appear very soon after the delivery. In more than one-fifth of our positive cases, the birth of a child was the life event and the onset of mania was immediate. This high proportion of births among the life events recorded would support the view of Kadrmas *et al* (1979) that childbirth may be a very 'special' event in the context of mania.

While the 'puerperal' notion has tended to imply biochemical causes, and related evidence exists, attention must be drawn to the presence of three 'new fathers' among the positive cases in the present sample where a biological link cannot be postulated.

Whether the role of life events is causal or precipitant can never be answered in an absolute manner. The findings of this study suggest that life events may have a specific and major responsibility for the manic presentation of affective psychosis. The observed temporal closeness between event and episode (also noted in puerperal illness), the selective association between life events and mania rather than depression in further episodes, would support such a view. Whatever the overall cause of manic-depressive psychosis, life events could be important for the manic component. This view does not contradict the evidence from genetic studies of the unipolar *vs* bipolar illness continuities. There is no reason why a specific diathesis and a life event could not both be required for the clinical picture of mania to be produced. Alternatively, the inherited diathesis may express itself as vulnerability to life events.

In a different vein, the psychodynamic model of mania (Klein, 1948) postulates a defence which is brought into play to protect the patient from the most destructive effects of depression and gives time for the slower mechanisms of reparation to take effect. This understanding requires a quick manic response to situations of loss, real or perceived. The findings of this study are consistent with such a model. The stress, very often loss, seems to be responded to by mania. The response is very quick, again as the theory assumed. Thus, a large number of patients go into a depressive phase before normalisation can be achieved, and mania could be construed as a temporary shield or buffer which makes the ensuing depression tolerable, or even causes it to be aborted.

Finally, the findings do not contradict the conceptualisation of mania as a syndrome of non-specific aetiology which can be brought into play by a variety of noxious stimuli—biochemical, hormonal but also, and quite frequently so, psychologically traumatic.

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References

AMBELAS, A. (1979) Psychologically stressful events in the precipitation of manic episodes. *British Journal of Psychiatry*, **135**, 15–21.

BLANKENBURG, W. (1964) Lebengeshichtliche Factoren bei manischen Psychosen. *Nervenartz*, **35**, 536–539.

BONNAFOUS-SERIEUX, H. & EY, H. (1938) Manies de veuvage. *Annales Medico-Psychologiques, Part 1*, **96**, 225–232.

BORGNA, E. (1964) La provocazione psicoreattiva delle ciclotimie nei suoi aspetti psicopatologici clinici e teoretici. *Giornale di Psiciatria e Neuropatologia*, **92**, 795–814.

BROCKINGTON, I. F., SCHOFIELD, E. M., DONNELY, P. & HYDE, C. (1978) A clinical study of puerperal psychisis. In *Mental Illness in Pregnancy and the Puerperium* (ed. M. Sandler). New York: Oxford University Press.

BROWN, G. W. & BIRLEY, J. L. T. (1968) Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behaviour*, **9**, 203–214.

DEAN, C. & KENDELL, R. E. (1981) The symptomatology of puerperal illnesses. *British Journal of Psychiatry*, **139**, 128–133.

DUNNER, D. L., PATRICK, V. & FIEVE, R. R. (1979) Life events at the onset of bipolar affective illness. *American Journal of Psychiatry*, **136**, 508–511.

FEIGHNER, J. P., ROBINS, E., GUZE, S. B., WOODRUFF, R. A. J., WINOKUR, G. & MUNOZ, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

GLASSNER, B. & HALDIPUR, C. V. (1983) Life events and early and late onset of bipolar disorder. *American Journal of Psychiatry*, **140**, 215–217.

HEDRI, A. VON (1976) Zur Frage der Umweltabhängigkeit bei Manie. *Zeitschrift für Allgemeinmedizin, Dresden*, **52**, 474–475.

HOLLENDER, M. H. & GOLDIN, M. L. (1978) Funeral mania. *Journal of Nervous and Mental Disease*, **166**, 830–832.

KADRMAS, A., WINOKUR, G. & CROW, R. (1979) Post-partum mania. *British Journal of Psychiatry*, **135**, 551–554.

KATONA, C. L. E. (1983) Puerperal mental illness: comparisons with non-puerperal controls. *British Journal of Psychiatry*, **141**, 447–452.

KENNEDY, S., THOMPSON, R., STANCER, H. C., ROY, A. & PERSAD, E. (1983) Life events precipitating mania. *British Journal of Psychiatry*, **142**, 358–403.

KLEIN, M. (1948) Contribution to the psychogenesis of manic depressive states. In *Contributions to Psychoanalysis*. London: Hogarth Press.

KRAUTHAMMER, C. & KLERMAN, G. L. (1978) Secondary mania. *Archives of General Psychiatry*, **35**, 1333–1339.

PARKES, C. M. (1964) Recent bereavement as a cause of mental illness. *British Journal of Psychiatry*, **110**, 198–204.

PAYKEL, E. S., MCGUINESS, B. & GOMEZ, J. (1976) An Anglo-American comparison of the scaling of life events. *British Journal of Medical Psychiatry*, **49**, 237–248.

RICKARBY, G. A. (1977) Four cases of mania associated with bereavement. *Journal of Nervous and Mental Disease*, **165**, 255–262.

SCOTTI, F. & CERLETTI-SCOTTI, A. (1969) Il lutto maniacale. *Rivista Sperimentale di Freniatria e Medicina Legale delle Alienazioni Mentali*, **93**, 110–130.

STERN, E. S. (1944) The psychopathology of manic-depressive disorder and involutional melancholia. *British Journal of Medical Psychology*, **20**, 20–32.

TOLLE, R. & FRITZ, H. (1971) Zur Auslosung von Manien. *Psychiatria Clinica (Basle)*, **4**, 12–18.

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Hysterical Personality Disorder

The Process of Diagnosis in Clinical and Experimental Settings

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A study of the case notes of 52 patients with a diagnosis of hysterical personality disorder showed that 27 had no recorded features of that condition, but were frequently described as aggressive, uncooperative, or attention-seeking. Although 60% were recorded as depressed, only 17% received antidepressant treatment. In an experimental study, the core traits of the disorder were found to have a low inter-rater reliability. Two patients, who had received hospital diagnoses of hysterical personality disorder, were shown to display behaviour that caused psychiatrists to make a diagnosis on wholly inadequate information.

The present classification of personality disorders remains unsatisfactory and attracts much criticism, particularly for the category of hysterical personality disorder (Presley & Walton, 1973). The status of this disorder as a diagnosis is closely linked with the clarity of the definitions: "It has been used to describe quite different and poorly delimited varieties of behaviour; 'hysterical' has taken on some of the attributes of both a fossil and a chameleon, so that it is understandable that there is a total failure of agreement as to just what is meant when something is referred to as 'hysteria' or 'hysterical'" (Chodoff & Lyons, 1958). The definition found in the ICD 9 provides a standard description: "Shallow, labile affectivity, dependence on others, craving for appreciation and attention, suggestibility and theatricality. There is often sexual immaturity, e.g., fridgidity and overresponsiveness to stimuli. Under stress hysterical symptoms (neurosis) may develop".

Perhaps the most successful attempt to refine the diagnosis is in DSM-III, where it has been renamed 'histrionic personality disorder' to avoid the semantic confusion and unfortunate associations inherent in the terms 'hysteria' or 'hysterical', and is defined as:

A. Behaviour that is over-dramatic, reactive, and intensely expressed, as indicated by at least three of the following: (1) Self-dramatisation, e.g. exaggerated expression of emotions; (2) incessant drawing of attention to oneself: (3) craving for activity and excitement; (4) over-reaction to minor events; (5) irrational, angry outbursts or tantrums.

B. Characteristic disturbances in inter-personal relationships, as indicated by at least two of the following: (1) perceived by others as shallow and lacking genuineness, even if superficially warm and charming; (2) egocentric, self-indulgent, and inconsiderate of others; (3) vain and demanding; (4) dependent, helpless, constantly seeking reassurance; (5) prone to manipulation suicidal threats, gestures, or attempts.

The aim of the present study was to describe how the category of hysterical personality disorder is used as a diagnosis; it was in two parts—a retrospective case note study and an experimental study. The first examined the use of the diagnosis in a clinical situation, the criteria on which it is made, and its effect on subsequent management. The experimental study investigated the process of diagnosis, using videotaped interviews, where the information available to the raters was presented in three separate extracts, each controlled for duration and content. The ratings made by the psychiatrists are examined to describe the patterns of diagnostic behaviour for each patient, in order to identify the underlying criteria for the diagnosis.

Preliminary study

The preliminary study describes the characteristics of a sample with the diagnosis of hysterical personality disorder. From a total of 1129 patients discharged from the Department of Psychiatry, Withington Hospital, Manchester in 1975, 52 received this diagnosis without any secondary one, and composed the sample. An item checklist was drawn up from an examination of ten case notes, taken at random. The information retrieved covered those clinical features on which the diagnosis appeared to be based, and included details of the patients's demographic characteristics, recent and past psychiatric and medical history, mental state findings, and subsequent management.

The case notes of all patients were retrieved and the data extracted on to the item checklist. Mean age of the sample was 37.1, s.d. 12.6 years; the sex ratio was 83% female, 17% male. These findings are comparable with other studies (Slavney & McHugh, 1974; Reed, 1975).

The mental state findings are summarised in Table I, and include all recorded abnormalities. A finding of particular interest is that 27 patients, i.e. about half of the sample, were not described with any of the core behavioural traits for hysterical personality disorder.

TABLE I

Findings on Mental state Examination and in continuation sheets of 52 patients diagnoses as hysterical personality disorder

	Index admission	*Past admission*
Depressed	31	3
Anxious	20	1
Aggressive	18	8
Unco-operative	10	3
Attention seeking	10	7
Histrionic	10	5
Suicidal	10	3
Manipulative	6	1
Evasive	6	1
Labile	5	2
Dramatic	5	2
Violent	3	1
Amnesia	3	1
Gansering	2	0
Hostile	2	0
Dependant	1	1
Belle indifference	1	0
Suspicious	1	1
Seductive	1	0
Egocentric	1	0
Immature	1	1
Suggestible	0	0

Examination of the case notes revealed a requirement for an additional diagnosis in half of the sample: these diagnoses were arrived at using conventional operational definitions where appropriate, but confirmed by an independent assessor. Nine patients suffered from alcohol or drug addiction, four suffered from depression with three or more biological symptoms, eight from epilepsy, three from hysterical neurosis, and two from a toxic confusional state.

The association with affective disorder was evident, both from the mental state findings and from the psychiatric history; 23 patients had a history of affective disorder, 12 described depression as their main presenting complaint, and 31 were described as depressed and ten as suicidal by the admitting doctor. A history of one or more overdoses was present in 38, and self-mutilation in 14; four were assigned an additional diagnosis of primary depressive illness by the criteria of Feighner *et al* (1972). In contrast, only nine patients received antidepressant medication and two had been treated with ECT.

Experimental study

Method

In the experimental study, two patients from the hospital sample were selected and invited to take part in a semi-structured videotaped interview. This consisted of three parts; the first extract, lasting one minute, included no clinical material, taking the form of a discussion about a neutral topic, e.g. a recent holiday, which simply allowed the viewer to see and hear the patient. The second extract, lasting 6 minutes, focussed on the patient's interpersonal difficulties, at the time of the index admission and during the subsequent 5-year period up to the present study; a detailed description was elicited of the patient's behaviour towards others—under usual circumstances and at times of stress. The final extract, lasting one minute, provided a vignette of the patient's presentation at the index admission, with relevant mental state findings, but without giving a diagnosis. Videotaped interviews of an identical format were prepared for two control patients who had been discharged in the same year, with diagnoses of an organic psychosis and a manic-depressive psychosis respectively.

The videotapes were shown to 14 psychiatrists, of varying seniority, who were unaware of the nature of the study. The order of presentation was: (a) hysterical personality disorder, (b) control, (c) control, (d) hysterical personality disorder. The videotape was stopped after each extract, and the psychiatrists asked to make their ratings. Each set of ratings consisted of an attempt at diagnosis, with the provision for a maximum of two alternatives allowed at each point, together with ratings on a scale of 0 to 10 for eight behavioural traits, presented as the following bipolar adjectival pairs: labile/stable, seductive/modest, dramatic/phlegmatic, hostile/friendly, immature/mature, anxious/relaxed, depressed/elated and dominant/submissive.

Results

The experimental study recorded the diagnostic behaviour of the rating psychiatrists, when making ratings of the four videotaped interviews for the two index hysterical personality disorders from the hospital sample and the two control patients. The first set of results focussed on the diagnoses made. Each of the 14 psychiatrists could make up to a maximum of two diagnoses after each extract, and therefore the maximum number in each cell was 28.

TABLE II

Number of diagnoses made by 14 psychiatrists allowed up to two diagnoses at each stage of viewing four videotapes. (Maximum number in each cell = 28)

	Recent holiday	*Interpersonal relations*	*Clinical details*
HPD	28	26	26
Control	9	16	20
Control	7	18	22
HPD	23	27	26
Comparison index with control groups	$P<0.01$*	$P<0.01$*	$P<0.05$

*Mann-Whitney U-Test

Table II shows that the number of psychiatrists making any diagnosis was significantly greater for the two index patients at the 1/1000 level. A significant difference between the two groups was most evident following the first extract, where one of the index patients actually received 28 diagnostic attempts—the maximum number possible.

TABLE III

Number of diagnoses of the general category of personality disorder made by 14 psychiatrists allowed up to two diagnoses at each of the three presentations. (Maximum number in each cell = 28)

	Presentation		
	1	*2*	*3*
HPD	7	17	18
Control	4	2	0
Control	1	8	5
HPD	8	11	16
Comparison of HPD with control groups	$P<0.1$*	$P<0.1$*	$P<0.01$*

*Mann-Whitney U-Test

The results presented in Table III refer to the number of diagnoses awarded for the general category of personality disorder. Any category of personality disorder was included in this figure, the data being reduced to the three figure ICD code—i.e. 301. The two index patients were again assigned a significantly greater number of diagnoses of personality disorder, the difference becoming strongest following presentation of the third extract.

TABLE IV

Number of diagnoses of hysterical personality disorder made by 14 psychiatrists at each of the three presentations (maximum number in each cell = 14, see text)

	Presentation		
	1	*2*	*3*
HPD	4	8	8
Control	1	1	0
Control	0	2	3
HPD	0	1	1
Comparison of HPD with control groups	NS	$P<0.5$*	NS

*Mann-Whitney U-Test

Table IV records the number of diagnoses of hysterical personality disorder, given after each extract. In this analysis, the denominator is 14, this being the maximum number of identical diagnoses that could be given by the 14 rating psychiatrists at each rating point. Comparing the number of diagnoses of hysterical personality assigned to the index and control patients, the results failed to reach a consistent statistical difference. One index patient received only two diagnoses of hysterical personality disorder, whilst one of the control patients received five diagnoses of the disorder.

TABLE V

Inter-rater reliability scoring behavioural traits—(Kendall's Coefficient of Concordance

		Presentation		
Trait	*Overall*	*1*	*2*	*3*
Depression	0.62	0.76	0.61	0.63
Anxiety	0.15	0.28	0.13	0.03
Immaturity	0.57	0.50	0.48	0.53
Dramatic	0.36	0.30	0.35	0.44
Seductive	0.28	0.20	0.39	0.15
Dominant	0.41	0.37	0.42	0.29
Hostility	0.78	0.70	0.87	0.87
Lability	0.25	0.02	0.22	0.23

At each rating point, concurrent with attempting a diagnosis, the psychiatrists made ratings for each of the eight behavioural traits. Comparing the two index and the two control patients, analysis of the ratings revealed the index patients to be rated as significantly more depressed, immature, dominant, and hostile after each videotaped extract.

From an examination of the diagnoses made during the videotaped interviews, it is apparent that the diagnosis of hysterical personality disorder was made on a total of 26 occasions. The results can be examined to identify the behavioural traits that are rated as present when the psychiatrist assigned the diagnosis of hysterical personality disorder, whether to an index or a control patient. Extracting the data for the 29 points where a diagnosis of the disorder was given shows that the patient is then rated as more immature, dramatic, seductive, and labile, all at the 1/100 level of statistical significance.

The inter-rater reliability for each trait was calculated using Kendall's coefficient. Although the reliability improved as more information was provided, the core traits of the hysterical personality disorder—seductiveness, dramatic lability and immaturity-remained unacceptably low (Table V).

Discussion

Patients receiving a hospital diagnosis of hysterical personality disorder are markedly heterogeneous, suffering from affective disorder, alcohol and drug addiction, epilepsy, and toxic confusional states. Despite the obvious diversity of the sample, there are particular features that single out patients for inclusion in this diagnostic category: the results of this study suggest that a major contribution derives from the mental state findings, which characteristically describe the patient as aggressive, unco-operative, attention-seeking, histrionic, manipulative, and evasive. Characteristics of the 52 subjects bear a close resemblance to those described by Forrest (1967), as shown by patients who do not comply with the hospital regime and are seen as difficult and demanding. However, it is remarkable that 27 patients were diagnosed as 'hysterical personality disorder' without one single core trait of the disorder being recorded in the case notes.

In keeping with the findings of Reed (1975) and of Slavney & McHugh (1974), there is a marked association with affective disorders, particularly primary depressive disorder. A second and more disturbing conclusion is that awarding the diagnosis of hysterical personality disorder distracts the doctor from formally recognising other significant psychopathology. This is reflected in half of the sample, where careful inspection of the case notes revealed evidence of an additional diagnosis. Similarly, the relatively meagre prescription of antidepressant treatment would indicate a failure to translate the recorded features of depression which were noted on admission into adequate treatment for the depression. This closely resembles the findingsof Bibb & Guze (1972), who reported a retrospective study of patients with Briquet's syndrome, where 80% of the sample was hospitalised for depression, yet the index cases were less likely to receive ECT than the controls.

The present experimental study examined the process of diagnosis in a situation where the psychiatrist might be presumed to be on his best diagnostic behaviour. The patient sample was very small, but the problems of persuading a group of psychiatrists to sit through a series of long videotapes prevented a more elaborate study.

The results indicate a significantly greater number of total diagnoses accorded to the index patients, most evident after a 1-minute video extract of the patient's appearance and behaviour during a clinically neutral interview. This observation compares with that of Kendell (1973) on 28 psychiatrists rating diagnostic interviews presented in three different formats—videotape, audiotape, and transcript; ratings were made at 2 minutes and 5 minutes. Comparing these ratings, the second interview showed an increase in the frequency of diagnoses for psychotic illness, with a corresponding fall in those of neurotic conditions: one may speculate that the increased amount of verbal and non-verbal information generated by the two index patients resulted in a more rapid diagnostic process and decision, resulting in a relative under-diagnosis of functional psychiatric illness and a parallel over-diagnosis of personality disorder.

One of our index patients achieved a respectable level of agreement amongst the rating psychiatrists as a genuine case of hysterical personality disorder, obtaining in total 20 such diagnoses. The other received only two diagnoses of hysterical personality disorder, and was not rated as having its core behavioural traits. Nevertheless, there are features common to both index patients which contribute to an understanding of why they received this hospital diagnosis. Both were rated as significantly more depressed, dominant, immature, and hostile, and their behaviour during the first extract of a one-minute interview had features that encouraged the psychiatrists to make significantly more diagnoses without adequate clinical information.

When assigning the diagnosis of hysterical personality disorder to either an index or control patient, the psychiatrists rated the patient as seductive, labile, dramatic, and immature; this finding is consistent with the classical description. It suggests that in an experimental situation, those assigning such a diagnosis have accurately assimilated the pattern of behavioural traits. On the other hand, having assigned the diagnosis, the psychiatrists may have simply rated what they believed should be seen. Either of these explanations would indicate that the psychiatrist has an accurate internal model of the behavioural traits classically described in the hysterical personality disorder.

Examination of the inter-rater reliability reveals a low coefficient of concordance for the core traits—dramatic, seductive, and labile—despite the videotaped interview focussing on areas of interpersonal difficulties which might be supposed to elicit such behavioural traits. So far as the surprisingly low inter-rater reliability for the trait of anxiety is concerned, it must be presumed either that the interview did not address itself to this trait, or alternatively that the actual recording of the interview in a studio produced anxiety in the patient that was interpreted differently by the psychiatrists.

Conclusion

The diagnosis of hysterical personality disorder is frequently made without the core features being present: over half of this hospital sample had none of the features of the disorder recorded in the case notes, and 50% of the patients had some other diagnosable condition. Two influences emerge from the present study that would seem to account for these findings. The first involves the use of the diagnosis to label those patients who are perceived as hostile, difficult, and unco-operative, with the result that the doctor is distracted from recognising an underlying diagnosis. By their very nature, the core traits of 'dramatic' and 'attention-seeking' indicate a performance that is overtly unconvincing and being played out by the patient; the availability of a recognised diagnostic category may allow doctors to resolve their negative feelings toward such patients. Yet these behaviours should cause doctors to try and understand the underlying problems, rather than attributing them exclusively to a long-standing character disorder. The second factor has not previously been identified; it is that patients with a hospital diagnosis of hysterical personality disorder have a set of characteristics that encourages psychiatrists to make diagnoses on wholly inadequate information. These characteristics were most evident in the first one-minute extract, and apparently consist of an excess of non-verbal behavioural cues, which lead to rapid and perhaps premature diagnostic decisions. The contribution of these two factors, translated into the clinical situation, may well account for the excessive use of the diagnosis reported by Walton & Presley (1973).

The definition of 'Histrionic Personality Disorder' in DSM-III may be helpful in answering some of the problems associated with the diagnosis, yet this definition itself relies on the accurate recognition of such traits as self-dramatisation and attention-seeking behaviour, which in this study have been found to have a low inter-rater reliability. Until these issues have been resolved, the diagnosis should be avoided; patients with undeniably genuine psychopathology may then be assured of an assessment not clouded by judgements about the peculiar nature of the personality traits.

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References

BIBB, R. C. & GUZE, S. B. (1972) Hysteria (Briquets Syndrome) in a psychiatric hospital: The significance of depression. *American Journal of Psychiatry*, **129**. 224–228.

CHODOFF, P. & LYONS, H. (1958) Hysteria, the hysterical personality and hysterical conversion. *American Journal of Psychiatry*, **114**, 734–740.

FEIGHNER, J. P., ROBINS, E. & GUZE, S. B. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–70.

FORREST, A. D. (1967) Manifestations of 'hysteria': phobic patients and hospital recidivists. *British Journal Medical Psychology*, **42**, 263–270.

KENDELL, R. E. (1973) Psychiatric diagnosis: a study of how they are made. *British Journal of Psychiatry*, **122**, 437–445.

PRESLEY, A. S. & WALTON, H. J. (1973) Dimensions of abnormal personality. *British Journal of Psychiatry*, **122**, 269–276.

REED, J. L. (1975) The diagnosis of hysteria. *Psychological Medicine*, **5**, 13–17.

SLAVNEY, P. R. & MCHUGH, P. R. (1974) The hysterical personality; a controlled study. *Archives of General Psychiatry*, **30**, 325–329.

WALTON, H. J. & PRESLEY, A. S. (1973) Use of category system in the diagnosis of abnormal personality. *British Journal of Psychiatry*, **122**, 259–268.

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Brief Reports

Deliberate Self-harm and Predictors of Out-patient Attendance

Kreitman (1979) reported that up to one-half of patients given out-patient appointments one week after an episode of deliberate self-harm (DSH) fail to attend, and gave a number of possible explanations for this. Firstly, parasuicide is often the result of a crisis which may have resolved (albeit temporarily) by the end of a further week. Secondly, someone in a state of heightened tension may find one week too long to wait, and may resort to other strategies to deal with his problems. Thirdly, many parasuicides may find a psychiatric label unacceptable in the context of their problems, and fourthly, an appointment made for a fixed day and a fixed hour may not fit the need for immediate action which the subject's subculture had inculcated in him as a habit pattern. Morgan *et al* (1976) reported that up to 40% of their DSH patients either did not attend any appointment or failed to complete their treatment. Two possible explanations for this were that they either felt that they did not need psychiatric treatment, or else believed that psychiatric treatment was not an answer to their problems. Kessel & Lee (1962), probably in line with much psychiatric practice, did not give a follow-up appointment to 40% of their self-poisoners; this was for two reasons. Firstly, these patients did not have a problem for which psychiatric treatment was appropriate and secondly, many of these patients had an entrenched personality disorder, which made it unlikely that psychiatric intervention would be beneficial.

Our study was designed to discover potential predictors of non-attendance. All consecutive referrals were given automatic out-patient appointments, with visits arranged at one week and one month after their DSH.

Method

All consecutive adult DSH referrals in a district general hospital in Newcastle upon Tyne were seen; it is still the policy of this hospital for all DSH cases to be referred to the psychiatric service for assessment and disposal. In addition to a standard psychiatric interview each patient was investigated with the following diagnostic and rating instruments: Suicide Intent Scale (Pierce, 1981); Newcastle Diagnostic Scale (Carney *et al*, 1965); Research Diagnostic Criteria (Spitzer *et al*, 1978); Hamilton Rating Scale for Depression (HAMD) (Hamilton, 1960) the 17-item version.

All patients received the standard psychiatric interview, and were investigated on the various scales at initial interview. At the one-week and one-month follow-up of those referred to out-patients', the RDC and HAMD were again completed. In offering patients follow-up, it was explained to them that this was both to assess their progress and also to help them deal with any problems they might have. Patients were followed-up by the assessor who originally saw them. A total of 98 patients were seen, of whom 80 were overdose takers, 14 self-mutilators, and four were both.

Twelve patients were admitted to a psychiatric unit, and the remaining 86 each given a psychiatric out-patient appointment; these 86 formed the subject of this study. They were given an appointment one week after their initial interview; those who attended at one week were given a further appointment at one month. Those who failed to attend at one week were sent a follow-up appointment at one month.

The statistical methods used were the χ^2 for categorical variables and the t-test for continuous variables.

Results

At one week, the attendance rate was 40%. At week four, attendance for the total sample was 31%. There were no significant differences between the attenders and non-attenders on the following items: marital status, state of marriage, children to the marriage, presence or absence of financial stress, history of Court appearances, employment status, history of unhappy childhood, early parental separation (under 15 years of age), and early parental death (under 15 years of age). There were also no significant differences with age, although there was a trend for a fall in the rate of attendance with increasing age. The two groups also did not differ significantly on the following items: past psychiatric history, past overdose, past laceration, psychiatric history in the family, and illicit drug use. With regard to psychiatric diagnosis there was once again no significant difference between attenders and non-attenders on various RDC categories, as shown in Table I.

A crisis situation preceding the DSH was also not predictive of attendance at follow-up: 42% with a crisis situation attended (similar to the overall attendance rate of 40%).

TABLE I

Category	*Attendance rate*
Mental disorder present	40%
No mental disorder	42%
Personality disorder present	41%
No personality disorder	29%

With regard to initial score on the Suicide Intent Scale, once again there was no significant difference between the attenders and non-attenders: Only 53% of patients with DSH rated as having been of serious intent came for out-patient follow-up. As to initial score on the HAMD, only 22% of those who scored 18 and over attended for follow-up.

On the Newcastle Depression Scale, 40% (five patients) of those with a diagnosis of endogenous depression attended (although the numbers were too small for any significance to be attached to them, compared to 39% with a diagnosis of neurotic depression—once again a non-significant difference between attenders and non-attenders.

Discussion

A working hypothesis which suggested itself to us before we commenced our study was that the patients who were more likely to be non-attenders would be those who were younger, who had a personality disorder, and who did not suffer a psychiatric illness. Our findings did not confirm this. On no major item investigated did we find clear-cut differences between those who attended and those who did not. A failure to attend applied equally to those with a psychiatric disorder, with clear symptoms of depression, and who had a high intent score, as it did to those without mental disorder or depression, or who had a low suicide intent score at initial interview.

Although we have not been able to identify the characteristics of those who failed to attend out-patients, our findings may well suggest that a different approach to the management of post-DSH patients may be required. Some patients may not see their problems in psychiatric terms (Kreitman, 1979), so that help from other professionals such as a social worker may be more appropriate—and this also suggests itself from the fact that most cases of DSH occur in the setting of social and interpersonal problems. Kreitman also suggested that attendance one week after the DSH may be too long for a patient to have to wait for help with his problems—an appointment within 2 or 3 days may therefore be more appropriate—and that seeing the patient away from the hospital setting, possibly at home, may improve the delivery of help offered to him. In this setting, the skills of a social worker or a community psychiatric nurse may be usefully applied, and such an approach would also be useful for a number of patients the psychiatrist would have wished to see at his clinic, but who default on this attendance. However, the early involvement of the general practitioner for the latter would also be valuable, more especially for the elderly depressive who may be at greater risk of suicide (Stengel, 1970). For the latter, the general practitioner and psychiatrist should liaise closely, in the event of the patient requiring either admission or a psychiatric domiciliary visit.

References

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

KESSEL, W. I. N. & LEE, E. M. (1962) Attempted suicide in Edinburgh. *Scottish Medical Journal*, **7**, 130–135.

KREITMAN, N. (1979) Reflections on the management of parasuicide. *British Journal of Psychiatry*, **125**, 275.

MORGAN, H. G., BARTON, J., POTTLE, S., POCOCK, H. & BURNE-COX, C. J. (1976) Deliberate self-harm: a follow-up of 279 patients. *British Journal of Psychiatry*, **128**, 361–368.

PIERCE, D. W. (1981) The predictive validation of a suicide intent scale. *British Journal of Psychiatry*, **139**, 391–395.

STENGEL, E. (1970) *Suicide and Attempted Suicide*. Harmondsworth: Penguin Books.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1978) *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders*. New York: Biometrics Research, New York State Psychiatric Institute.

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Khat-induced Paranoid Psychosis

For many hundreds of years the fresh leaves of the Khat shrub have been used for their stimulant properties. Several million people in East Africa and Arab countries chew the fresh leaves and with the growth of rapid air transportation the habit has spread.

Cathinone is considered the most active ingredient and has been recently isolated and synthesised (United Nations Document, 1975). The effects of cathinone closely resemble those of amphetamine (Halbach, 1972), to which it is chemically related. Regarding the beneficial effects of Khat, Carothers wrote in 1945:

> The chewing of this weed induces a happy and mellow friendliness and an increased intellectual vigour and acuity, thought tends to rise to a high plane, desire for war and women ceases, and conversation tends to concern itself with 'the affairs of God' and the accumulation of wealth by peaceful trade.

Amphetamine like sympathomimetic stimulation occurs with elevated blood pressure, mydriasis, hyperthemia, anorexia, insomnia, increased arousal, elevated mood and loquacity (Halbach, 1972).

Khat abuse may cause psychic dependence (Eddy *et al*, 1965), aggressive behaviour, paranoid ideation and ultimately personality disorders (Kalix, 1984). It may cause manic psychosis (Carothers, 1945; Laurent, 1962; Giannini & Castellani, 1982), similar in many features to that which may be induced by amphetamines. A North Africán student, in the United States developed a manic psychosis following ingestion of Khat which he had grown in his own apartment (Giannini & Castellani, 1982).

Case Report

F. K. a 34-year-old Somali woman was admitted to a medical ward following an overdose of propranolol, paracetamol and choroquine. She is a part-time student, separated from her husband and two children since 1978, who lives alone in a Council flat. She had no significant past medical history and there was no history of past psychiatric illness either in herself or in her family.

When seen on the medical ward she was noted to have paranoid delusions and evidence of thought broadcast which took the following form. Over several weeks prior to admission she had felt convinced that there were microphones located on the stairway leading to her flat which were repeating her thoughts. She felt she was being watched and followed and a searchlight was focussed on her flat monitoring her activities. She reported that the television repeated her thoughts. During these weeks she was chewing Khat every day and on the day of admission had chewed it for 8 hours. Prior to admission she had noted a loss of appetite and said she had not slept for 48 hours. She had felt that everything built up inside her to such an extent that she took the overdose.

At this stage insight was completely lacking, and she was still actively suicidal, but her mental state was otherwise normal. She was noted to have a mild otitis media which was treated with antibiotics. Full blood count was normal, a chest X-ray showed evidence of a little right basal shadowing and atelectasis consistent with infection, and a urine drug screen taken 9 days post-admission showed evidence of morphine and dihydrocodeine. From the second day she was prescribed trifluoperazine 5 mg three times daily which she took regularly for 5 days and intermittently thereafter. She remained severely paranoid for 11 days after admission but on day 12 her mental state had reverted to normal and she was discharged shortly afterwards. We felt it probable that the trifluoperazine had been a contributory factor in her recovery.

She returned to be seen on the ward 3 weeks after discharge bringing a sample of Khat which was identified by the Regional Poisons Unit. Her mental state was quite normal at that stage.

On a recent visit she admitted to having used Khat on occasions since her admission 4 months ago. She still felt at times her activities were being monitored and felt there were possibly microphones on the stairway. However, she described that when she chewed Khat these feelings intensified and on enquiry, were found to be fully delusional. Indeed, after taking Khat on two occasions since discharge she has again felt like taking an overdose.

Discussion

We were unable to fully explain the positive urine test for morphine and dihydrocodeine. The urine test may remain positive for up to 4 days following ingestion, which would indicate she may have taken these substances while on the ward. She adamantly denied use of these substances and was not prescribed any compound containing them.

The case of F. K. serves to highlight the importance of knowledge of Khat use in the United Kingdom. Use of Khat has serious socio-economic consequences in that Khat users harm their families by neglect, dissipation of family income and by their inappropriate behaviour (Kalix, 1984). It may cause a functional psychosis following consumption of exceptionally potent material or in predisposed persons (Kalix, 1984).

Finally, although this is the first case known to the Regional Poisons Unit, it is likely that greater awareness of the problem may result in other cases coming to light. It is the impression of F. K. that extremely large numbers of the Somali community in London (approximately 4000) use Khat and this is likely to represent only a proportion of the problem. In the case of F. K. Khat appears to have induced a full-blown paranoid psychosis with the added complication of a suicide attempt.

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References

CAROTHERS, J. C. (1945) Miraa as a cause of Insanity. *The East African Medical Journal*, Jan. 4–6.

EDDY, N. B., HALBACH, H., ISBELL, H. & SEEVERS, M. H. (1965) In "Drug Dependence: its significance and characteristics" *Bulletin of the World Health Organisation*, **32**, 721–733.

GIANNINI, A. & CASTELLANI, S. (1982) A manic-like psychosis due to Khat *Journal of Toxicology—Clinical Toxicology*, 19(5), 455–459.

HALBACH, H. (1972) Medical aspects of the chewing of Khat leaves. *Bulletin of the World Health Organisation*, **47**, 21–29.

KALIX, P. (1984) The Pharmacology of Khat *General Pharmacology* **15** (no 3), 179–187.

LAURENT, J. M. (1962) Toxique et Tixocomonie peu connus, "Le Cath". *Annales Medico-Psychologiques* **120**, 649–657.

UNITED NATIONS DOCUMENT (1975) Études sur la composition chimique du Khat; recherches sur la fraction phenylalkylamine. *MNAR*, No 11.

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Down's Syndrome with Mania

Sovner *et al* (1985) proposed that the presence of Down's Syndrome (DS) precludes the possibility of concomitant mania, and this is not a new claim, since Earl (1961) had stated that neither cyclothymia nor manic-depressive psychosis occurred in the mentally retarded. While Sovner *et al* conceded that cases of mania in individuals with DS may simply have gone unrecognised or unpublished, they felt this to be unlikely. However, lest this suggestion enter the canon,. we present a previously unreported case of a young adult with both DS and recurrent mania.

Case report

A moderately mentally retarded, 23-year-old man was brought to a university psychiatric clinic by his mother because she was concerned about his increasing activity, increasingly grandiose ideas and decreased sleep. Down's Syndrome had been previously diagnosed on the basis of physical stigmata at examination, as well as trisomy 21 on chromosomal analysis. There was no family history of psychiatric illness. The patient had had a similar episode 2 years previously, characterised by increased hyperactivity, increased rate and volume of speech, and decreased appetite and sleep. Thioridazine treatment was instituted at that time, following which the patient became somewhat sedated; the symptoms had remitted within a month.

Two months before the most recent referral, the patient had once again shown increasing levels of activity and impulsivity and his mother had been forced to restrict him to the home because he had wandered off and almost got lost. His deteriorating behaviour was accompanied by a preoccupation with grandiose delusions, including that of his becoming a famous musician. He also became increasingly irritable, particularly when his grandiose ideas were challenged. Physiological changes included decreased appetite, a weight loss of 9 kg (20 lb), and decreased sleep. On each of the two nights immediately before being seen, he slept at most 2–3 hours, and was reportedly dancing and singing when awake, although it was in the middle of the night.

When seen, the patient was dressed like a certain pop singer, and demanded to be called by that singer's name, stating that the examiner was a television producer interviewing him for a job. The patient's affect was characterised by rapid swings from elation to irritation, when his mother did not respond to his grandiose statements, but when not challenged, he was most engaging. The examination revealed no evidence of hallucinations or mood-incongruent delusions. The physical, neurological, and laboratory examinations were all within normal limits, while a cardiology consultation revealed no change in the patient's asymptomatic ventricular septal defect with intraventricular and intra-atrial conduction defects.

The diagnosis was made of bipolar affective disorder, manic with mood-congruent psychotic features (DSM-III). This diagnosis was later confirmed independently by two attending psychiatrists.

Discussion

Mania and DS can and do occur concomitantly, as this case illustrates; there is a strong suggestion that ours is not the first report of this coincidence. Although not designated as cases of 'mania' (perhaps because of the then-prevailing diagnostic criteria), Rollin (1946) described a behaviour change, frequent among 'mongols', in which patients underwent

> a period of excitement amounting in some cases to an almost manic phase. They became wild, noisy and restless, talking incessantly, emitting weird animal noises or inarticulate gibberish. This phase passes after a variable interval, only to recur without any obvious cause.

Information in Rollin's three case vignettes is not sufficient to diagnose mania retrospectively, but it is of interest that one patient was noted to have become irritable and 'talked incessantly all day'.

Although unipolar mania and bipolar affective disorder may be less prevalent than unipolar depression in individuals with DS, this case shows that the two disorders are not mutually exclusive. However, much remains unknown about the incidence and prevalence rates of psychiatric disorders in DS, as well as in other types of mental retardation. If the prevalence rate of bipolar illness in DS is indeed less than in control populations, interesting possibilities are posed for further investigation of genetic predisposition to and protection from psychiatric illness.

Sovner *et al* (1985) have suggested an explanatory hypothesis for mania being incompatible with DS—that alterations in monoamine activity inhibit the development of mania—but present understanding of the pathophysiology of both conditions is too limited to support the contention of mutual exclusiveness.

In fact a case can even be made for a heightened association of DS and bipolar affective illness. Prange *et al* (1974) hypothesised that low indoleamine activity 'permits' development of mania, when it is present with elevated catecholamine activity. In subjects with DS, blood 5-hydroxyindole levels (Rosner *et al*, 1965; Tu & Zellwegger, 1965) and serotonin uptake into platelets (Boullin & O'Brien, 1971) have been reported to be decreased, compared with controls. According to Prange's hypothesis, this should lead to an *increase* in the incidence of mania in individuals with DS, but as yet, too little is known of the biology of both DS and mania: there are still contradictory data. For example, CSF 5-hydroxyindoleacetic acid levels in patients with DS have been found *not* to differ from those of neurological controls (Lott *et al*, 1972, Tu & Partington, 1972).

Epidemiological studies of psychiatric illness among the mentally retarded have been only limited; as a whole, they suggest that such illness is at least as prevalent as in the general population (Parsons *et al*, 1984). Psychiatric diagnosis in the mentally retarded illustrates the importance of diagnosing each case on the clinical data, rather than on theories of pathogenesis (Sovner & Hurley, 1983).

References

BOULLIN, D. J. & O'BRIEN, R. A. (1971) Abnormalities of 5-hydroxytrytamine uptake and binding by blood platelets from children. *Journal of Physiology*, **212**, 287–297.

EARL, C. J. C. (1961) *Subnormal Personalities*. Baltimore: Williams and Wilkins.

LOTT, I. T., MURPHY, D. L. & CHASE, T. N. (1972) Down's syndrome—central monoamine turnover in patients with diminished serotonin. *Neurology*, **22**, 967–972.

PARSONS, J. A., MAY, J. G. & MENOLASCINO, F. J. (1984) The nature and incidence of mental illness in mentally retarded individuals. In *Handbook of Mental Illness in the Mentally Retarded* (eds F. J. Menolascino & J. A. Stark), London: Plenum.

PRANGE, A. J., WILSON, I. C., LYNN, C. W. *et al* (1974) L-Tryptophan in mania. *Archives of General Psychiatry*, **30**, 56–62.

ROLLIN, H. R. (1946) Personality in mongolism with special reference to the incidence of catatonic psychosis. *American Journal of Mental Deficiency*, **51**, 219–237.

ROSNER, F., ONG, B. H., PAINE, R. S. & MAHANAND, D. (1965) Blood-serotonin activity in trisomic and translocation Down's syndrome. *Lancet*, **i**, 1191–1193.

SOVNER, R. & HURLEY, A. D. (1983) Do the mentally retarded suffer from affective illness? *Archives of General Psychiatry*, **40**, 61–67.

——, —— & LABRIE, R. (1985) Is mania incompatible with Down's syndrome? *British Journal of Psychiatry*, **146**, 319–320.

TU, J. & PARTINGTON, M. W. (1972) 5-Hydroxyindole levels in the blood and CSF in Down's syndrome, phenylketonuria and severe mental retardation. *Developmental Medicine and Child Neurology*, **14**, 457–466.

—— & ZELLWEGER, H. (1965) Blood-serotonin deficiency in Down's syndrome. *Lancet*, **ii**, 715–717.

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Capgras Syndrome in a Patient with Dementia

The Capgras syndrome was first described as an 'illusion of doubles' in 1923 by two French psychiatrists, Capgras and Reboul-Lachaux. Since then, a number of cases have been reported of this syndrome co-existing with functional as well as organic disorders. Enoch & Trethowen (1979) regarded the syndrome as purely functional—a manifestation of schizophrenia. Recently, there have been case reports describing the occurrence of Capgras syndrome with diabetes (MacCallum, 1973), hypothyroidism (Madakursira & Hall, 1981), hepatic encaphalopathy (Pies, 1982; Cummings, 1985), pseudo-hypoparathyroidism (Hay *et al*, 1974), temporal lobe epilepsy (Chawla & Virmani, 1977) and vitamin B12 deficiency (Zucker *et al*, 1981).

The following report discusses the presence of Capgras syndrome in a patient with dementia.

Case report

A 73-year-old married woman was referred for evaluation of her memory problem. Her husband complained that it was difficult to manage her because she told him "You are not my husband but an impostor". This delusional misidentification persisted, occasionally only for hours, sometimes for days. During the episodes, her husband tried to persuade her of his identity by showing her photographs and reminding her of their earlier life, but without success.

On mental state examination she was not hallucinating, but her memory and orientation were impaired. There was no evidence of depressive disorder and she had no suicidal ideas. At the clinic, she was able to recognise her husband; but when reminded of her earlier statements, she again expressed occasional suspicions that he might be an impostor. She had insight into her memory impairment, and became very anxious and upset when she could not perform well in tests. Blood biochemistry, including T_3, T_4, serum B12 and folic acid, was within normal limits. Electroencephalographic and computer axial tomographic scan findings were consistent with a generalised degenerative process. Most probably, she was suffering from dementia of the Alzheimer type.

The Capgras phenomenon persisted on and off during an 8-week follow-up period. As the patient's husband was coping satisfactorily, prescription of psychotropic medication was postponed.

Discussion

Reports of the Capgras delusion co-existing with various brain lesions or with metabolic and endocrine disorders are appearing with increasing frequency. Although some authors (Hayman & Abrams, 1977; Alexander *et al*, 1979; Quinn, 1981) have suggested that the syndrome is commoner in localised right than in left hemisphere disease, the present report is the first which suggests that Capgras features can also accompany a general degenerative disorder of the brain such as dementia of the Alzheimer type.

It is difficult to establish that the Capgras delusion is caused by an organic state, and the simultaneous occurrence of both in one patient may be coincidental. However, it would seem desirable in all cases of Capgras syndrome to investigate thoroughly for an organic pathology. It also seems that the syndrome is not as specific to schizophrenic or affective psychosis as has been suggested in the past.

References

Alexander, M., Struss, D. T. & Benson, D. F. (1979) Capgras syndrome: a reduplication phenomenon. *Neurology*, **29**, 334–339.

Capgras, J. & Reboul-Lachaux, J. (1923) *Bulletin de la Société clinique de médécine mentale (Paris)*, **11**, 6.

Chawla, H. M. & Virmani, V. (1977) Capgras phenomenon in a case of temporal lobe epilepsy. *Folia Psychiatrica et Neurologica Japanica*, **31**, 615–617.

Cummings, J. L. (1985) Organic delusions—phenomenology, anatomical correlation and review. *British Journal of Psychiatry*, **146**, 184–197.

Enoch, M. D. & Trethowen, W. H. (1979). *Uncommon Psychiatric Syndromes*, 2nd ed. Bristol: John Wright and Sons.

Hay, G. G., Jolley, D. J. & Jones, R. G. (1974) A case of the Capgras syndrome in association with pseudo-hypoparathyroidism. *Acta Psychiatrica Scandinavica*, **50**, 73–77.

Hayman, M. A. & Abrams, R. (1977) Capgras' syndrome and cerebral dysfunctions. *British Journal of Psychiatry*, **130**, 68–71.

MacCallum, W. A. G. (1973) Capgras symptoms with an organic basis. *British Journal of Psychiatry*, **123**, 639–652.

Madakursira, S. & Hall, T. B. III (1981). Capgras syndrome in a patient with myxedema. *American Journal of Psychiatry*, **138**, 1506–1508.

Pies, R. (1982) Capgras phenomenon, delirium and transient hepatic dysfunction. *Hospital & Community Psychiatry*, **33**, 382–383.

Quinn, D. (1981). The Capgras syndrome: two case reports and a review. *Canadian Journal of Psychiatry*, **26**, 126–129.

Zucker, D. K., Livingstone, R. L., Nakra, R. & Clayton, P. J. (1981). B12 deficiency and psychiatric disorders: case report and literature review. *Biological Psychiatry*, **16**, 197–205.

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A Pilot Study of Phenelzine in the Treatment of Post-traumatic Stress Disorder

In recent years, there has been renewed appreciation of the morbidity which can result from unusual or overwhelming stress and while many situations can give rise to post-traumatic disorder, the most frequently studied of these is probably military combat. Psychiatric disorder pursuant to combat experience can not only become chronic, but may intensify with advancing age, decades after the original trauma (Archibald & Tuddenbaum, 1965; Wilmer, 1982). Moreover, a high percentage of combat veterans are believed ultimately to develop chronic psychiatric morbidity (Walker & Cavenar, 1982). The drug treatment of such post-traumatic states remains an important question, largely overlooked until the last 2 years but recent case reports suggest that doxepin and imipramine (White, 1983; Burstein, 1984) are beneficial in treating post-traumatic stress disorder (PTSD), which may be either combat or non-combat related. Hogben & Cornfield (1981) described five veterans whose PTSD improved when treated with phenelzine, while Van der Kolk (1983) has described beneficial results with antidepressants, lithium, benzodiazepines, beta-blockers, and neuroleptics in uncontrolled studies of PTSD.

Having studied phenelzine in depressed inpatients (Davidson *et al*, 1978, 1981), depressed outpatients (Raft *et al*, 1981), and during long-term continuation treatment (Davidson & Raft, 1984), we decided to undertake a further study of phenelzine in PTSD. Our aim was to extend the earlier reports and to utilise quantitative measures of drug effect. We employed observer and self-rating scales, the latter being considered a useful counter-balance to any observer bias that might operate in an open study, particularly when a new drug indication is being explored and, permitting assessments of specific symptom complexes within PTSD. We present here the results of a pilot study of phenelzine in 11 veterans of World War II or Vietnam.

Method

During a 7-month period, all potentially suitable subjects were interviewed for the study. It was necessary for patients to meet DSM-III criteria for chronic PTSD, which are grouped into four distinct sets of items.

(a) Presence of a recognisable stressor that would evoke stress in almost everyone.

(b) Intrusive re-experiencing of the trauma, as shown by at least one of the following:
  (i) recurrent recollections of the event,
  (ii) recurrent dreams of the event,
  (iii) acting as if the traumatic event were recurring because of a reminder.

(c) Numbing of responsiveness to, or reduced involvement with the external world following the trauma, as illustrated by at least one of the following:
  (i) diminished interest,
  (ii) estrangement from others.
  (iii) constricted affect.

(d) At least two of the following symptoms that were not present before the trauma:
  (i) exaggerated startle or hyperalertness,
  (ii) disturbed sleep,
  (iii) guilt over survival or behaviour required for survival,
  (iv) impaired memory or concentration,
  (v) avoidance of reminders of the event,
  (vi) symptoms worse by exposure to symbols or reminders of the event.

In addition, we required that at least one key symptom each out of sections (b) and (c) be present to at least a moderate degree on a 0–4 rating scale, in which 0 = absent, 1 = mild, 2 = moderate, 3 = severe and 4 = extreme; we thus ensured that patients manifested at least a moderate degree of active symptoms. All patients gave informed consent for the study and receiving treatment as out-patients.

A dose of either 45 mg or 60 mg phenelzine was administered for 6 weeks, with a minimum of 4 weeks' treatment to complete the study. We chose a conservative approach to dosing, since there were no accepted guidelines for using phenelzine in this condition. No other psychoactive medicine was administered.

Patients returned for assessment at weeks 2, 4, and 6. All the rating scales were administered at each visit, and platelet MAO activity was measured at weeks 0, 4 and 6, by a method described by Davidson & White (1983).

To assess progress, we used three observer and three self-rating scales. One observer (J. I. W.) independently assessed the DSM-III features of PTSD, after which the patient was interviewed by another observer (J.D.) for assessment with the Hamilton anxiety and global improvement measures (Hamilton, 1959). Patients completed the Impact of Events Scale (IOES) of Horowitz *et al* (1979), which measures the intrusive ('positive') and avoidant ('negative') aspects of post-traumatic stress. Patients also completed a self-rated global improvement assessment. Treatment emergent effects were rated by JRTD, as a measurement of possible drug-related adverse events.

Anchor points on the global improvement scale were 1 = very much improvement, 2 = much improvement, 3 = moderate improvement, 4 = minimal improvement, 5 = no change, 6 = deterioration. These can be conveniently divided up into responder and non-responder groups, using ratings of 1, 2, or 3 to denote the former and ratings of 4, 5, and 6 for the latter.

The full dose of phenelzine was begun on the first day of treatment.

Results

Eleven patients entered the study, one of whom dropped out at week 2 because he did not wish to remain on the restricted diet. Ten patients completed 4 weeks' treatment, at which point two were removed from the study on account of poor response or deterioration, while another was removed because of side-effects. All subsequent analyses refer to those cases completing 4 or 6 weeks.

The mean age ± s.d. was 45.7 ± 12.8 years, the median being 39 (Table I). Five of those completing were veterans of World War II, and five of the Vietnam War. Nine patients received 45 mg phenelzine per day from the first day of treatment, and one 60 mg per day, which had to be reduced at week 4 due to side-effects.

Measures of efficacy

The degree of observed and self-rated improvement at weeks 2, 4, and 6 is presented in the Table I; 50% of the group were assessed as having responded by the two global improvement scales at week 2. At week 4, 70% of the group had responded to treatment according to the observer CGI, while 80% rated themselves as having responded on the self-rated CGI. Observed and self-rated responder rates at week 6 were 85% and 100%.

Platelet MAO activity was measured in nine patients at weeks 4 and 6. (It had not been possible to obtain a baseline value for the first patient.) The median levels of inhibition at weeks 4 and 6 were 81% and 85% respectively, while the three non-responders or equivocal responders (patients 5, 8, 10) showed inhibition values of 31, 48, and 80% at week 4. The responders achieved inhibition levels of 81, 100, 87, 92, 65, and 89% at week 4.

The observer ratings showed significant reduction of score, relative to baseline, using a paired two-tailed t-test for within-group differences. The DSM-III criteria score was 27.9 ± 6.5 at baseline, falling to 18.8 ± 7.4 at week 2 ($P<0.1$), 16.0 ± 7.7 at week 4 ($P<0.05$), and 13.2 ± 6.4 at week 6 ($P<0.1$).

The Hamilton anxiety score was 26.7 ± 6.9 at baseline, dropping to 15.7 ± 4.5 at week 2 ($P<0.05$), 15.7 ± 7.2 at week 4 ($P<0.02$) and 14.5 ± 3.5 at week 6 ($P<0.1$).

The amount of change in each symptom was examined with respect to week 4, calculating change as a percentage relative to baseline. Thus, if baseline score was 3, and the post-treatment score was 1, the percentage of improvement would be 66%. For the items in group (b), improvement for intrusive recollections, recurrent dreams, and acting as if back in combat were 61%, 42%, and 65% respectively. For the items in group (c), improvement in diminished interest, estrangement, and constricted affect was 37%, 56%, and 78% respectively. For the items in group (d), improvement for hyperalertness/startle was 39%, sleep disturbance 41%, guilt 44%, impaired memory or concentration 60%, avoidance of activities 18%, and symptoms worse with reminders 20%.

The self-rated IOES failed to show any evidence of change on either intrusive or avoidant items. The mean ± s.d. scores at each time point for avoidant items were 16.9 ± 4.9, 15.9 ± 4.7, 15.8 ± 5.1, 14.8 ± 6.7. The scores for intrusive items were 16.6 ± 3.8, 15.9 ± 5.0, 13.8 ± 4.1 and 16.4 ± 4.0.

TABLE I
Demographic information and response to treatment

Patient No.	*Age*	*Daily dose mg/day*	*Week 2*		*Week 4*		*% MAO*	*Week 6*		*% MAO*
			O-CGI	*SR-CGI*	*O-CGI*[1]	*SR-CGI*[1]	*Inhibition*	*O-CGI*[1]	*SR-CGI*[1]	*Inhibition*
1	60	45	1	4	2	2	NA	1	3	NA
2	58	45	DROPOUT							
3	32	45	3	3	1	1	81	3	3	85
4	39	45	3	4	2	3	100			
5	49	45	5	5	6	5	31			
6	59	45	5	4	2	3	87	2	2	98
7[2]	36	45	2	2	3	2	92	2	3	100
8[2]	59	45	4	2	4	3	48	4	3	86
9	35	45	4	3	3	3	65	2	3	30
10	31	45	4	4	4	4	80			
11	37	60	3	3	2	3	89	3	3	69
Median	39	45	4	3	2	3	81	2	3	85

1. Scores of 1 = very much improved, 2 = much improved, 3 = moderately improved, 4 = minimally improved, 5 = no improvement, 6 = worse.

O-CGI denotes Observed Clinical Global Improvement

SR-CGI denotes self-rated clinical global improvement

Scores of 1, 2, 3 are grouped as 'responders', scores of 4, 5, 6 are grouped as 'Non-responders'.

2. Coexistent major depressive disorder.

Treatment-emergent events

At each week, patients were questioned about the emergence of any new symptoms or complaints that had not been present prior to treatment, and note was also made of problems that appeared to worsen during treatment. Clinically significant events were reported by eight (80%) patients, who averaged 2.0 events per person. Most frequent and troublesome were intensification of pre-existing sleep disturbance (four patients), emergence of dizziness (four), and erectile failure (three). Other side-effects noted in one case each were delayed ejaculation, delayed urination, constipation, dry mouth, blurred vision, drowsiness, behavioural disinhibition, blackouts, perceptual changes, and hypomania.

The aggravation of sleep disturbance was especially troublesome, since sleep impairment had been a leading reason for treatment, having caused significant distress to patient and spouse. These treatment-emergent events necessitated dose reduction from 60 to 45 mg in one instance, and to treatment being discontinued in three others. From our previous experience with phenelzine (Evans *et al*, 1983), we had not expected to find so many problems with tolerance of this dose, but no serious or life-endangering toxicity occurred.

Discussion

This pilot study suggests promising effects of phenelzine in the treatment of PTSD, and thereby potentially extends further the application of MAO inhibitors in psychiatry. However, we should not automatically assume that the improvement which we saw was PTSD-specific, since many features of this condition are non-specific symptoms, also found in depression and anxiety (e.g. loss of interest, sleep disturbance, impaired concentration). It is therefore of interest to note that three out of the five symptoms which improved by greater than 50% were specific to PTSD: intrusive recollections, acting as if the trauma were recurring, and estrangement from others. Somewhat less improvement was noted on sleep, nightmares, startle, and guilt, although there was noticeable amelioration on these measures.

Our findings need to be followed by a double-blind placebo-controlled assessment of phenelzine, since it could be argued that we were merely observing placebo effects, e.g. most improvement occurred in the first 2 weeks. Furthermore, a placebo-controlled study would enable one to assess which particular symptoms were most drug-responsive.

The self-rated IOES failed to detect any drug effect, and we wonder whether it is unsuitable for measuring treatment effects, or if patients perhaps had difficulty in fully understanding the scale's wording. Yet another explanation is that secondary gain (e.g. disability pension) made patients reluctant to admit too much progress.

Forty-five mg of phenelzine is thought to be a borderline dose, and our MAO data suggest that one reason for poor response could have been insufficient dosing. On the other hand, an important finding was the high incidence of troublesome side-effects at the moderate doses used, thus making the dose of phenelzine itself a topic for further assessment in PTSD.

The findings can be speculatively related to recent animal studies which used an analogue of PTSD—the inescapable shock model. Weiss *et al* (1975, 1976) showed that both phenelzine and clonidine decreased the long-term effects of inescapable shock in rats. Since clonidine down-regulates locus ceruleus activity, and phenelzine results in a centrally mediated down-regulation of adrenergic activity, we are tempted to speculate that the effects of phenelzine in PTSD are also mediated through the locus ceruleus, and further evidence that it is of importance in PTSD comes from the successful use of clonidine in controlling the hyper-reactivity of PTSD (Kolb *et al*, 1984). Van der Kolk (1985) has proposed one association between positive symptoms and noradrenergic hyper-reactivity, and another association between negative symptoms and depletion of catecholamines. Further studies are clearly needed in order to understand the neurobiological basis for PTSD, and to integrate basic mechanism with treatment response.

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References

Archibald, H. C. & Tuddenham, R. D. (1965) Persistent stress reaction after combat: a 20-year follow-up. *Archives of General Psychiatry*, **12**, 475–481.

Burstein, A. (1984) Treatment of post-traumatic stress disorder with imipramine. *Psychosomatics*, **25**, 681–686.

Davidson, J. R. T. & White, H. L. (1983) The effect of isocarboxazid on platelet MAO activity. *Biological Psychiatry*, **18**, 1075–1079.

—— & Raft, D. (1984) Continuation therapy with phenelzine. *Neuropsychobiology*, **11**, 191–194.

——, McLeod, M. N., White, H. L. (1978) Inhibition of platelet MAO activity in depressed subjects treated with phenelzine. *American Journal of Psychiatry*, **135**, 470–472.

——, ——, Turnbull, C. D. & White, H. L. (1981) A comparison of phenelzine and imipramine in depressed inpatients. *Journal of Clinical Psychiatry*, **42**, 395–397.

Evans, D. L., Davidson, J. R. T. & Raft, D. (1983) Early and late side-effects of phenelzine. *Journal of Clinical Psychopharmacology*, **2**, 208–211.

Hamilton, M. (1959) The assessment of anxiety states by rating. *British Journal of Medical Psychology*, **32**, 50–55.

Hogben, G. L. & Cornfield, R. B. (1981) Treatment of traumatic war neurosis with phenelzine. *Archives of General Psychiatry*, **38**, 440–448.

HOROWITZ, M., WITNER, N. & ALVAREZ, W. (1979) Impact of Events Scale: A measure of subjective stress. *Psychosomatic Medicine*, **43**, 209–218.

KOLB, L. C., BURRIS, B. C. & GRIFFITHS, S. (1984) Propranolol and clonidine in the treatment of the chronic post-traumatic stress disorders of war. In *Post-Traumatic Stress Disorder: Psychological and Biological Sequelae*, (ed. B. A. van der Kolk). Washington, DC, American Psychiatric Press.

RAFT, D., DAVIDSON, J. R. T., WASIK, J. & MATTOX, A. (1981) Inhibition of platelet MAO and response to phenelzine in a clinical trial. *Neuropsychobiology*, **7**, 122–126.

VAN DER KOLK, B. (1983) Psychopharmacological issues of post-traumatic stress disorder. *Hospital and Community Psychiatry*, **34**, 683–685.

——, GREENBERG, M., BOYD, H. & KRYSTAL, J. (1985) Inescapable shock, neurotransmitters, and addiction to trauma: toward a psychobiology of post-traumatic stress. *Biological Psychiatry*, **20**, 314–325.

WALKER, J. I. & CAVENAR, J. O. (1982) Forgotten warriors: continuing problems of Vietnam veterans. In *Critical Problems in Psychiatry* (eds J. O. Cavenar & H. K. H. Brodie), Philadelphia: Lippincott.

WEISS, J. M., GLAZER, H. I. & POHORECKY, L. A. (1975) Effects of chronic exposure to stressors on subsequent avoidance-escape behavior and on brain norepinephrine. *Psychosomatic Medicine*, **37**, 522–524.

——, —— & —— (1976) Coping behavior and neuro-chemical changes: An alternative explanation for the 'learned helplessness' experiments. In *Animal Models in Human Psychobiology* (eds B. Serban & A. Kling).

WHITE, N. S. (1983) Post-traumatic stress disorder. *Hospital and Community Psychiatry*, **34**, 1061–1062.

WILMER, H. A. (1982) Post-traumatic stress disorder. *Psychiatric Annals*, **12**, 995–1006.

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Electroconvulsive Therapy and Cognitive Improvement in a Very Elderly Depressed Patient

A computer search of the literature revealed that the oldest documented patient to receive electroconvulsive therapy (ECT) was a 94-year-old woman (Bernstein, 1972) who was diagnosed as having 'anorexia nervosa', but the history was suggestive of paranoid schizophrenia. She received a course of five ECTs and became much less paranoid, ate well, and put on weight. Her daughter lamented the fact that ECT had been deemed to be contra-indicated 15 years earlier on the grounds of advanced age.

Depression in the elderly can be difficult to treat and relapse is common (Raskind, 1984). However, ECT appears to be at least as effective in the elderly as in younger depressed patients and it is also a relatively safe therapeutic tool in this age group (Weiner, 1982).

We now describe the treatment of a 91-year-old patient with ECT and report her improved cognitive state shortly after the treatment was completed.

Case history

E. D., whose ninety-second birthday fell on 5 May 1985, was referred for admission to our hospital by an outside psychiatrist attached to a general hospital because of 'failing cognition', 'dementia', and 'agitated behaviour'. Long term in-patient care was suggested. She had previously been admitted in September, 1984 to that hospital where she was treated by the referring psychiatrist for agitated depression with dothiepin hydrochloride and diazepam. She showed a 'slow, but steady, improvement' but her cognitive state was sufficiently suggestive of a dementing illness to warrant arranging the index admission on 15 March 1985 from the home for the aged at which she resided.

When she was admitted to our unit she was very depressed and agitated. She complained of feeling very weak and requested to be transferred to the 'Hospice for the Dying'. She had given up all hope of surviving, believed that she was not worth bothering with, and clutched to her rosary beads with fanatical strength. She was obviously in great psychological distress. A diagnosis of psychotic depression was made. On Day 1 of her admission she scored 18 on the Mini-Mental State test (Folstein *et al*, 1975; DePaulo *et al*, 1980), a test of cognitive function which can be repeated at intervals and wherein a score of less than 24 indicates cognitive disorder. It is a reliable and valid 30-point bedside examination (DePaulo *et al*, 1980).

Physical examination, apart from mild chronic obstructive airways disease and slightly reduced peripheral pulses, was unremarkable for a woman of her age.

The patient was suffering to such a great extent that we asked her if she would consent to ECT. She was unable to make up her mind and also believed that her doom was nigh. We approached her family who readily agreed to have her status changed from that of a voluntary patient to an involuntary patient—the latter is termed 'temporary' patient under our 1945 Mental Treatment Act, and they then signed a consent form for ECT.

E. D. received three applications of ECT on 22 and 27 March, and on 3 April 1985. The drugs used during the procedure were Brietal, atropine and Anectine. During the period of ECT administration she was put on prophylactic Septrin and Ventolin. She was slightly confused during, and for about 10 days after ECT. This cleared rapidly. She refused psychotropics for a week after ECT but then consented to take thioridazine 25 mg four times a day. Her depression and agitation had disappeared after the second ECT.

Serial Mini-Mental State tests were performed on 15 March (score = *18*) and on 4 April (*18*–24 h after 3rd ECT), 9 April (*22*), 10 April (*22*), 18 April (*23*), and on the day of her discharge, 1 May (*24*). She had obviously learned new material during the course of ECT since she constantly reminded us that she would pick the same items from her environment for memory testing as she had done previously. She took great pride in 'passing' the tests.

She had borne seven children, five boys remaining alive. One died at 14 months, cause uncertain, and another died at 12 years of age of a 'bladder complaint'. A 62-year-old son was a chronic schizophrenic in-patient of our hospital.

E. D. had experienced no previous serious psychiatric or physical problems before the onset of her recent depression. The latter we estimate to have started in December 1982. She improved with out-patient dothiepin over about 4 weeks and had maintained this improvement until some weeks before her first admission in September 1984.

Discussion

We have presented the case of a 91-year-old female, the second oldest in the literature to have received ECT, whose cognitive functioning improved following its administration.

Weiner (1982) points out that the effectiveness of ECT is not reduced when it is used to treat the elderly, and that if it is performed with appropriate precautions and modifications, it is a relatively safe means of treating severe depressive disorders occurring in this age group. It is not only more effective than drug therapies for many patients with severe physical illnesses, especially those with heart disease (O'Shea, 1985), but it may also be safer (Fink, 1979). Despite an increased frequency of medical problems, particularly affecting the cardiovascular system, ECT was still considered to be a safe procedure for elderly psychiatric patients in one study (Alexopoulos *et al*, 1984). Small & Mandel (1981) state that unilateral, non-dominant ECT results in transient memory deficits that are independent of the patient's age. They also state that ECT should be strongly considered in the treatment of elderly patients with endogenous symptoms who fail to respond to tricyclic antidepressants or who cannot tolerate their side-effects. Our patient received bilateral ECT—Bernstein (1972) does not give details on his 94-year-old, and she had refused to take psychotropic drugs.

In a retrospective review (Karlinsky & Shulman, 1984) of ECT in the elderly, the charts of 33 elderly inpatients (age range, 62–85 years) who received ECT in a psychogeriatric unit of a general hospital disclosed that ECT was safe but not uniformly effective. 42.4% of patients obtained an immediate good outcome. The authors felt that clinically one was still compelled to use ECT on an 'urgent and humane' basis.

In another study (Fraser & Glass, 1978) nine elderly depressed patients were given ECT in courses which alternated unilateral and bilateral electrode placement. Recovery took on average five times as long from unilateral treatment and nine times as long from bilateral treatment as had been found in younger patients. Bilateral treatment was significantly more sensitive to cumulative and interval effects in these elderly subjects. Their mean age was 72.4 years; s.d. 5.

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References

ALEXOPOULOS, G. S., SHAMOIAN, C. J., LUCAS, J., WEISER, N. & BERGER, H. (1984) Medical problems of geriatric psychiatric patients and younger controls during electroconvulsive therapy. *Journal of the American Geriatrics Society*, **32**, 651–654.

BERNSTEIN, I. C. (1972) Anorexia nervosa: 94-year-old woman treated with electroshock. *Minnesota Medicine*, **55**, 552–553.

DEPAULO, J. R., FOLSTEIN, M. F. & GORDON, B. (1980) Psychiatric screening on a neurological ward. *Psychological Medicine*, **10**, 125–132.

FINK, M. (1979) *Convulsive Therapy—Theory and Practice*. New York: Raven Press.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12, 189–198.

Fraser, R. M. & Glass, I. B. (1978) Recovery from ECT in elderly patients. *British Journal of Psychiatry*, 133, 524–528.

Karlinsky, H. & Shulman, K. I. (1984) The clinical use of electroconvulsive therapy in old age. *Journal of the American Geriatrics Society*, 32, 183–186.

O'Shea, B. (1985) Antidepressant cardiotoxicity. *Irish Medical Journal*, 78, 22–24.

Raskind, M. (1984) Electroconvulsive therapy in the elderly. *Journal of the American Geriatrics Society*, 32, 177–178.

Small, G. & Mandel, M. R. (1981) The medically ill elderly, tricyclic response, and ECT. *American Journal of Psychiatry*, 138, 703–704.

Weiner, R. D. (1982) The role of electroconvulsive therapy in the treatment of depression in the elderly. *Journal of the American Society*, 30, 710–712.

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Fictitious Epilepsy associated with Amnesia

Epilepsy can occur in patients without any changes apparent in the electroencephalogram. Thus the diagnosis of epilepsy is essentially made on clinical grounds. The clinical features of epilepsy are relatively easy to learn to mimic. Feigned epilepsy can occur in adulthood or it may occur in childhood, as part of a Munchausen syndrome by proxy as described by Meadows (1984). We would like to describe a case of feigned epilepsy with global amnesia, and several other symptoms of aberrant illness behaviour.

Case report

A woman of 30 years of age was admitted to the Royal Liverpool Hospital on 26 September 1984, brought to the Accident and Emergency Department via the Samaritans. She claimed that she had lost her memory, and alleged that she could not remember her name or any of her personal details. She said that she thought she was an epileptic, and in her handbag were bottles of anticonvulsants. Ward observers noted fits, some of which appeared genuine. Neurological examination following one such fit revealed weakness of the sixth cranial nerve and decreased sensation and weakness of the right side. Phenytoin and phenobarbitone were recommenced, and a Heminevrin infusion given for 6 days. Liver function tests revealed after 2 days raised liver enzymes: gamma-glutamyl transferase = 290 U/l (NR 0–65) and alanine amino transferase = 54 U/l, (NR 7–54). These continued to rise for 6 days to 580 and 280 U/l respectively, before gradually decreasing. She had a swinging pyrexia, and because of a history of a mastoidectomy, an ENT surgeon examined her, and prescribed antibiotics. An EEG and a repeat EEG showed no abnormality. Later she was seen holding a lighter near a thermometer. Suspicion was raised as to whether she was feigning illness and she was transferred to a psychiatric ward on the 18 October 1984.

She revealed that she had been a patient at the North Manchester General Hospital, and subsequent enquiries revealed she had been admitted there under the care of a physician in July 1984, and had only been discharged 7 days prior to her admission in Liverpool. The history then had been a very similar one of amnesia and epilepsy. As a result of her non-response, she had been prescribed an escalating dosage of phenytoin and had developed phenytoin toxicity. The character of her epilepsy was later thought to be feigned and some of the fits responded to placebo treatment. Numerous investigations including EEGs, a CAT scan and lumbar puncture proved negative.

Following extensive enquiries the following details of her personal history were obtained. She was born in Blackpool in 1954. Her father was a greengrocer, and she was their only child. She was enuretic until the age of 10 years and truanted from school. She left school at 15 and followed a pre-nursing course for one year. Her first child was born when she was 17. She married a soldier, but the marriage only lasted one year, and her second child, a boy, was born then. She worked over a long period as a taxi driver. She lived with her parents until 1980 and was drinking heavily. She had frequent contacts with the local hospital and attended various clinics, including ENT and gynaecology clinics. In 1980 she was admitted to a hospital in Blackpool following an overdose of alcohol and drugs. In November 1980 she was admitted to Lancaster Moor Hospital with a history of amnesia. Since 1980, when she left home, she has had numerous admissions to hospitals in Preston, Bolton, Leeds, Bradford, Chester, Rochdale and Manchester. She has used various names and had numerous investigations for her amnesia and fits. These include several EEGs, CAT scans, X-rays, blood tests, and lumbar punctures. These have shown no abnormality except for raised liver enzyme levels on some occasions. She is also known to have had five convictions, and was in Risley Remand Centre as recently as April 1984. At the time of her admission she was still on probation.

On admission to our unit, mental state examination revealed that her affect was bland and she appeared unconcerned about her memory loss. Formal testing of her short-term and long-term memory showed them intact. However she continued to maintain that she did not know her name or any of her personal details, except the odd titbit, e.g. she could remember being in a car accident, and that she had been divorced. There was no other cognitive defect or other evidence of mental illness. The nursing staff observed her to have some 'fits'—some of which appeared genuine, and at times she bit her tongue and was incontinent. On 24 October 1984 she presented the nursing staff with a sample of apparently blood-stained urine which she claimed to have voided. No physical cause of haematuria was found. On 19 November 1984 she went to the hospital's Casualty Department, giving a fellow patient's address. She complained of chest pain and an ECG was performed. She was on the point of being admitted to a medical ward, when she was discovered.

She was discharged on 19 December 1984 to a probation hostel in Liverpool. A few days later, on New Year's Eve, she again presented herself to the Casualty Department complaining of chest pains and haemoptysis. She was admitted to a medical ward with a suspected pulmonary embolus and heparin therapy commenced. When her identity was discovered a few days later she was discharged.

Comment

This case shows many of the typical features of a Munchausen syndrome (Enoch & Trethowan, 1979). The patient appears to be in an early phase of her career as a hospital addict; the best documented case so far being McIlroy (Pallis & Bamji, 1979). Although Munchausen Syndrome cases usually present with physical complaints, 'psychological' Munchausen Syndrome cases do exist as shown by feigned bereavements (Cremona-Barbaro, 1983). This case shows the co-existence of physical and psychological symptoms. The only physical abnormality found in this case were the raised liver enzyme levels. These findings are difficult to explain, but could be related to liver damage due to her past alcohol abuse, a recent bout of heavy drinking, or a paracetamol overdose such as she has taken in the past.

Management of Munchausen syndrome cases is extremely difficult, as it depends on the patient's motivation, which is usually concerned with more 'treatment'—they seek the mode of cure rather than the cure itself. It has been suggested that local casualty departments should be informed of such cases to aid in their detection. One possible solution is the setting up of a register, and with modern technology it would be relatively simple to keep a national database of such cases on a micro-computer.

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References

CREMONA-BARBARO, A. (1983) Munchausen syndrome. *British Journal of Psychiatry*, **143**, 524–525.

ENOCH, M. D. & TRETHOWAN, W. H. (1979) *Uncommon Psychiatric Syndromes*, 2nd ed. Bristol: John Wright.

MEADOWS, R. (1984) Fictitious epilepsy. *Lancet*, **ii**, 25–28.

PALLIS, C. A. & BAMJI, A. N. (1979) McIlroy was here. Or was he? *British Medical Journal*, **i**, 973–975.

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Ephedrine Psychosis Rediscovered

The use of ephedrine for medicinal purposes dates back over 5000 years, when native Chinese physicians first used a herb, Ma Huang, which contained the active alkaloid. Ephedrine was extracted from

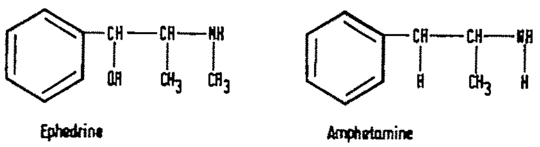

FIG. 1 Chemical structure of ephedrine and amphetamine.

Ma Huang in the late 1800s, but was considered too toxic for clinical use; it was rediscovered by Chen & Schmidt (1930), who demonstrated its sympathomimetic action and introduced it into Western medicine, where it was found to be a big improvement on adrenaline in the treatment of asthma. That the structure of ephedrine is very similar to that of amphetamine is not a coincidence, since Alles (1933) synthesised the latter as a synthetic substitute for ephedrine.

Herridge & A'Brook (1968) described two cases of ephedrine psychosis, stating that "so far as we can discover there have been no previous reports of psychosis following abuse of ephedrine." However, according to Otto & Nause (1980), "the appearance of psychoses following abuse of ephedrine has been known since the end of the 1930s and repeatedly described in the German literature in the 1940s. We report here a paranoid psychosis following increased use of ephedrine.

Case report

A 59-year-old single night watchman was brought to the casualty department by the police after entering a woman's flat uninvited. He had no history of psychiatric illness, alcoholism, or drug abuse, but had a long history of chronic asthma, for which he had taken ephedrine for 25 years; his personality was considered schizoid. There was no family history of psychiatric illness. Over the previous 2 weeks he had been working excessive overtime and had been concerned regarding recent thefts at his place of work. He was worried that he would be unable to apprehend trespassers because of his asthma and so doubled his ephedrine intake to 360 mg daily; this was taken as ephedrine hydrochloride plus 'Bronchipax' (ephedrine resinate 30 mg, theophylline 40 mg and salicyclamide 250 mg). Within 24 hours of this increase, he began to experience auditory hallucinations in the form of a woman's cries, and believed that a woman was being tortured in the flat below him. He made tape recordings of these cries and took them to the police, who did not believe him. He therefore took matters into his own hands and decided to search the flat for himself, alarming the female occupant, who called the police.

He was taken to the casualty department where he was seen by a psychiatrist and admitted under a compulsory order to a psychiatric ward. He described a 2-week history of auditory hallucinations and delusions, presenting in a setting of clear consciousness without mood disturbance. On admission, the dose of ephedrine was reduced to 60 mg. Ten days later, the patient was apparently symptom-free and was discharged, but he had been denying symptoms, and was in fact still experiencing auditory hallucinations. However, all psychotic symptoms disappeared 3 days later, and the patient gained full insight. He retrospectively described Schneiderian first-rank symptoms of thoughts spoken aloud and voices commenting on his actions and discussing him, which he had also been concealing from the staff during the psychotic episode.

Discussion

Psychosis associated with ephedrine use has been reported in a number of countries and there have been 20 case reports with adequate details. The reference to these cases are available on application to the authors.

Their ages range between 19 and 65 years. Ten were male and ten female. In only two cases was there a previous history of psychiatric illness: one patient suffered from 'neurasthenia' and another from panic attacks and hyperventilation. Only two patients had a family history of psychiatric illness, both their fathers being alcoholic. Three patients were considered to have an abnormal premorbid personality: one schizoid, one asthenic, and one obsessional. The most common reason for taking ephedrine was asthma, accounting for 60% of cases, although two asthmatics increased the dose for its stimulant effect. Two patients were drug abusers who had previously taken morphine, amphetamine and LSD, although not prior to the psychotic episode. Three patients took ephedrine purely for its stimulant effect, although they had not abused other drugs. Other reasons for taking ephedrine included hypotension, neurasthenia, and anxiety state with hyperventilation attacks where the patient believed that she was suffering from asthma. The length of consumption of ephedrine varied from 3 days to 25 years, with 80% of the patients having taken ephedrine for at least a year. In the majority of cases, the dose of ephedrine was gradually increased. The daily dose of ephedrine prior to the psychotic episode varied from 125 mg to 2500 mg, the average being 510 mg.

The main clinical features of the 20 cases are listed in Table I. The typical clinical picture is a paranoid psychosis with delusions and auditory hallucinations, in a setting of clear consciousness. This is very similar to amphetamine psychosis, with 45% of the patients experiencing visual hallucinations—a similar figure to the 50% in the report of amphetamine psychoses by Connell (1958). Clouding of consciousness was described in three patients and has also been noted in a minority of cases of amphetamine psychosis

TABLE I
Clinical features of patients with ephedrine psychosis (n=20)

Clinical feature	%
Delusions	100
Persecutory delusions	55
Delusions of reference	10
Delusions of grandeur	10
Delusions of jealousy	10
Hypochondriacal delusions	10
Religious delusions	5
Auditory hallucinations	90
Visual hallucinations	45
Tactile hallucinations	15
Olfactory hallucinations	5
Schneiderian first rank symptoms	35
Clear consciousness	85
Affective disturbance	30
Insomnia	55
Agitation	55
Suicide attempt	15

(Ellinwood, 1969). Many patients had more than one episode of ephedrine psychosis, and one experienced recurrent episodes over a 10-year period. Two patients remained psychotic despite abstinence from ephedrine, and two developed chronic defect states.

In its typical presentation, ephedrine psychosis raises some theoretical problems regarding the nosology of 'symptomatic schizophrenia'. This was discussed in 1959 by Slater in his review of Connell's monograph on amphetamine psychosis: "It is a matter of much theoretical interest how closely a pseudo-schizophrenic symptomatic psychosis may resemble the true endogenous schizophrenia . . . It seems probable that with adequate attention to specific tests, a much larger number of patients would have shown evidence of some degree of clouding of consciousness". It was thought at that time that a toxic psychosis would manifest as an acute confusional state. Apart from Kendell's work (1975) and Davidson & Bagley's (1969) paper on schizophrenia-like psychoses further discussion of the nosology of 'symptomatic schizophrenia' seems to have been swept aside in the English speaking world by the advent of the American DSM classification with its atheoretical basis. However, the appearance of ephedrine psychosis in the 1930s in Germany led to a solution being proposed in 1956, with Wieck's description of the '*Durchgangs-Syndrom*' (translated as: 'passageway' and indicating a transitory state). This was based on Bonhoeffer's ideas of the non-specificity of psychiatric symptoms (Scheid, 1962), and described a reversible organic state, without clouding of consciousness and no 'obligatory' symptom or psychopathological picture, except its transient nature and organic aetiology. It has proved to be a clinically useful concept in Germany, renewing interest in the reversible functional syndrome as a medical rather than an existential diagnostic problem. Certainly, the patient described here could retrospectively give a detailed account of his illness, indicating no clouding of consciousness, and thus representing a classical '*Durchgangs-Syndrom*'.

Ephedrine is no longer widely used as a bronchodilator, as the more specific beta$_2$ stimulants are now favoured; however, it is available without prescription and is a powerful psychostimulant. Martin *et al* (1971) compared the following drugs, administered subcutaneously under double-blind conditions: d-amphetamine, methamphetamine, ephedrine, phenmetrazine, and methylphenidate; ephedrine was found to be capable of producing the subjective and physiological effects of the other stimulants studied. Ephedrine has been marketed in the USA as 'the new legal cocaine substitute' under the name of Ma Huang Incense (Siegel, 1980). In recent years, certain 'garage laboratories' in the USA have also been manufacturing what have become known as 'look alikes'; which closely resemble amphetamine tablets in size, colour, numbering and trademark, but they contain non-controlled drugs such as ephedrine, phenylpropanolamine, and caffeine. These drugs, also known as 'pseudo-speed', are advertised quite legally in magazines and are inexpensive. Dougherty (1982) found that most of the 13 to 25-year-old drug abusers he interviewed in New York said that pseudo-speed gave them a 'rush' that was not quite as good as cocaine but sufficient to produce an adequate 'buzz', particularly when combined with alcohol; after alcohol and cannabis, pseudo-speed was the most commonly abused drug by adolescents in central New York. Young people who abuse pseudo-speed are commonly seen in emergency departments with adverse effects; e.g. one 20-year-old male was admitted one hour later with a subarachnoid haemorrhage (Wooten *et al*, 1983).

In 1965, Chapple claimed that ephedrine was being abused by young people in this country. There have been no further reports of abuse of ephedrine in the British literature, but it may nevertheless be occurring in the UK and pharmacists are aware that young people purchase ephedrine for its psychostimulant properties. In view of ephedrine's psychostimulant effects and its ability to induce paranoid psychosis, there seems to be a strong case for making it available only on prescription.

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References

Alles, G. A. (1933) The comparative physiological actions of *dl-β* phenylisopropylamines. I. Pressor effect and toxicity. *Journal of Pharmacology and Experimental Therapeutics*, 47, 339–354.

Chapple, P. A. L. (1965) New drugs of addiction (letter). *British Medical Journal*, 2, 108.

Chen, K. K. & Schmidt, C. F. (1930) Ephedrine and related substances. *Medicine*, 9, 117.

Connell, P. H. (1958) *Amphetamine Psychosis* (Maudsley Monographs) London: Oxford University Press.

Davidson, K. & Bagley, C. R. (1969) Schizophrenia-like psychoses associated with organic disorders of the central nervous system: a review of the literature. In *Current Problems in Neuropsychiatry* (ed. R. N. Herrington). London: British Journal of Psychiatry Special Publication No. 4.

Dougherty, R. J. (1982) Pseudo-speed, look alikes or pea shooters. *New York State Journal of Medicine*, 1, 74–75.

Ellinwood, E. H. (1969) Amphetamine psychosis: a multidimensional process. *Seminars in Psychiatry*, 1, 208–226.

Herridge, C. F. & A'Brook, M. F. (1968) Ephedrine psychosis. *British Medical Journal*, ii, 160.

Kendell, R. E. (1975) *The Role of Diagnosis in Psychiatry*, Oxford: Blackwell.

Martin, W. R., Sloan, J. W., Sapira, J. P. & Jasinski, D. R. (1971) Physiologic, subjective and behavioural effects of amphetamine, methamphetamine, ephedrine, phenmetrazine and methylphenidate in man. *Clinical Pharmacology and Therapeutics*, 12, 245–258.

Otto, K. R. & Nause, R. (1980) Paranoid hallucinatory psychosis following chronic Asthmolytan. *Psychiatrie Neurologie und medizinische Psychologie*, 32, 492–495.

Scheid, W. (1962) Die Lehre von den 'exogenen Reaktionstypen' vor einem halben Jahrundert und Heute. In *Psychopathologie Heute* (ed. H. Kranz), Stuttgart.

Siegel, R. A. (1980) Cocaine substitutes (letter) *New England Journal of Medicine*, 302, 14, 817–818.

Slater, E. (1959) Book review. *British Medical Journal*, i, 488.

Wieck, H. (1956) Zur klinik der sogenannten symptomatischen Psychosen. *Deutesche medizinische Wochenschrift*, 81, 1345–1349.

Wooten, M. R., Khangure, M. S. & Murphy, M. J. (1983) Intracerebral haemorrhage and vasculitis related to ephedrine abuse. *Annals of Neurology*, 13, 337–340.

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Delusion Parasitosis: Successful Non-pharmacological Treatment of a Folie-à-deux

Delusional parasitosis, originally described by Ekbom in 1938, is an illness in which the individual believes small animals such as insects, lice, vermin, or maggots are living in or thriving on his/her skin. The delusion is not secondary to another psychiatric illness, and in general the personality remains otherwise preserved. Munro (1980) classifies delusional parasitosis as a subtype of monosymptomatic hypochondriacal psychosis, and has shown that a substantial proportion of patients with MHP respond to pimozide. Prior to the introduction of that drug, the prognosis of the condition was held to be very poor.

In delusional parasitosis, *folie-à-deux* is relatively common; Skott (1978) found that 25% of her patients were involved in a *folie-à-deux*. Berrios (1985) points out that very little is known of the natural history of the illness, and suggests that the prognosis may not be as bad as is currently believed, since the evidence has generally come from the very severe and chronic cases which only come to a psychiatrist 'in extremis'. The following *folie-à-deux* of delusional infestation is reported because, as far as the author is aware, it is unique, in that remission occurred in both partners without pharmacological treatment, after only a brief period of acute psychosis.

Case report

Mr H, the dominant partner of the *folie imposée*, was a 55-year-old man, who was divorced, unemployed, and living on his own in a two-room flat. He had been referred by his general practitioner for treatment of his 'phobia', and complained that since he had laid an old carpet, 6 weeks previously, he had become infested with minuscule insects, which lived under his skin and caused intense itching and burning sensations. He had visited the Casualty and Dermatology Departments of his local hospital three times since then, with complaints of 'gritty' sensations in his eyes, and skin rashes showing 'samples' of insects for inspection. He was hurt, angry, and disbelieving when no confirmation of his infestation was forthcoming. To escape the insects, he had got rid of the carpet, had his flat fumigated, got rid of virtually all his furnishings and clothing, and finally moved to a bedsit, to no avail. He said that when outdoors, he was constantly under attack with 'black specks', which rained down on him from the air.

While firmly adhering to his view that he was infested, Mr H volunteered that he had been suffering a great deal of stress—"enough to make anyone need to see a psychiatrist". He had married at the age of 40, and this was followed 3 years, during which he exhausted himself trying unsuccessfully to obtain professional help for his wife, whom he described as irresponsible and a risk to the health of their daughter, conceived pre-maritally. Eventually, his wife left him, and took their daughter with her to the USA. Over the following 10 years, he had remained distressed and guilt-ridden about this situation. However, in 1983, 6 months after he had been made redundant from his life-long job as a clerk in a steel firm, he was informed that his ex-wife was sending his daughter to live with him. This was something he knew she did not want, and something he could not support financially; it produced great distress, and led to protracted discussions with his solicitors and with the Immigration Authorities, which were on-going at the time he developed his delusions of infestation.

His early life and development had been unremarkable, and there was no prior history of psychiatric illness; he described himself as a shy, introverted man. At interview, he talked at great length about his infestation, in an agitated fashion; apart from this delusion, there were no other abnormalities in the mental state.

Two weeks after Mr H developed this condition, his only social contact, Mr P, developed similar symptoms; he was a divorced 54-year-old man, a cardiac invalid, living a similarly isolated life to Mr H, in a flat in the same block. They had known each other for about a year, and played chess together; Mr P had continued the friendship because he felt sorry for Mr H, viewing him as a man with considerable emotional problems. Mr P said that he had begun to itch frequently, and had developed bite marks and red spots on his skin. He believed Mr H carried the insect infestation, and that close contact allowed the infestation to be transmitted temporarily to him. He had observed how bathing relieved the itching, and that his symptoms also receded when he did not see Mr H for some time; he felt Mr H had become infected because he was unhygienic. Mr P himself had presented for investigation at a dermatology clinic on two occasions; at initial assessment, the content of his speech focussed exclusively on infestation, but as much on Mr H's as his own. He was not excessively concerned about his condition, and did not press for treatment; he presented as a rather naive and suggestible man, of limited intelligence, and apart from the pre-occupation with infestation, there was no abnormality in his mental state.

Subsequently, Mr H was seen on a weekly basis over 5 weeks; a detailed history of his symptoms and life experiences was taken, and a thorough medical work-up carried out, including further assessment by a dermatologist. At no point was it suggested that Mr H was psychiatrically ill; he found this approach supportive, was appreciative of his treatment, but equally complained that he was not getting the specific help he required for the infestation. During this time, he continued his own self-help efforts to eradicate the infestation by methods such as fumigation and repeated bathing; after 5 weeks, he indicated that he felt he had finally cured himself. He described how the infestation had finally left him after two trips to the public baths, during which he had seen the insects hover around him in a white cloud, before eventually leaving him; at this point, he indicated he wished for no further interviews with the author.

Following the initial assessment, Mr P deliberately pursued a policy of avoiding contact with Mr H, and a fortnight after Mr H was cured, he indicated he was free of his symptoms. Mr H was seen a year later, in response to a request by the author; he had remained symptom-free over this time, while remaining convinced that he had been infected by some insects a year previously. At this time, Mr P was also symptom-free. Mr H now spontaneously asked for counselling and practical advice from the authors regarding his continuing distress over the family traumata, and is currently seeing the author on an infrequent basis.

Discussion

To the author's knowledge, no report of non-pharmacologically induced remission of delusional parasitosis has previously been documented. Several features may account for the favourable short-term prognosis in the case of Mr H. Firstly, the onset of his symptoms was acute, and led to a rapid involvement of potentially therapeutic agencies; many patients have been chronically deluded, sometimes for up to 20 years, before they come to the attention of psychiatrists. Secondly, and atypically, treatment could be directed both at the symptomatic and (the putative) psychological levels of Mr H's problems. He was supported in his request for medical investigation, and was encouraged to ventilate at length his distress and conflicting feelings surrounding the life experiences which he felt had distressed him over some years. Each of the therapeutic sessions was followed by a decrease in his level of distress about his emotional problems—a sense on his part that his problems were seen as important in the eyes of a significant other. Thirdly, he retained his sense of his own agency, through his own self-help measures: he maintained a belief that some measure would be effective eventually in getting rid of his infestation, and each cleansing technique generally had a short-term therapeutic effect, calming and temporarily cleansing him.

That this supportive type of approach was effective should not be totally surprising; prior to the advent of phenothiazines, undoubted cases of schizophrenia responded to supportive, non-pharmacological interventions. What is surprising is rather that no cases have been reported similar to the above: this may be because many cases go undetected, and long-term follow-up of the natural history of the condition is virtually absent. Equally, cases seen by psychiatrists are loaded towards those where organic factors such as dementia are likely to be present (Berrios, 1985).

The remission of the illness in the second partner followed separation from Mr H. The transient nature of the symptoms, their relative mildness, and their relationship to contact with the itching partner, make it likely that this illness represents an over-valued idea, rather than a primary delusional illness. The implication of this case of *folie-à-deux* is that the spectrum of illness subsumed under the label delusional parasitosis, is wider and of more variable prognosis than has heretofore been considered.

References

Berrios, G. E. (1985) Delusional parasitosis and physical disease. *Comprehensive Psychiatry*, **26**, 395–403.

Ekbom, K. A. (1938) Der Presenile Dermatozoenwahn. *Acta Psychiatrica et Neurologica Scandinavia*, **13**, 227–259.

Munro, A. (1980) Mono-symptomatic hypochondriacal psychosis. *British Journal of Hospital Medicine*, **24**, 34–38.

Skott, A. (1978) Delusions of infestation. *Reports from the Psychiatric Research Centre*, No. 13. Sweden: St Jorgen Hospital.

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This *Journal* accepts papers from any part of the world provided they are of appropriate scientific quality. The only exception would be where there had been evidence in a paper from any country (by no means only South Africa) that unacceptable practices of political, racial, or other discrimination or abuse had been followed in the course of the work reported – *Ed.* (see Richman, N., *Journal*, 1986, **149**, 797).

Admission Rates and Lithium Therapy

SIR: Dickson & Kendell (1986) have reported their observations in Edinburgh. From 1970 to 1981, while long-term lithium therapy was increasingly employed for affective illness, the admission rates for affective disorders rose substantially. The authors find the explanation of these changes uncertain, yet they conclude that the findings cast doubt on the efficacy of lithium prophylaxis under ordinary conditions.

The question of lithium efficacy in clinical practice is very important. The authors' observations are interesting, and their discussion scholarly, but it remains unclear how their failure to explain the rise in admission rates could lead them to question the efficacy of lithium treatment in practice. As the discussion part in their paper leaves several important issues open to further questioning, connecting directly the two increases they observed appears unfounded and rather arbitrary.

The possibility of a shift in diagnostic practice may serve as one example of an unanswered question. Their data shows that while the admission rates for mania and depression rose steadily, the admission rates for schizophrenia fell. As the total number of admissions remained approximately the same, a shift in diagnostic practice would be one of the possible explanations. In many other hospitals the diagnosis of manic-depressive illness increased substantially in the 1960s and 1970s, probably because of changed therapeutic expectations (Baldessarini, 1970; Symonds & Williams, 1981). Thus, this issue is obviously critical for any conclusions about the efficacy of lithium. The authors therefore compared a sample of 20 new admissions and 20 readmissions, from both the first 3 years and the last 3 years, as to the proportion of patients fulfilling the RDS criteria for the main diagnostic categories, and they felt that these had not changed. However, this does not fully dispose of the concern; and a shift in diagnostic practice remains a strong possibility. For one thing, one would require a considerably larger patient sample to detect a statistically significant shift in diagnostic proportions. Secondly, the number of diagnosed manics has increased approximately three times, a change similar in type to the diagnostic shift described elsewhere (Baldessarini, 1970). Finally, although the data present a ten-fold increase in lithium use in Edinburgh, the authors did not find any such change in the sub-samples selected, which certainly casts doubt on how representative the sub-samples are and how relevant they are for interpretation.

In attempting to explain their findings, Dickson & Kendell had to face difficulties common to most retrospective studies. As they address an important question, one can easily identify with their predicament, as well as share their view that the explanation of the findings is uncertain. There are always major risks involved in attempting to make inferences about therapeutic efficacy from epidemiological data (Shepherd, 1961). To wit, in Hamilton, Ontario, we have observed admission changes similar to those reported by Dickson & Kendell, yet the data suggest a different interpretation. From 1970 to 1981 – a period of time identical with that of the Edinburgh study – in Hamilton Psychiatric Hospital there was a similar increase in admission rate of patients with the diagnosis of major affective disorders: from 111 to 190. This increase was paralleled by a rise in admission of these patients in all hospitals in Ontario, from 1036 to 2105 during the same time period. As in Edinburgh, the admissions of schizophrenics to Hamilton Psychiatric Hospital dropped from 520 in 1970 to 435 in 1981. The use of lithium in the area also increased dramatically, reaching in the early 1980s a rate of 2 per 1000 population; over 700 patients were on lithium in the Hamilton area, a similar figure to that in Edinburgh. Yet, despite these striking similarities with the Edinburgh trends, a cohort of 240 lithium treated patients at Hamilton Psychiatric Hospital exhibited concurrently a profound drop in expected recurrences, described in our earlier studies.

The important question of lithium efficacy in clinical practice could perhaps be approached more productively by following admissions of a clinical practice cohort of patients who actually are on lithium.

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References

BALDESSARINI, R. J. (1970) Frequency of diagnoses of schizophrenia versus affective disorders from 1944 to 1968. *American Journal of Psychiatry*, **127**, 759–763.

DICKSON, W. E. & KENDELL, R. E. (1986) Does maintenance lithium therapy prevent recurrences of mania under ordinary clinical conditions? *Psychological Medicine*, **16**, 521–530.

SHEPHERD, M., GOODMAN, N. & WATT, D. C. (1961) The application of hospital statistics in the evaluation of pharmacotherapy in a psychiatric population. *Comprehensive Psychiatry*, **2**, 11–19.

SYMONDS, R. L. & WILLIAMS, P. (1981) Lithium and the changing incidence of mania. *Psychological Medicine*, **11**, 193–196.

SIR: We agree with Schou (*Journal*, December 1986, **149**, 798–799) and Grof (above) that the findings we reported in *Psychological Medicine* last year (**16**, 521–530) do not by any means prove that prophylactic lithium therapy is ineffective, even in our own city. We ourselves emphasised that we could not exclude a number of possible explanations, including changing diagnostic criteria, for the threefold rise in the admission rate for mania that occurred between 1970 and 1981. On the other hand, we failed to find any evidence to support any of these alternative explanations. The samples of case notes we compared (40 from 1970–72 and 40 from 1979–81) yielded no hint either that diagnostic criteria had changed or that the threshold for admission had fallen between these two time periods. We are aware, of course, that diagnostic criteria for mania changed very dramatically in North America in the course of the 1970s. But Baldessarini's comments on the American scene cannot be extrapolated to Scotland. In many parts of the USA a diagnosis of mania was a rarity in the 1960s but this was never so in the UK. For example, in the comparison of admissions to mental hospitals in New York and London carried out by the US/UK Diagnostic Project in 1968 only 0.5% of the New York patients had a hospital diagnosis of mania compared with 6.9% of the London patients (Cooper *et al*, 1972). What is more, Eagles & Whalley (1985) found no significant increase in the first admission rate for mania to Scottish mental hospitals between 1969 and 1978 and it is difficult to see how any major change in Scottish criteria for a diagnosis of mania could have occurred without affecting that rate.

We do not pretend to understand why the admission rate for mania should have increased so much during a time period when the use of lithium was steadily increasing, but we are impressed by the evidence, which neither Schou nor Grof refers to, that lithium withdrawal, deliberate or inadvertent, may result in a temporarily *increased* risk of a manic episode. There are at least four reports in the literature of patients relapsing within a fortnight of their normal lithium tablets being replaced by placebo, and it is not far-fetched to suggest that patients may, for a variety of reasons, end up taking lithium intermittently more frequently under the conditions of ordinary clinical practice than in the context of a closely supervised clinical trial.

We published our findings not to deter others from putting their patients on prophylactic lithium but in the hope that they would provoke them to ask questions about mania and about lithium which they had not asked previously, and to design new studies to answer those questions. In the meantime we cannot do better than repeat the last sentence of our paper – "whatever the true explanation, there is no comfort in these findings for those, including ourselves, who have believed for the last 15 years that maintenance lithium provides an effective prophylactic treatment for at least a substantial minority of patients with recurrent affective disorders".

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References

COOPER, J. E., KENDELL, R. E., GURLAND, B. J., SHARPE, L., COPELAND, J. R. M. & SIMON, R. (1972) *Psychiatric Diagnosis in New York and London*. Maudsley Monograph No. 20. Oxford: Oxford University Press (p. 100).

EAGLES, J. M. & WHALLEY, L. J. (1985) Decline in the diagnosis of schizophrenia among first admissions to Scottish mental hospitals from 1969–1978. *British Journal of Psychiatry*, **146**, 151–154.

Panic Attacks: New Approaches to an Old Problem

SIR: Gelder's paper (*Journal*, September 1986, **149**, 346–352) should not be given more weight than it claims, as a somewhat ephemeral expression of his picture of the subject and reflecting his well-known interest in behavioural psychotherapy. However, there is the danger that some readers might mistake it for a serious appraisal of the subject, placing new ideas in relation to a review of the old ones. In particular, since the paper begins and ends with approving references to Freud, some readers might not realise the almost total omission of everything that Freud thought important on the subject. There is a case for expunging his rather dotty theories of 1895 but it seems a pity to try and understand panic attacks without any recourse whatsoever to the insights available from 90 years of psychoanalysis.

Gelder reminds us that Freud coined the term 'anxiety neurosis' in a very early (1895) paper entitled "The justification for detaching from neurasthenia a particular syndrome: the anxiety neurosis." Gelder summarises some biochemical and neurophysiological hypotheses and a theory involving hyperventilation. Turning to "psychological mechanisms", he does say that they may be equally complicated but "for the present discussion it will be enough to consider one important psychological component: the cognitive changes." This is the only hint that there might be any more to it, any more in the way of human experience relevant to the problem. In particular, there is no glimmering of what Freud's paper was about. The fact is that he was on the verge of discovering the unconscious, the interpretation of dreams and inventing psychoanalysis. In the particular paper quoted, he thought he had discovered something rather different, a theory of "actual neuroses", syndromes due to adverse sexual practices. Almost the entire paper to which Gelder refers is devoted to an exploration of the idea that "anxiety neurosis" is caused by frustrating sexual habits and by coitus interruptus in particular. He thought that neurasthenia, his other "actual neurosis", was caused especially by excessive and compulsive masturbation. These seemingly naive ideas may have made more sense in 1895 but were soon to give way to the larger insights into the workings of the unconscious that changed the shape of the planet for every literate person since. The early stumblings of a genius are fascinating to follow and Freud's *obiter dicta* had a way of getting into everyone else's language afterwards.

After all this it is rather astonishing that Gelder, asking whether the psychological component of neuroticism in panic attacks can be identified more precisely than cognitive theory permits, recalls only that "Freud (1895) stressed the anxious patient's preoccupation with fears of dying, a stroke, or loss of sanity." He adds that Beck *et al* (1974) added fears of heart disease and fainting, which sounds like an observation of almost sublime triviality – the list of expressed fears must be almost endless. Surely we could grasp Freud's point in first describing "anxiety neurosis": far from stressing the presenting symptoms, so oppressive to the patient, a psychiatrist does well to look deeper. It is a weakness of cognitive therapy that it tends to make psychology thoughtless and mindless. Of course, cognitive theory can and should take account of fantasy life and unconscious thought but too often it does not.

I find it piquant that in these very early papers Freud's approach is closer to that of the behaviour therapist than latter-day behaviour therapists seem to notice. He was then very interested in behavioural modification as a two-way interaction with thought modification and he certainly wasn't above telling people what to do. He must, after all, have been one of the astutest clinical observers and most powerful intuitive therapists ever, even though he did warn us of the dangers of *furor therapeuticum*. Furthermore, being originally a celebrated neurologist, he remained all his life deeply preoccupied with the mind–body interrelation, always wondering about the physical basis of mental processes. Perhaps we may look forward to Gelder's re-appraisal of Freud, the behaviour therapist.

Finally, Gelder gives the reference to Freud's paper as it appears in the *Collected Papers* (reprinted 1940). We probably share a sentimental attachment to those old editions but younger readers would be better referred to the Standard Edition of Freud, published jointly by the Hogarth Press and the Institute of Psychoanalysis. Strachey's translation has been criticised but remains a definitive master-work. The editorial introductions to every item are indispensable for anybody liking to place the text in its context and time.

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Psychotherapy and Placebo

SIR: Michael DeMowbray (*Journal*, November 1986, **149**, 666) returns to our debate concerning psychotherapy and placebo treatment by suggesting that "the allotment of the various therapeutic factors to the categories 'specific' and 'non-specific'... is purely arbitrary and relative to the theoretical standpoint of the investigator concerned". This surely is a complete mis-statement of the position taken by psychotherapists in general. Whether they embrace the principles of psychoanalysis or gestalt, or whatever, they start with a theory concerning the origins and nature of neurosis, and derive methods of psychotherapy from these principles. It is common knowledge that these principles vary widely from one school to another, and the fact that all are equally successful (or unsuccessful) and do not do better than placebo treatment surely demonstrates once and for all that the theories involved are erroneous, and that the treatment effects are in that sense non-specific. Similarly, the fact that behaviour therapy is significantly more successful suggests that its effects are specifically derived from, and verify, the theory on which these treatments (desensitisation, flooding with response prevention, modelling, etc.) are based. All this does not relate to the theoretical standpoint of the investigator; he is simply testing the hypotheses of different psychotherapists, and assumes a neutral standpoint, as is appropriate for the research scientist.

When Smith, Glass & Miller (1980), in their book on *The Benefits of Psychotherapy,* discover, on the basis of a meta-analysis of all published studies, that there is no relationship whatever between success of therapy and duration of therapy, or between success of therapy and duration of the training of the therapist, we must surely begin to doubt the wisdom of advocating lengthy training of therapists, or lengthy treatment of patients. I have argued (Eysenck, 1980) that all psychotherapy and behaviour therapy treatments, as well as placebo treatments and spontaneous remission effects, rely on identical psychological principles, namely those underlying Pavlovian extinction of conditioned emotional responses, and that the superiority of behaviour therapy is dependent on its recognition of these principles and their explicit use in designing the treatment. The various psychotherapies, placebo treatments and spontaneous remission effects make use of these principles in a random and indiscriminate manner, and are hence less successful. Also, orthodox Freudian therapy contravenes these principles, and hence frequently harms, rather than cures, the patient (Strupp *et al,* 1977). It seems important to me that these facts should be known, and that they should be taught to budding psychiatrists. There is still an aura of successful achievement surrounding psychotherapy which is quite unjustified by all the available evidence (Rachman & Wilson, 1980). Similarly, the vast majority still spend a great deal of time being taught principles of psychotherapy which the evidence suggests are, in fact, untrue. These are serious matters, concerning the health and happiness of countless individuals, and it is important that they should not be swept under the carpet but discussed and debated on a factual basis.

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References

SMITH, N. L., GLASS, G. V. & MILLER, T. (1980) *The Benefits of Psychotherapy*. London: Johns Hopkins University Press.

EYSENCK, H. J. (1980) A unified theory of psychotherapy, behaviour therapy and spontaneous remission. *Zeitschrift für Psychologie,* **188,** 43–56.

STRUPP, H., HALLEN, S. & GOMEZ-SCHWARTZ, R. (1977) *Psychotherapy for Better or Worse*. New York: Jason Arrenson.

RACHMAN, S. J. & WILSON, G. D. (1980) *The Effects of Psychological Therapy*. London: Pergamon Press.

Suicide in Physicians

SIR: In their paper on "Suicide in young doctors" (*Journal,* October 1986, **149,** 475–478), Richings *et al* revisit the troubling problem of the apparent vulnerability of members of the medical profession to suicide. During a sabbatical year spent in England, I used similar published and unpublished data recorded by the Office of Population Censuses and Surveys (OPCS) for 1970–72. Deaths among physicians and their wives from suicide, liver cirrhosis, accidents, poisoning, violence, and all causes combined were compared with deaths among other occupational groups (Sakinofsky, 1980*a*, 1980*b*). The standardised mortality ratio (SMR) for suicide in male physicians aged 15–64 was 335 and in those aged 65–74 was 155. Of the 15 occupational groups studied, male physicians (aged 15–64) ranked third for suicide, after male pharmacists (SMR 464) and unskilled labourers (SMR 370). Members of the political bureaucracy ranked lowest (SMR 34) after the clergy (SMR 51) and university teachers (SMR 56).

I was not able to study suicide in married female doctors owing to the peculiarity that married female physicians were at that time frequently classified by the registrars of deaths according to the occupations of their husbands! However, like Richings *et al,* I did find an excessive SMR (257) among unmarried female physicians aged 15–64 (based, however, on only 3 deaths recorded between 1970–72). Here again, far higher SMRs were recorded for female pharmacists (763 based on 3 deaths) and unskilled labourers (623 based on 18 deaths). Caution should obviously be used in interpreting SMRs based on such small numbers as are indeed also present in the data of Richings *et al.*

A further group investigated in my study was that of doctors' wives, and again I found excess mortality from suicide (SMR 458 based on 31 deaths aged 15–64) as well as from accidents, poisoning, and violence (SMR 322 based on 62 deaths). These ratios considerably exceeded those from deaths of wives of husbands with other occupations, including pharmacists (SMR from suicide 141 and from accidents, poisoning and violence 98).

It would be an oversimplification to suggest that excessive suicidal mortality in doctors and their wives is simply due to their special knowledge about lethal means as well as the ready availability of such means. However, the even greater suicidal ratio for male pharmacists clearly suggests that these must be significant factors. On the other hand, if being a doctor or a doctor's wife carries with it a special kind of distress loading, clearly the physician is not unique in this either. Being an unskilled worker may be a worse predicament. By the same token, there appear to be some professions (e.g., the clergy, politicians) that are spared excessive risks of suicide. If we are to understand why doctors and their wives are at higher risk for suicide, we might benefit from undertaking in-depth comparisons of their demographies, lifestyles and value systems with those of other occupational groups.

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References

SAKINOFSKY, I. (1980*a*) Suicide in doctors and wives of doctors. *Canadian Family Physician*, **26**, 837–844.

——(1980*b*) Suicide in doctors and their wives. *British Medical Journal*, **281**, 386–387.

Mania Following Bereavement

SIR: In his letter (*Journal*, August 1986, **149**, 244) Bridges raises interesting points apart from, rather gallingly, misreading our intendedly ironic "Freudian" reference (*Journal*, April 1986, **148**, 468–70). He claims that in opposing grief and mania we create a false paradox in our patient's manic sequel to bereavement. If it is argued that grief and mania are not opposites then, by Bridges' own statements, grief and depression should have no relation. The disjunction of grief and depression is a little more problematic. The phenomenological closeness of these conditions is embedded in DSM-III, which requires the phenomena of "major depression" not to be caused by grief (p. 214). I do not believe DSM-III necessarily embodies 'truth', but it does conform to a body of respectable opinion, as do concepts of pathological grief, and to the extensive literature on bereavement and depression. Grief may be a normal experience, but it is not clearly differentiated from depression except by the presence of bereavement or loss. Bridges' placement of "happiness" as the polar opposite of grief is no less suspect than our opposition of grief and mania, or his separation of grief and depression. Only a psychiatrist who dealt with the extremes of illness would have the luxury of seeing such clear separations.

One of the worst sins of the analytic movement was to treat the hypothetical entities of "defences" as real or phenomenological entities. Bridges refers to "mani[a]-an illness" with a conviction that similarly treats the hypothetical entity of illness as a real or phenomenological entity. Moreover, psychoanalytic theory offered trite and circular 'explanations' for many mysteries of human behaviour and thereby closed them to investigation for decades. To speak of "specific vulnerability/non-specific stress' creates an illusion of explanation which threatens to do the same.

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The Prognosis of Depression in Old Age

SIR: Baldwin & Jolley (*Journal*, November 1986, **149**, 574–583) point out that "much thought needs to be given to research methodology in this field." I could not agree more; the authors' retrospective study of case notes can hardly be regarded as rigorous or comparable with a prospective follow-up study of the type I conducted. It is well recognised that case note reports of outcome frequently bear no relation at all to the mental states discovered in face-to-face interviews and no doubt the same vagaries of reporting afflict doctors' notes in Manchester as they do in East London. Baldwin & Jolley did, however, interview those alive for their long-term study. They reported a mortality rate (35%) remarkably similar to the 37% mortality rate of the East London cohort over a 4 year period (in press). If the dead are excluded, then 40% of their patients remaining alive were either 'continuously ill" or suffering from "depressive invalidism". No different from Post's findings and not much different from mine!

It seems to me that where our methodology is similar, the results are similar, though perhaps they view the same results with more optimism. Baldwin & Jolley's pint pots are always half-full, whereas mine are half-empty!

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Depression in School Phobia

SIR: We note with concern the comments of Weinberg *et al* (*Journal*, March 1986, **148**, 335). Some of the phraseology suggests that their views may be influenced by considerations beyond the substance of the Newcastle method. They enunciate standards which are easier to apply to the work of others and also over-emphasise shortcomings which we have already listed. In the selection of diagnostic criteria, we had pointed out (p. 353) that, as there was no control group (normal controls), the items identified would not necessarily discriminate between those who were well and those who were depressed.

As to his other points: (i) We believe it is valid to attempt to distinguish between neurotic disorders in childhood with and without depression, but have stated that further work needs to be undertaken to establish whether our formula can be applied more widely. Recent research on a fresh population has validated the formula. (ii) Reliability has been previously reported (p. 355 and Berney *et al* (1981). (iii) We are aware of the evidence for a genetic basis for bipolar affective disorder, but the case for this is less strong in the milder depressive disorders which present in out-patient practice. (iv) The theory of an underlying substrate of right cerebral dysfunction is controversial and cannot be supported as yet.

We regret the confusion about the origin of the Weinberg criteria, which derive from the Washington University research criteria. However, other workers (Puig-Antich, 1980) have already pointed to serious methodological flaws in the use of these criteria by Weinberg. By RDC criteria we imply the variation used by Puig-Antich. The current Newcastle Depression Project has looked again at the various sets of diagnostic criteria in relation to a clinic population (Kolvin *et al* – in preparation).

We also note with interest the comments by Atkinson (*Journal,* March 1986, **148,** 335–336). There are some major as well as some simple misunderstandings of the procedures used. We disagree with some of the views expressed. He also seeks extensive statistical details which are not usually published.

Comparison of groups (Table I). It is axiomatic that statistical critics should get their formulae right. On Atkinson's own analysis, a 31-item total global depressive score would lead to a probability of 1.5 and there is no such thing! Further, the formula would need to be modified when using higher levels of significance.

Initially our data in Table I was presented both as percentages and as means and standard deviations. Subsequently we used the device of the best cut merely for the ease of presentation. We agree that in the comparison of the groups it would have been best for us to make a cut on *a priori* theoretical grounds. The above use of the term 'best cut' when comparing groups has given rise to confusion.

Best cut approach. We agree this is best confined to the technical sense of identifying the optimum point for discrimination on a scale used for screening purposes. We merely revised the threshold of the cut so as to identify the optimum point for the purpose of discriminating cases from non-cases. Making a cut on *a priori* theoretical grounds does not allow the emergence of the most sensitive discrimination. This is a legitimate technique and does not inflate the discriminant efficacy of the measures in question.

Item analysis. The ideal sample would have included children matched for age and sex from the general population, but this was not feasible.

Use of weights derived from discriminant function analysis. Discriminant function analysis was used to explore a series of hypothesised classifications. Data from our multivariate analyses did not contribute to our diagnostic formula. We have not used weights derived from discriminant function analyses. This is a laborious technique which offers no clear advantages over simpler methods (Goldberg, 1972).

Principal component analysis. (i) This is a robust technique well suited to clinical data which may not fit classical assumptions of multi-normality. Atkinson cites a ratio (of subjects to variables) of 5 : 1, but others recommend a ratio of not less than three or four times as many observations as variables (Taylor, 1977). It is true that we interpreted this rather liberally in relation to social and family background data, but we were cautious in the conclusions we drew. While in PCA the ideal sample size should be relatively large, we again contend that it can be used as an exploratory tool on a moderately sized sample. Dogmatic statements about sample size suggest an inflexibility in the use of statistics. (ii) In the first PCA the correlation matrix was only moderately consistent with an underlying structure and our criterion for determining the number of components was that of a variance which stood out as far larger than the rest. This is a pragmatic and arbitrary approach but more sophisticated techniques are inappropriate in a small sample (Lawley & Maxwell, 1971). In the second PCA the correlation matrix proved more consistent with an underlying structure and the first two components accounted for a substantial amount of the variance. A retrospective analysis, using the rule whereby the number of factors extracted is equal to the number of Eigen values greater than unity, supported our decision. We did not use rotation because the '*n*' was moderate. While some of the factor loadings which were noted were rather low, this does not detract from the differentiating bipolar second component which contrasts affective symptomatology with premorbid personality traits.

As clinical investigators we have presented the early steps in the development of a set of diagnostic criteria – fully aware of the pitfalls of our method. We were equally aware both of the importance of using scarce opportunities to their full, and of the necessity to ensure further validation and replication on fresh samples.

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References

BERNEY, T. B., KOLVIN, I., BHATE, S. R., GARSIDE, R. F., JEANS, J., KAY, B. & SCARTH, L. (1981) School phobia: a therapeutic trial with clomipromine and short-term outcome. *British Journal of Psychiatry*, **138**, 110–118.

GOLDBERG, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. Maudsley Monograph. Oxford: Oxford University Press.

LAWLEY, D. N. & MAXWELL, M. A. (1963) *Factor Analysis as a Statistical Method*. London: Butterworth.

PUIG-ANTICH, J. (1980) Affective disorders in children. *Psychiatric Clinics of North America*, 3, 403–424.

TAYLOR, C. C. (1972) *Principal Component and Factor Analysis in Exploring Data Structures*, Vol. 1. (eds C. A. O'Muircheartaigh & C. Payne). New York: Wiley.

Psychiatric Morbidity and the Mentally Handicapped

SIR: Day (*Journal*, December 1985, **147**, 660–667) reported that 30% of mentally handicapped residents aged over 40 years and 20% of those admitted to a psychiatric day hospital for the mentally handicapped aged over 40 years had a significant psychiatric disorder. This study contains artefacts which lead to an over-estimation of the prevalence of psychiatric disorder.

No information is given concerning reliability. Poor reliability may have occurred at the time of diagnosis, during transcription of diagnoses from case notes, when diagnoses were re-classified into five super-ordinate categories (e.g., into the categories "psychosis" vs "psychosis"), and when diagnoses were made from case notes only (as in 6% of cases).

If the aim of the study was to estimate the current prevalence of psychiatric disorder then the inclusion of people with a history of psychiatric disorder clearly inflates this. For example, people may have previously suffered a single episode of an illness or may have been treated and no longer show the disorder.

Day gives no formal definition of "behaviour disorders" although he refers to examples of this. Whilst some instances of such behaviours have been shown to be associated with specific disorders (e.g., Lesch-Nyhan syndrome) it is not clear what *proportion* of such behaviours are indicative of psychiatric morbidity. Some behaviour disorders are associated with painful physical illness such as otitis media, undetected dental abscesses and chronic nasal infection. Others may be considered examples of learned behaviours. Behaviour disorders are clearly of heterogeneous origins and only some are psychiatric in nature (Jacobsen, 1982).

Finally, Day includes a number of offensive and troublesome behaviours as psychiatric morbidity. These include behaviours such as wandering, stealing, and public masturbation. Such problems may not reflect psychiatric morbidity in all cases but a variety of other problems such as lack of privacy, poor learning history or an environment which maintains aberrant behaviours.

Behaviour disorder accounted for 50.5% of psychiatric disorders in the long-stay residents and 35.5% of the day hospital admissions. If a substantial proportion of these could not be demonstrated to be psychiatric in nature then the prevalence of psychiatric morbidity would fall substantially.

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JACOBSEN, J. W. (1982) Problem behaviour and psychiatric impairment within a developmentally disabled population. I: Behaviour frequency. *Applied Research in Mental Retardation*, 3, 121–129.

The Effect of Sulpiride on Negative Symptoms of Schizophrenia

SIR: Activating or disinhibitory effects of neuroleptics given in low doses to patients with schizophrenia were described when these drugs were first introduced to psychiatry (Delay *et al*, 1957), but subsequent experience with conventional anti-psychotic drugs has failed to provide convincing evidence of a useful dose-related bipolarity of effect. From the time of its original use in psychiatry, however, subsidiary effects of sulpiride – described in a variety of terms such as "anti-autistic" or "thymo-analeptic" have repeatedly been noted (Collard, 1969), and neuropharmacological studies (Sokoloff *et al*, 1980; Brown & Arbuthnott, 1983) suggest sulpiride may show a clinically useful separation of dose-related effects on psychiatric symptoms, low doses having activating and/or antidepressant properties, while higher doses are effective against positive symptoms of schizophrenia.

Since there are no controlled studies in this area, we have undertaken a double-blind comparison of normal versus low dose sulpiride in patients with chronic schizophrenia characterised predominantly by the negative symptoms poverty of speech and flattening of affect.

Six women and 11 men, aged 18 to 35 (mean = 27), with a diagnosis of schizophrenia according to Feighner's diagnostic criteria for 2 to 12 (mean = 7) years, and complying with Wing's 1961 criteria for negative symptoms, were randomly allocated on a double-blind basis to treatment with sulpiride at a dose of either 50 mg ($n=9$) or 400 mg ($n=8$) three times daily. All patients completed 30 days, and 12 (low dose $n=7$, normal dose $n=5$) completed 60 days of treatment.

Brief Psychiatric Rating Scale (BPRS) and Clinical Global Impression (CGI) scores were obtained at standard intervals during the course of the study, and the patients were examined daily for side-effects. Prior to the study, two patients had never had drug treatment, and the remainder had received no drugs for a mean of 82 days (range 7–540).

Only the low dose patients underwent significant improvements, both clinically and statistically. At 30, 45, and 60 days the BPRS mean total had decreased significantly from the Day 1 value, and analysis of symptom scores showed this improvement was due mainly to a fall in the portion of the total score contributed by the items 'emotional withdrawal' and 'blunted affect', i.e. the negative symptoms. After 30 days' treatment, mean 1200 mg versus 150 mg dose negative symptoms sub-totals were 10.5 vs 7.7 (Day 1 scores 10.8 vs 9.7), t-test (2-tailed) between changes of each score from Day 1 significant at $P<0.01$; after 60 days, 10.0 vs 2.7, $P<0.01$. The CGI, which would have been largely determined by the predominantly negative symptom profile of each patient, showed a similar result of significant improvement from Day 30 onwards restricted to the low dosage group. Two patients on normal dosage experienced an episode of acute dystonia at the start of treatment, which resolved spontaneously; there were no other significant side-effects.

Full details of the study are being submitted for publication. However, in recent years, particularly as a result of the work of Johnstone *et al* (1978) on the isomers of flupenthixol, it has been considered increasingly likely that the beneficial effects of conventional neuroleptics are restricted to positive symptoms (delusions, hallucinations, and thought disorder) of schizophrenia, and we think this result with an atypical anti-psychotic drug is of interest.

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References

DELAY, J., DENIKER, P., GREEN, A. & MORDRET, M. Y. (1957) Le syndrome excitomoteur provoqué par les médicaments neuroleptiques. *Presse Médicale*, **65**, 1771–1774.

COLLARD, J. (1969) Le sulpiride, un neuroleptique singulier, anti-autistique et thymoanaleptique. *Sem. Hôpitaux Paris*, **45**, 3028–3031.

SOKOLOFF, P., MARTRES, M. P. & SCHWARTZ, J. C. (1980) Three classes of dopamine receptors (D-2, D-3, D-4) identified by binding studies with ^{3}H-apomorphine and ^{3}H-domperidone. *Naumyn-Schmiedeberg's Archives of Pharmacology*, **315**, 89–102.

BROWN, R. J. & ARBUTHNOTT, G. W. (1983) The electrophysiology of dopamine (Dé) receptors: a study of the actions of dopamine on corticostriatal transmission. *Neuroscience*, **10**, 349–355.

JOHNSTONE, E. C., CROW, F. J., FRITH, C. D., CARNEY, N. W. P. & PRICE, J. S. (1978) Mechanism of the anti-psychotic effect in the treatment of acute schizophrenia. *Lancet*, **22**, 848–851.

Allergic Type Response to Trazodone

SIR: Trazodone is a triazole pyridine derivative unrelated in structure and pharmacology to the tricyclics, tetracyclics and monoamine oxidase inhibitors. It has been associated with dermatological reactions including erythema multiforme (Ford & Jenike, 1985) and leukocytoclastic vasculitis (Mann *et al*, 1984). We wish to report what appears to be a previously undescribed reaction.

Case report: C.M. is a 52-year-old male executive with a history of myocardial infarction in 1972 and 1983. He underwent triple bypass surgery in 1984 and has been physically well since. Six weeks prior to presentation he was involved in a minor road traffic accident with no physical injuries. He began to complain of persistent anxiety, marked insomnia, loss of confidence, lethargy, social withdrawal, irritability, loss of interest in his job, and consistent depression with diurnal variation. He had no previous personal or family history of psychiatric disorder. A diagnosis was made of a depressive anxiety state precipitated by a traumatic episode. In view of his cardiac status he was commenced on trazodone (50 mg t.i.d.). He was on no other medication. Within 24 hours he noted swelling of the index finger of his right hand and within a further 24 hours both his hands became markedly swollen to the extent of his being unable to make a fist. He reported this to the local casualty officer but neglected to mention that he was on medication. Observation was advised and no medication was prescribed. He continued to take trazodone and within a further 24 hours both his feet and legs developed significant swelling. He also complained of a generalised throbbing headache. At this stage he discontinued the trazodone and his symptoms totally resolved over the next 48 hours. He reported this condition on follow-up. In view of his cardiac condition he was not challenged again with trazodone.

This pattern of clinical presentation and resolution with cessation of the drug indicates that the symptoms were directly related and is suggestive of an allergic type response.

We wish to thank Roussel Laboratories for their kind help and co-operation with this report.

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References

FORD, H. E. & JENIKE, M. A. (1985) Erythema multiforme associated with trazodone therapy: case report. *Journal of Clinical Psychiatry*, **46**, 294–295.

MANN, S. C., WALKER, M. M., MESSENGER, G. G. & GREENSTEIN, R. A. (1984) Leukocytoclastic vasculitis secondary to trazodone treatment. *Journal of the American Academy of Dermatology*, **10**, 669.

Obsessive-Compulsive Disorder – A Complication of Benzodiazepine Withdrawal

SIR: There has been increased awareness in recent years of the symptoms of benzodiazepine withdrawal. Although obsessional symptoms have been described as part of this withdrawal syndrome (Ashton, 1984), obsessive-compulsive disorder (American Psychiatric Association, 1980) has not previously been reported.

Case Report: The patient, a 32-year-old married woman, had a history of recurrent depression treated by diazepam (6 mg daily) for the previous 7 years. She discontinued this abruptly in June 1985 on her general practitioner's advice as she was planning a pregnancy. Two weeks after discontinuing medication she developed symptoms of anxiety, insomnia, nightmares, and hyperacusis, similar to those reported by Tyrer *et al* (1983), which persisted for several weeks. Four weeks after discontinuing diazepam, however, she also developed obsessive-compulsive symptoms related to a fear that she might inadvertently reveal information to other people which would lead to the loss of her home and family. This resulted in her hoarding rubbish and avoiding going out alone. When outside her home she would stop and collect any rubbish on the road or pavement. She repeatedly checked the contents of her dustbin and also her own and other people's clothes, shoes, pockets, and money. She frequently asked her family for reassurance and help with her checking rituals. She refused to be left alone in the house for fear that she might throw "evidence" out of the window and, ultimately, required a family member to accompany her to the toilet or bath. During the night she would wake her husband and request him to go into the garden to check that she had not thrown anything out of the window. Her symptoms temporarily abated when in September 1965 she recommenced diazepam for a 2-week period, but they returned and increased in intensity on its cessation. In December 1985 she developed a severe depressive illness and was admitted to hospital in February 1986 following her general practitioner's request for a psychiatric opinion. As an in-patient she was treated with clomipramine (150 mg daily) and after 4 weeks was free of depressive symptoms. However, her obsessive-compulsive symptoms remained until April 1986 when a treatment programme of graded exposure in real life with self-imposed response-prevention (Marks *et al*, 1975) was instituted. In July she was discharged from active behavioural treatment with marked improvement in her obsessive-compulsive symptoms. She has continued to improve with homework practice and at follow-up in September 1986 was able to perform home-management tasks with little fear, although remaining anxious when walking along outside her home.

As well as suggesting a previously unreported psychiatric complication of benzodiazepine withdrawal, this case demonstrates that when depression coexists with obsessive-compulsive disorder, treatment of the depressive symptoms may not lead to resolution of the obsessional symptoms (Marks *et al*, 1980).

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References

ASHTON, H. (1984) Benzodiazepine withdrawal: an unfinished story. *British Medical Journal*, **288**, 1135–1140.

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) (DSM-III). Washington DC: American Psychiatric Association.

TYRER, P., OWEN, R. & DAWLING, S. (1983) Gradual withdrawal of diazepam after long term therapy. *The Lancet, i*, 1402–1406.

MARKS, I. M., HODGSON, R. & RACHMAN, S. (1975) Treatment of chronic obsessive-compulsive neurosis by *in vivo* exposure: a 2 year follow-up and issues in treatment. *British Journal of Psychiatry*, **127**, 349–364.

MARKS, I. M., STERN, R. S., MAWSON, D., COBB, J. & MCDONALD, R. (1980) Clomipramine and exposure for obsessive-compulsive rituals: I & II. *British Journal of Psychiatry*, **136**, 1–25.

Schizophrenia and Ethnicity

SIR: The frank suggestion by Teggin *et al* (*Journal*, November 1986, **149**, 667–668) that South African Xhosa patients are members of the Third World whilst their White compatriots live in the First World is to be welcomed. Nevertheless, it seems somewhat disingenuous to take as instances of "scientific truth" social and psychological variables which are dependent upon a system of racial classification which for many years has had no scientific support from social scientists or physical anthropologists. "Ethnicity" is a complex and polyphemous notion; to use it unproblematically, however, as if the psychiatric characteristics of a particular group can be understood in independence of a political system of classification and social control, is extraordinary.

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Relationship of Mood Alterations to Bingeing Behaviour in Bulimia

SIR: In their study of 12 patients with bulimia, Kaye *et al* (*Journal*, October 1986, **149**, 479–485) have presented an example of the use of paradox in treatment. Subjects were first told they were entering hospital for a study rather than for treatment. They listed their favourite binge foods before admission and every effort was made to obtain these. They were given a private room and encouraged to take as much time as they needed to achieve the "desired effect" of their binge and vomit, having been supplied with a tray of 5000–6000 Kcal of food for the purpose. This was followed by the attentions of a physician as soon as the binge and vomiting was completed to the subjects' satisfaction. It would be interesting to know whether such a programme had any effect on the subjects' bingeing and vomiting behaviour. Could this be a model for a novel treatment approach?

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Is There a Lithium Withdrawal Syndrome?

SIR: The paper by Mander (*Journal*, October 1986, **149**, 498–501) contains problems and an attractive enigma. The author used the words "withdrawal" and "relapse" interchangeably: they are not so. In any chronic condition, whether on or off treatment, in a stable state, making any change to that regimen (e.g. stopping lithium) is likely to effect a reaction (e.g. a relapse). The author proves this point. The pursuit of the question as to whether there is a withdrawal effect on stopping lithium that increases the number of relapses in the early stages can only be shown if there is a fall in the relapse rate lower than expected for a period after the withdrawal syndrome has ended. However, the theoretical relapse rate can never be known since the withdrawal syndrome "shadows" it. How can one distinguish a withdrawal state causing relapse and relapse alone?

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CORRIGENDA

Journal, October 1986, **149**, 471–474 (P. Thompson). In Table I (page 472) the number of patients with 4–9 seclusions in 1984 should be 7 and not 1.

Journal, November 1986, **149**, 592–602 (W. A. Arrindell & P. M. G. Emmelkamp). The second sentence of the fourth paragraph of the 'Results' section (page 598) should read as follows: "On neuroticism, the partners scored as high as partners of other patients and as high as maritally non-distressed males, and significantly lower than maritally distressed males ($P < 0.0001$), who as a group had the highest mean score (Table I)". The first sentence of the 'Sexual maladjustment and sexual dissatisfaction' section (page 598) should read "Agoraphobics were found to rate their sexual relationships with their marital partners as less adjusted than maritally non-distressed females did, but as significantly more adjusted than those of female non-phobic psychiatric subjects, and as 'adjusted' as the sexual relationships of maritally distressed female subjects".

Journal, November 1986, **149**, 631–635 (R. Noyes *et al*). The following should be added to the penultimate paragraph (page 634): "Anxious patients often report unsatisfactory interaction with physicians and alienation from them as well (Clancy & Noyes, 1976). In fact, their hypochondriacal attitudes and behaviour can seriously disrupt the doctor–patient relationship, resulting in unsatisfactory treatment (Pilowsky, 1980)."

A HUNDRED YEARS AGO

Notes and News

We desire to draw attention to the usefulness of the "After-care Ladies Working Society" in aid of the association of the after-care of poor and friendless female convalescents on leaving the asylums for the insane. The object of this Society is to assist poor female convalescents, after leaving asylums, with gifts of clothing according to the special requirements of each case. The annual subscription is five shillings. Gifts of dresses, etc, are thankfully received. Communications on the business of the Society to be addressed to Mrs Richardson, Parkwood House, Whetstone, N.; or Miss Hawkins, Chaplin's House, Colney Hatch, N. – *Eds*.

Reference

Journal of Mental Science (April, 1884) Vol. XXX.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

British Journal of Psychiatry (1987), **150**, 274–283

Book Reviews

Editor: Sidney Crown

Psychotherapeutic Intervention in Hysterical Disorders. By WILLIAM J. MUELLER and ALBERT S. ANISKIEWICZ. Northvale: Jason Aronson. 1986. Pp. 300. $30.00.

The authors are Professors of Psychiatry at Michigan State University who practise counselling psychology. Their book is devoted to the intensive psychodynamic treatment of their female clients with hysterical personality disorders.

They state simply that male hysterical personalities are the subject of extensive treatises in their own right. They aim to deal with the processes of psychotherapy in this field, for the benefit of a broad range of mental health personnel, and regard the client/patient distinction as semantic and not substantive. Their chapters work through this topic steadily and clearly.

Within the limits which they set themselves, they do an excellent job. It is not quite what their title implies, since actual conversion and dissociative disorders are totally ignored. Indeed, at the outset they identify hysteria, which is notoriously old, with hysterical personality, which so far as its description is concerned is a twentieth century concept. They also provide no systematic evidence for their ideas except for recurring vignettes of various transactions with patients which illustrate their theme. Reference is made to other work in which they have diagnostic and follow-up data on a large number of female hysterics, but this is not quoted in any detail nor is it described. Thus, the reader will be disappointed if he seeks organised factual data from which arguments can be adduced or refuted. He will have to accept or reject their statement that "... the determinants of the hysterical personality can be recast as a series of six critical developmental stages: (1) unseating the mother, (2) the mother's resignation, (3) the father-daughter pact, (4) the unmasking of the father when the daughter reaches puberty, (5) the mother's collusion in rebinding the daughter, and (6) the daughter's attempts to renegotiate a relationship with her parents".

These ideas are presented in a clear, sensitive manner which is sympathetic to the patient (client) and realistic about management, transference, and countertransference. It is not scientific psychiatry but it is instructive and gives valuable anecdotal material about what happens in interviews with many patients – perhaps not only with those who conform to the stereotype of the hysterical personality. Anyone learning psychotherapy or polishing his established skills could well benefit from some of the authors' descriptions of their dealings with their clients.

In the literature today the concept of the hysterical personality is intriguing and quite well defined. Many assumptions are held about it on the basis of psychoanalytical theories which lack solid evidence. To provide good data for or against such assumptions is a task which receives little attention, although Lazare and his colleagues made a start in work which was published as long ago as 1970. The present volume is a useful description of what is believed rather than of what can be said to be known.

HAROLD MERSKEY, *Professor of Psychiatry,*
London Psychiatric Hospital, Ontario, Canada

Bereavement: Studies of Grief in Adult Life (2nd edition). By COLIN MURRAY PARKES. London: Tavistock. 1986. Pp. 265. £12.00.

The first edition of *Bereavement*, published in 1972, was rightly acclaimed as a landmark; the second edition is no less valuable, particularly because it introduces the reader to some significant research findings which have accumulated in the intervening years. Understandably, Dr Parkes' review of the research literature is selective, but he is generous in his reference to more extended reviews.

The content and the easy literary style will make the book attractive to a wide readership, including researchers, teachers, clinicians and the bereaved. Theoretical concepts and practical principles are comfortably interwoven, and the reader is shielded from an excess of detailed research data by the fact that the latter have been presented separately in an Appendix. In the main text there are good descriptions of various features and forms of grief, including that which follows other kinds of loss, e.g. amputation. In his descriptions Dr Parkes does not stray from his most valuable source of information and insights – the bereaved – and, thus, his descriptions are punctuated with illuminating quotations and references to real people and incidents. Fortunately, however, he avoids generating the kind of mulch of sentimental anecdotes which some authors in this field provide for us.

Of particular practical relevance are the chapter devoted to helping the bereaved and the list of organisations concerned with such help.

This is a commendable book. It is to be hoped, however, that the sense of understanding which readers will derive from Dr Parkes' generalisations does not blunt their sensitivity to individual differences. The author's own comment should be borne in mind: '.... every person and every grief is different...'. (P154).

D. A. ALEXANDER, *Senior Lecturer in Mental Health, Medical School, University of Aberdeen*

AIDS: A Plague in Us. A Social Perspective. By ANTHONY A. VASS. Huntingdon: Venus Academica. 1986. Pp. 177. £7.95.

Dr Vass's book provides a stimulating account, not of the medical, but of the social, impact of AIDS for the individual and society. As a sociological analysis it is an important contribution both for the policy maker and the general reader.

The book draws on international sources but focuses on data from the UK. The first chapter documents the role of the media in the 'making of a panic' and the subsequent individual, government and policy response to the 'crisis' of the AIDS epidemic.

The second chapter reports on a small survey of laypersons' and social workers' opinions. Personal fear of contagion in the face of the unknown, the public call for information and victimising responses to members of high-risk groups are the predominant themes emerging from this work.

An account of the medical response draws attention to the immense research efforts, but not without valid criticism of the contribution of the medical profession to the premature presentation of research findings, and of frank conjecture in the presentation of 'facts' about the epidemic.

The book is as up to date as any can be on such a fast-moving subject. It provides an important analysis of society's response to a problem whose ultimate size we cannot predict. In some places the material is repetitive and its diversity occasionally makes it difficult to follow a clear thread in the development of the sociological analysis. By his own admission, the author raises as many questions as he answers. Of course, for the individual clinician there are no simple solutions for clinical management; for the Public Health Doctor there are no answers for epidemic control but there are plenty of lessons in the pitfalls to avoid.

The reader is forced to face the tightrope which we walk in making a humanitarian response to a social problem, which is ultimately everybody's, and which we seek to control. We do not yet know if this can be achieved without creating the reactionary and punitive measures of the plagues of old.

In Dr Vass's concluding words, the answer 'lies in the way in which we can manage our strained social relationships and come out of them fairly intact and safe as a society'. We would do well to heed his warning.

ANNE M. JOHNSON, *Lecturer in Genito-Urinary Medicine, Middlesex Hospital Medical School, London*

Essential Papers on Object Relations. Edited by PETER BUCKLEY. New York: New York University Press. 1986. Pp. 477. US $50.00 (hb), $22.50 (pb).

This book is one in a series entitled "Essential Papers in Psychoanalysis", the other titles in the series being on Borderline Disorders, Narcissism, and Depression. After an introduction, the book is divided into three parts. The first part is entitled 'Instincts Versus Relationships: The Emergence of Two Opposing Theories' and starts with part of Freud's three essays on the 'Theory of Sexuality' and follows with Melanie Klein's paper on 'The Psychogenesis of Manic-Depressive States' and further papers by Fairbain and Arlow.

The second part is entitled 'Object Relations and the Development of the Child' and contains classical papers by Bowlby, Mahler, and Winnicott, including his paper on 'Transitional Objects'. There is also a paper by the Sandlers 'On the Development of Object Relationships and Affects'. The final part is entitled 'Object Relations, Psychopathology and the Clinical Situation'. It contains an interesting paper by Annie Reich on 'Narcissistic Object Choice in Women', a paper by Edith Jacobson on depression, one by Modell on 'Primitive Object Relationships in Schizophrenia' and an article summarising Kernberg's views. Finally, there are papers by Loewald, mainly on his classic 'The Ailment' and Guntrip's personal experience of analysis. It would have been nice if each paper had a date with the title so it could easily be seen in its historical context.

Despite the quality of the papers, I was left questioning the purpose of the anthology. Most of the papers are familiar already to anyone involved in the psychoanalytic field, a substantial number being accessible in the journals. Also, with such an enormous topic it is not possible to cover the whole field and any collection must be, to some extent, the individual's choice. There seems to be an overlap with the other books in the series, in the sense that there are articles here on narcissism, borderline states and depression. If the intention of this book is to highlight the development in understanding of particular types of object relations, I would like to have seen included a paper on Bion's concept of the "bizarre object" in psychosis, and also more on the place of symbol formation.

Despite these reservations, the anthology might prove useful in terms of a basis for reading seminars for psychiatrists and related workers, as part of an overall psychotherapy teaching programme. It would be important that the papers are evaluated in relationship to one's clinical experience, to get the most out of them.

RICHARD LUCAS, *Consultant Psychiatrist, Claybury Hospital, Essex*

Mental Deficiency: The Changing Outlook (4th edition). Edited by ANN M. CLARKE, ALAN D. B. CLARKE and JOSEPH M. BERG. London: Methuen. 1985. Pp. 834. £40.00.

This is the 4th edition of a book that has played a major part in the education of many professionals who work with people with a mental handicap. The reading of the 4 prefaces, the first written in 1958 and all included in this book, gives the historical perspective written as services were developing which is far more powerful than any retrospective account of such a series of changes. Throughout the book there has been a determined effort to use modern terminology for the inevitable process of labelling but the book itself is still called "Mental Deficiency", which will surprise many but is debated and defended by those responsible for its retention.

The book has both a multidisciplinary and a multinational authorship with the various chapters displaying a remarkably even standard of approach and complexity. All embody a review of the literature and reflect the need to value the truly scientific approach to the increasingly complex issues surrounding the understanding and management of a person with a mental handicap.

There continues to be an emphasis on the biomedical, the biosocial and educational aspects with much of the book devoted to aetiology and prevention in its widest context. Therapists will be interested in the chapter on "Microelectronics and Computer-Based Technology", and nowhere is the gap seen more clearly between research and practice than in the chapter on "Communication".

In most of the chapters the sophistication of the knowledge base as portrayed demands a commitment and careful study by the reader and this is not a book to browse through in an odd moment. It is hard to find anything missed out from this book, but perhaps more attention will be paid to the carers' needs for support and the risk of "burn out" in a subsequent edition, on which I suspect and hope that work has begun already.

JOAN BICKNELL, *Professor of the Psychiatry of Mental Handicap, St George's Hospital Medical School, London*

Chemotherapy in Psychiatry: Principles and Practice (revised and enlarged edition). By ROSS J. BALDESSARINI. London: Harvard University Press. 1986. Pp. 354. £21.25.

Most of those commencing their training in psychiatry are on the look-out for a comprehensive, informative and readily accessible textbook concerning the use of drugs in psychiatry. Unfortunately, although this book contains much useful advice the information is not easy to extract and I would not feel that it is a book I would recommend to trainees.

The first chapter provides an historical context for modern psychopharmacology with a description of drug development in the pharmaceutical industry. The second chapter on antipsychotic agents has an overall three-part layout: pharmacology, clinical use, and toxicology. This pattern is repeated in the three following chapters which cover lithium salts and anti-manic agents, antidepressant agents, and anti-anxiety drugs respectively.

The chapter on antipsychotic agents covers the short and long-term use of such drugs and contains much useful information, but at times this proves hard to extract from the prose. The following chapter on anti-manic agents provides a comprehensive review of the advantages and disadvantages of lithium therapy but relegates the use of carbamazepine to a paragraph in the section on 'alternative and experimental treatments'. The chapter on antidepressant agents reaches a mammoth length of 104 pages, in contrast to the 34 pages on anti-anxiety drugs. Despite mention being made in both these chapters of the use of antidepressants in panic disorder, no clear guidelines are given. All these chapters mention research investigations but no methodological detail is provided, making critical assessment problematic for the reader, although a good reference list is provided. The final chapter covers 'special topics' which includes useful notes on geriatric and paediatric prescribing as well as a discussion of clinical and medical responsibility.

It is to the author's credit that despite this being an American text mention is made of drugs used in Europe but not in the USA. The absence of mention of what approach to take towards alcohol withdrawal or of the recent work on the placebo response reduces the usefulness of the text to trainees in psychiatry. Although the book has good aspects it cannot be considered a comprehensive text. It aims to be a practical guide but repetition and the inaccessibility of the information mitigate against its use in teaching and clinical practice.

ANNA HIGGITT, *Research Worker,*
Institute of Psychiatry, London

Paradoxical Strategies in Psychotherapy. A Comprehensive Overview and Guidebook. By LEON F. SELTZER. Bognor Regis: John Wiley. 1986. Pp. 323. £31.75.

If I write "Readers of this *Journal* will not be interested in this book because American psychotherapy texts are always sloppily written and unscientific" with the thought in mind that this will irritate readers sufficiently that they will want to read the book, then I have used a paradoxical strategy. Such strategies are particularly employed by family therapists who make use of systems theory. Phrases like "symptom prescription" and "relapse prediction" connote some of the techniques which have been described, particularly by workers at the Milan Centre for Family Studies since 1971.

However, in this well written and scientific book Dr Seltzer takes a broader view of the paradoxical element in psychotherapy. He draws in ideas from Freud, Adler, and others in the psychoanalytic tradition, techniques used in the behavioural therapies such as "massed practice" and "paradoxical intention", and important elements of gestalt therapy such as "staying with" unwanted thoughts and feelings. This discussion of links between techniques allied to disparate theoretical approaches highlights the value of utilising positively the patient's reluctance to change and of promoting conscious control of habitual, unwanted behaviours.

The sections on empirical research remind us how insubstantial are research results in psychotherapy generally and give the critical reader a sense of proportion in evaluating the theoretical contributions. There is a useful set of appendices which includes a selection of papers on the planning and execution of paradoxical strategies and a comprehensive bibliography.

This book should appeal to a wide variety of readers interested in psychotherapy theory, research or practice.

ERIC CROUCH, *Consultant Psychiatrist,*
St John's Hospital, Aylesbury

Health Needs of Women as they Age. Edited by SHARON GOLUB and RITA J. FREEDMAN. New York: The Haworth Press. 1985. Pp. 122. $24.95 (hb). $19.95 (pb).

I looked forward to reading this book about a topic of great importance but about which I felt ignorant. By the end of the century, the number of people aged 75 and over in this country is expected to increase by about one-fifth and the number aged 85 and over by no less than one half. Elderly women will outnumber elderly men considerably.

Most care of the elderly is undertaken in the community – much of it by women. Because of a lower birth rate, there is now a lower ratio of carers to those in need. Furthermore, many more women are now working, so are less free to look after their elderly relatives.

Unfortunately, this North American book was a great disappointment. It was parochial and rather boring. A number of topics were covered so superficially that it was not worth bothering. Some chapters did not even address issues related to elderly women.

After an introductory overview, there were chapters on social policy, long-term nursing, respite care, Alzheimer's disease, reproductive cancer, hypertension, eye problems, osteoporosis, and psychological aspects. It was not clear who the book was for and it provided little practical information that would be of use to clinicians, be they general practitioners or psychiatrists. Important omissions were health promotion, prescribing, nutrition, and arthritis. There are many better British books on the market dealing with the health needs of the elderly, and covering the special needs of women as well as men.

PETER ANDERSON, *General Practitioner and*
Senior Registrar in Community Medicine,
Community Health Offices, Oxford

Growing Points of Attachment Theory and Research. Edited by INGE BRETHERTON and EVERETT WATERS. Chicago: University of Chicago Press. 1986. Pp. 320. $14.50.

This monograph of the Society for Research in Child Development would be of prime interest to developmental psychologists, but could be useful also to child psychiatrists, particularly at the basic trainee stage. A collection of papers, mainly by experimental psychologists, it aims to review the work on attachment by Bowlby, Ainsworth, and others, and bring it up to date, drawing upon recent research material.

The book is divided into four parts of which Part 1, called "Introduction", is an overview of attachment theory in retrospect and prospect by Inge Bretherton. For those with limited reading time this would be the most useful chapter to read, as it highlights the important issues and refers forward briefly to some of the research data published later in the book. Part 2 deals with theory and current methods of assessment and there is a lot of detailed research material, including a description of a Q-sort method of assessing attachment security. The third part consists of six separate papers studying attachment in different situations, e.g. in institutionally reared infants, "high-risk" infants, or maltreated infants. The final part makes an attempt at cross-national study and includes three chapters presenting attachment research findings in North Germany, Kibbutz-reared Israeli children, and in Japan. In picking out just three countries it cannot, of course, be a comprehensive review, but it does pinpoint significant differences which could be attributed to differing attitudes to child-rearing.

Each chapter is by a different team of researchers and could be read separately. In fact, I would not recommend attempting to read the book through from cover to cover as this would inevitably seem repetitious; for example, most of the research studies make use of Ainsworth's "Strange Situation" experimental technique for assessment of attachment patterns.

PATRICIA AINSWORTH, *Lecturer in Child and Adolescent Psychiatry, University of Manchester*

Autistic Children: New Hope for a Cure. Edited by N. TINBERGEN and E. A. TINBERGEN. London: George Allen & Unwin. Pp. 362. £9.95.

The authors, distinguished ethologists, have used their skills as expert observers of animal behaviour to study autistic children. On the basis of their observations they conclude, in disagreement with the prevailing expert opinion, that the autistic condition is in essence an emotional or motivational disturbance, a state of anxiety-dominated inbalance which impairs the child's ability to interact with the environment. The various perceptual, cognitive, motor, and speech deficits are regarded as peripheral and secondary to the central emotional impairment. The basic problem would be brought about by early traumatic experiences in infancy, i.e. a difficult delivery, separations from parents, moving houses, the birth of a sib. The authors believe that many mothers manage to coax their autistic child back to normality, if top priority is given to the reduction of the child's abnormal feelings of insecurity and of the mother's inability to cope emotionally with her child. They particularly favour the technique of holding the child for prolonged periods in order to reduce distress and to facilitate the development of normal social responses.

Unfortunately, though their case may appear compelling, it is by no means proven. There is no attempt by the authors at testing their views with a controlled design, and insufficient consideration given to opposing evidence available from other sources. While some of the cases histories given in the book emphasise the fact that some children with marked autistic features can improve substantially, and the ability of some parents to respond extremely competently to them, this cannot be taken as conclusive evidence to support the authors' theories or their general optimism about outcome. Regrettably, this optimism could give false hopes to distressed parents. This is a disappointing book.

ELENA GARRALDA, *Senior Lecturer, Booth Hall Children's Hospital, Manchester*

Rehabilitation in Psychiatry: An Introductory Handbook. By CLEPHANE HUME and IAN PULLEN. Edinburgh: Churchill Livingstone. 1986. Pp. 230. £8.95.

This introductory handbook succeeds in its aim to acquaint the novice of whatever professional discipline with the scope of psychiatric rehabilitation, by providing readable basic information. It includes brief critical reviews of the history of institutions and of some of the notable literature on institutionalism and on community treatment. The sections on the nature of disability and the principles underlying rehabilitation are perhaps too short, but the right emphasis is placed on the practical aspects of assessment, planning, and service organisation, as well as on the importance of long-term (often life-long) commitment to chronic patients in the community. Working with families both as units and in groups is given due prominence. The book is particularly good on the organisational aspects of a rehabilitation service, including a commendable chapter on the dilemmas inherent in multidisciplinary teamwork. There are also chapters describing the rehabilitation problems of mental handicap, old age, and physical disability.

There is a certain amount of duplication; in the chapter on psychological approaches, for example, three pages are devoted to assessment which is also discussed elsewhere. The focus of the book could have been considerably sharpened by the omission of sections on the aetiology, clinical picture, and diagnosis of schizophrenia.

"Common sense" is a recurring phrase in this book and it characterises much of its message. The book is likely to whet the student's appetite for further reading.

MOUNIR EKDAWI, *Consultant Psychiatrist,*
Netherne Hospital, Surrey

The Adolescent Unit Work and Teamwork in Adolescent Psychiatry. Edited by DEREK STEINBERG. Chichester: John Wiley & Sons. 1986. Pp. 229. £12.50.

Adolescent units are complex places where role diffusion, dependent on the personalities and proclivities of the staff, obscures the underlying structure and purpose. This puzzles the observer who has to understand the implications in order to adopt ideas for use in his own work. Yet some form of cross-fertilisation is essential if adolescent units are to be the cornerstone in the growing network of services the Hospital Advisory Service describe in 'Bridges over Troubled Waters'. In the absence of a steady influx of trained staff, it is too easy for a unit to become inbred and isolated. An alternative is to turn to written accounts. Unfortunately, the usual text presents too simple and one-sided a perspective to be useful to the reader who wishes to introduce its ideas to a unit. However, this book notably differs; each chapter is written by one of 21 staff, most of them at the Bethlem Hospital, to give not just a clear and vivid account of their work with some theoretical background, but also how it dovetails with that of others. That this is a very coherent book is an editorial achievement which makes it possible to understand how all these people might knit together to form a team.

Facets of unit management are well covered – the support, supervision, and training of nursing staff; staff groups; medical training and research; as well as the central part played by the unit secretary.

My initial feeling was one of envy for the staffing and resources available to this unit. However, I found this book such a fertile source of ideas that not only did I order another copy for circulation, but it was brought home to me how essential it is to have a centre such as this to serve as a teaching base. Any retrenchment will be a national loss.

I am impressed by the evenly high standard throughout: no chapter was a disappointment. It is easily read and I would recommend it to anyone of whatever discipline who is working in, or touring, an adolescent unit.

T. P. BERNEY, *Consultant Psychiatrist,*
Prudhoe Hospital, Northumberland

The Psychodynamics of Medical Practice. By HOWARD F. STEIN. Los Angeles: University of California Press. 1985. Pp. 190. £14.50.

Stein is a psychoanalyst and medical anthropologist whose book is about the countertransference in medical work. In educational sessions with family doctors and medical students he found its role underestimated. (He defines it widely: "I use the term countertransference to mean all non-rational elements of the physician's work"). So one chapter is "an ethnographic odyssey in which I describe how I discovered the importance of countertransference in the daily work of the family physician and the significance of the unconscious in the dynamics of the physician's role". This is characteristic of the whole book, which shows the author impressed by his own discoveries and overestimating their originality. Thus his exposition of psychoanalytic theory and descriptions of the psychodynamics of the doctor-patient relationship in complex cases in family or specialist medical practice were preceded by Balint in *The Doctor, His Patient and the Illness* over thirty years ago. Stein tends to be repetitive and stodgy in style, and nearly always he is speaking in his own voice. Balint's book was crisply written, memorable, and did its author's job very modestly by using largely the general practitioners' contributions to develop the theme. Today's students will find this book heavy going. Balint is still in print – if money is short, buy a fresh copy for the library to attract their attention.

A. C. SMITH, *Consultant Psychiatrist,*
Greenwich District Hospital, London

Body Image, Self-esteem, and Sexuality in Cancer Patients (second revised edition).Edited by JEROME M. VAETH. Basel, Switzerland: Karger. 1986. Pp. 153. SFr. 79, DM 95, US$33.75, £24.70.

The impact of cancer and cancer treatments on the sexual lives of patients is the predominant theme of this book. Based on a symposium held in San Francisco, the book contains useful information about specific problems facing patients with gynaecological cancers, breast cancer, head and neck cancers, and cancers of the bowel, prostate, testis, and penis. A welcome feature is the participation of patients in the symposium. Their experiences, frankly and often movingly told, provide a sharp reminder to clinicians of deficiencies in current management of cancer patients. Attempts to remedy some of these deficiencies by methods ranging from psychological intervention to surgical procedures such as breast reconstruction and implantation of penile prostheses are outlined. One controlled study is described, in which significant short-term benefits are reported for women with gynaecological cancers treated by crisis intervention counselling.

Certain criticisms are warranted. The omission of an Index is a disadvantage. Occasionally, unrestrained enthusiasm appears to overwhelm clinical common sense. For example, one author recommends that women with pelvic cancer admitted to hospital for major gynaecological surgery should be questioned regarding the frequency of oral, anogenital, and masturbatory activities. The mind boggles. More serious is the use of the label "ostomates" to designate patients with stomas. To stigmatise patients in this way is likely to cause distress by increasing their sense of social isolation. But notwithstanding these criticisms, the book can be recommended for the pertinent, painfully honest comments by patients as much as for the contributions by physicians, psychiatrists, and other professionals.

STEVEN GREER, *Reader in Psychological Medicine, King's College School of Medicine and Dentistry, London*

Biologic Response Styles: Clinical Implications. Edited by HOWARD KLAR and LARRY J. SIEVER. Washington: American Psychiatric Press. 1985. Pp. 95. $12.00.

This volume is said to have been written with a view to filling 'a critical educational gap' relating to a failure to integrate psychobiological advances with the clinical thinking of trainees in psychiatry. Although the editors express satisfaction with their trainees' responses to the approaches described in this small volume I do not think it would be warmly received by the average trainee in this country. Indeed, I should be sorry if it were. In spite of its brevity the content of the book is wide-ranging, covering Harlow's work on monkeys and other later experiments in the same field, the study of neonatal behaviour, and the relationships of many biological variables to a variety of patterns of behaviour. The tone of the chapters is uniformly optimistic and there is no attempt in any of them to provide a balanced assessment of the literature – indeed, one of the authors concludes his chapter with the disarming and accurate statement, "The story is far from complete and I have not addressed some major disagreements among researchers in this area". The book is of some interest as a series of speculative essays, and some readers may find them a striking example of transatlantic English. I could not however recommend this volume for educational purposes for trainees in psychiatry.

EVE C. JOHNSTONE, *Honorary Consultant Psychiatrist, Northwick Park Hospital & Clinical Research Centre, Middlesex*

To Provide Safe Passage: The Humanistic Aspects of Medicine. Edited by DAVID RABIN and PAULINE L. RABIN. New York: Philosophical Library. 1985. Pp. 258. $19.95.

David Rabin was a professor of medicine and of obstetrics and gynaecology who died of amyotrophic lateral sclerosis at the age of 49. In partnership with his wife, who is a psychiatrist, he has written movingly about how he was rejected by his colleagues once they discovered he was incurably ill. This devastating and bewildering experience prompted him and his wife to explore the 'caring' component of medicine as opposed to the 'curing' component: in other words 'to provide safe passage' through illness.

Their 6 chapters are supplemented here by 14 by others, dealing with different aspects of caring, including chemotherapy for cancer, doctors and cancer, hospices, management of intractable pain, attitudes towards mental illness and mental retardation, genetic counselling, death, and dying.

Many of the contributors have been allowed to be wordy, relying on generalisations rather than precise observations. But there are exceptions, such as the chapter on genetic counselling; and of course the Rabins' chapter, Compounding the Ordeal of ALS: Isolation from my Fellow Physicians, which is stark and direct and should be read as a warning by all doctors. Overall the book is a useful introduction to these important topics, and (in paperback) could be recommended to medical students.

GLIN BENNETT, *Consultant Senior Lecturer in Mental Health, University of Bristol*

Human Development (3rd edition). By ERIC RAYNER. Hemel Hempstead: George Allen & Unwin. 1986. Pp. 300. £8.95 (pb), £22.50 (hb).

This is the third edition of a text well known to those who have had to study psychology as a part of their professional training – to those, that is, who use psychology informally, as part of their work.

For this purpose the book seems well suited. It is clear, easy to read and interesting – it is not abstract, but illustrates most of its points from descriptions of real life that most people can recognise.

The book is psychodynamic, based on the modern psychoanalytic viewpoint, although it does cite other references. This raises a slight doubt in the reviewer's mind: if this is the only book, say, a social worker reads, is it sensible that it be restricted to essentially one viewpoint, however revealing this is? The practicality of the book is emphasised by the examples; these include some modern ones (single parents, divorced parents, and sexual partnerships outside marriage) as well as the old-fashioned domestic issues of rearing children in loving families.

I can recommend this book as one which is humane and clearly written and which gives readers a good psychodynamic overview of human development.

PAUL KLINE, *Professor of Psychometrics, University of Exeter, Devon*

Behaviour Therapy in Primary Care. A Practical Guide. By RICHARD FRANCE and MEREDITH ROBSON. London: Croom Helm. 1986. Pp. 235. £10.95.

The aim is to provide a practical manual for primary care professionals with limited time at their disposal, but who wish to use behavioural methods with their patients. The interventions described are brief, and the emphases are on methods that can be used outwith consultation times and on patients' developing self-help skills. The first section deals with behavioural assessment, data collection and monitoring, and the basic concepts of intervention. Part two deals with the management of clinical problems: anxiety, interpersonal problems, habit disorders, the cognitive-behavioural management of depression, behavioural aspects of general medicine, and difficulties in study, work, retirement, childhood, and old age. Useful articles and books are listed.

The aim is fulfilled. Is it significant that the index contains no reference to illness, mental disorder, psychiatrist, or psychiatry?

GREG WILKINSON, *Honorary Lecturer,*
General Practice Research Unit,
Institute of Psychiatry, London

The Man Who Mistook His Wife for a Hat. By OLIVER SACKS. London: Duckworth. 1985. Pp. 233. £9.95.

Dr Sacks presents a series of case reports of a variety of neuropsychological disorders. These are rich in detail and full of the elegant prose that one has become to expect from the author. The book emphasises the limitations of the statistical approach often applied in the neuropsychological literature. More importantly, Dr Sacks takes neuropsychology out from behind the confines of statements of the location of the lesion and associated cognitive deficits to the impact on the patient's experience of the disorder. The book is essentially within the realm of the phenomenology of illness, full of Dr Sacks' provocative interpretations and insights as he peers into the worlds of people with neurological damage. The book covers case reports of prosopagnosia (whence the book's title is derived), visual agnosia, amnesia, neurosyphilis, and autism, amongst others.

This is, however, not a book for those who wish to add to their 'knowledge' of neuropsychology as much as it is for the reader with some interest or curiosity in the range of disorders following neurological damage. The specialist reader is likely to see that the book reflects Dr Sacks' own discoveries of the literature and it is this that may blunt their confidence in the author. For example, it is surprising to read that the author discovered the literature on visual agnosias & prospagnosia only after he completed the book! These concerns should not detract from the book's major contribution, which is to broaden our appreciation of the experience of these unusual and often bizarre disorders.

S. NEWMAN, *Senior Lecturer in Psychology,*
Middlesex Hospital Medical School, London

The Dilemma of Abortion. By EDWIN KENYON. London: Faber & Faber. 1986. Pp. 283. £6.50.

This rewarding Faber paperback by an Oxford academic and clinical psychiatrist integrates two fields of interest, one universal and one topical, by reviewing philosophical and religious attitudes to the human foetus and its rights throughout recorded history and linking them to a detailed, impressively up-to-date account of legal, medical, political and economic issues affecting pregnancy termination today.

In a presentation aimed at general readers as well as health specialists the perspective is broad, but the prose is precise, factual, lucid and elegant. There are manageably succinct references, mostly to prestigious sources. Dr Kenyon writes with a restrained passion, and the humanity and scholarship of his approach are evident from the book's first page, which carries a touching eighteenth-century poem to an aborted child:

> ..."Love, spite of honour's dictates, gave thee breath;
> Honour, in spite of love, pronounced thy death."

Considerations other than honour are nowadays liable (and in many settings, including Britain, likelier) to decree the death of an embryo or foetus: convenience, change of mind, the antenatal detection of disease, the suspicion of genetic defect, pressure from parents, pressure from grandparents, state intervention (as in China), being not of the preferred sex (as, we are warned, among certain cultural groups nearer home), poor housing, poverty, maternal overburdening, maternal age, and psychiatric illness. The author discusses them all, many with examples from his clinical experience. He describes the contributions made, and solutions offered, by embryologists, diagnosticians, family planning experts, lawyers, and psychiatrists. He summarises the attitudes towards contraception and abortion of the major world religions, theocentric or secular, and anybody who has ever had to establish rapport with a client of different background in these sensitive areas will be grateful for his guidance.

The book is pragmatic, objective, and tremendously informative. At £6.50, it deserves a wide sale.

MARGARET J. LITTLE, *Associate Specialist,*
Stobhill General Hospital, Glasgow

Divorce Therapy. Edited by DOUGLAS H. SPRENKLE. New York: The Haworth Press. 1986. Pp. 184. $29.95.

The book does not, as the title suggests, recommend divorce as a therapeutic procedure; rather, it acknowledges that, since divorce abounds, the therapist needs to be equal to the task of successfully steering people through it. Also published as Vol. 1, Nos. 3/4 of the Journal of Psychotherapy and the Family, the book comprises twelve concise sections, the last being an excellent annotated bibliography.

Divorce, it is proposed, like grief, with which it has a great deal in common, is divisible into three stages: the pre-divorce decision-making stage, the divorce restructuring stage, and the post-divorce recovery stage. I was left with the distinct impression that one of the principal objectives of therapy is to keep each individual moving through this sequence. I was impressed by the deceptively simple aphorism of Marcia Brown that once the reality that life will never be the same again hits home the client is faced with another reality – that what is not the same will inevitably have to be different.

Those who seek to understand relationships need also to concern themselves with relationships in transition. In systemic terms divorce involves the decoupling and recoupling of a variable number of family members, and not all members are decoupling or recoupling to the same extent or at the same rate. While focusing on the specific area of divorce, the book in fact is adding richly to a more general knowledge of family dynamics. I would therefore recommend that it be read not just by a specialist minority.

JOHN BIRTCHNELL, *Scientific Officer and Senior Lecturer, MRC Social Psychiatry Unit, London*

New Clinical Concepts in Marital Therapy. Edited by OLIVER J. W. BJORKSTEN. Washington: American Psychiatric Press. 1985. Pp. 165. $12.00.

This monograph emerged from a symposium held at the annual meeting of the American Psychiatric Association in May 1984 and represents the views of seven contributors. Although the last two chapters deal with the treatment of marital dysfunction, the book is not intended as a textbook of marital therapy. The aim is to inform readers about areas of marriage, as it is observed in the United States, which are currently considered to be important and of relevance to psychiatrists.

Five areas are covered. First there is an overview of sociodemographic aspects of contemporary American marriage (T. J. Stewart, O. J. W. Bjorksten and I. D. Glick) which provides a critical assessment of marital trends, pointing out how erroneous conclusions are often drawn from simplistic handling of available data. The next two chapters provide a competent scan of the relationships found in America, between marital status and psychiatric morbidity (R. T. Segraves) and between marital status and health (O. J. W. Bjorksten and T. J. Stewart). There follows a discussion (C. J. Sager) of the psychiatric implications of the phenomenon of the reconstituted family which have been brought into prominence by the current high remarriage rate, accompanied by a description of one form of intervention. Finally, there is a chapter (D. C. Polonsky and C. C. Nadelson) on an approach to couples therapy in which the authors illustrate how therapists can integrate different therapeutic models to deal effectively with changing clinical realities. The major theoretical positions dealing with the therapy of couples are not reviewed, the assumption being made that readers will be familiar with them.

The explanatory hypotheses used to make sense of the wide range of information cited express the accumulated experience of the schools of thought represented by the authors. Although not all psychiatrists may agree with these views, the monograph gives a succinct and refreshing overview of data of value both to clinicians and to research workers.

DOROTHY H. HEARD, *Lecturer, St James's University Hospital, Leeds*

Evoked Potentials: Frontiers of Clinical Neuroscience Volume 3. Edited by ROGER Q. CRACCO and IVAN BODIS-WOLLNER. New York: Alan R. Liss. Pp. 551. £70.00.

Evoked response techniques have provided an added impetus to the investigation of many types of patient including those with psychiatric disorder. This multi-authorship book with a firm editorship provides a comprehensive review with sections on basic aspects, methodology, animal models, and anatomy, as well as biochemistry and pharmacology. On the clinical side neurological, neurosurgical, and paediatric aspects are presented at some length. It is a pity therefore that psychiatry does not have a broad coverage and odd that the 'expectancy wave' (now called the CNV), a feature first noted to relate to 'mental state', is not even mentioned, although the P300 component is discussed, while learning disabilities and related matters as well as dementia are assigned chapters. This is therefore not a book for the general psychiatrist, but for those who have a special interest in clinical neurophysiology. For them there is much to be gained from this well-produced and otherwise comprehensive volume.

D. F. SCOTT, *Consultant in Neurophysiology, The London Hospital*

Research in Schizophrenic Disorders: The Stanley R. Dean Award Lectures Volume I.By ROBERT CANCRO and STANLEY R. DEAN. Lancaster: MTP Press. 1985. Pp. 287. £42.50.

This is the first of two volumes reprinting the lectures given annually from 1962 to 1982 by holders of the Dean Award. The award was intended to acknowledge major research contributions to schizophrenia. It is said that very little of importance has been done on schizophrenia in the last twenty years that has not involved an awardee; surprisingly, only one of the awardees is British.

Volume I relates to diagnosis and to social and familial factors in aetiology.

The papers that still retain most interest are those by Kanner on infantile autism, Manfred Bleuler on the natural history and on the children of schizophrenics, Langfeldt on diagnosis and prognosis, and Mednick and Schulsinger on studies of children at high risk.

Some of the authors have taken the opportunity of republication to comment on their papers. Bleuler summarises one of his main findings: on average, psychosis shows no change for the worse after a duration of five years. He believes that nowhere is sufficient assistance given to the children of schizophrenics.

Langfeldt's comments include the surprising statement that as late as 1980 he was led to realise that the terms depersonalisation and derealisation are not used in Anglo-American psychiatry in the same way as he used them, which was to describe certain delusions of passivity.

The book is of some historical interest and also of value because it makes the writings of the non Anglo-Americans accessible.

J. COOKSON, *Consultant Psychiatrist, The London Hospital (St Clement's)*

Infant Psychiatry: An Introductory Textbook. By CLAUS MINDE and REGINA MINDE. London: Sage Publications. 1986. Pp. 197. £19.75 (hb), £10.00 (pb).

This is a book dealing with the child under 3 from a psychological and psychiatric viewpoint. The historical review is most engrossing and the summary of theories of child development outstanding in its comprehensive yet brief account. It is broad-based and eclectic, drawing on ideas and research from many fields.

I read this book with complete satisfaction until page 99. From then on, I found myself in difficulties with their classification. Particularly disappointing was the sudden failure, after such circumspection and balance in the earlier chapters, to look critically at their diagnostic categories. The Reactive Attachment Disorders of Infancy are, of course, part of DSM-III but the authors suggest making this an even less well defined category than it is currently. Then there is a wholly unsatisfactory group of "Disorders in Behavioural Organisation"; no evidence is offered as to the validity of such a grouping. To most of us, this grouping refers to children who are very difficult temperamentally.

However, leaving aside the unsatisfactory classification, all the chapters on assessment and treatment, including those just mentioned which do have clinical appeal, make interesting reading and would add to most people's understanding and ways of intervening in problems in this age group. In particular, it adds to one's confidence in assessing young babies and early parenting difficulties.

I would recommend this book to anyone who wants to focus their thoughts on the specific problems of babies and the young child. Read with the reservations mentioned in mind, it makes a very good introduction.

CLAIRE STURGE, *Consultant Psychiatrist,*
Northwick Park Hospital, Middlesex

Institutions Observed: Towards a New Concept of Secure Provision in Mental Health. By LARRY GOSTIN. Oxford: Oxford University Press. 1986. Pp. 179. £6.50.

Larry Gostin's latest offering is something of a broadside, in which he reviews the inadequacies of the various institutions that cater for offender and/or disturbed mentally disordered patients. Prisons, the new regional secure units and local mental hospitals are all criticised, but he reserves his biggest salvoes for the special hospitals. Gostin says (and who would disagree?) that there is no effective, national organisation of services for mentally abnormal offenders; patients end up in the wrong place – in particular in the wrong institution. A succession of reports has not helped, and there is a preoccupation with secure accommodation.

Gostin argues for a service that provides "fluid security"; a flexible system of care, locally available, that will meet the changing clinical needs of the patient during the course of his illness. Locating patients according to assessments of dangerousness, he says, are bound to fail; dangerousness itself is a fluid, not a fixed, quality. Each chapter concludes with recommendations of which many are sensible, some are puzzling and a few are naive. Brief commentaries are provided at the end by Viscount Colville, who homes in on the particular, and by Dr Tim Harding, who confines himself to general principles. Few of his criticisms are new but Gostin puts his case together with customary skill. This is a book for those who are more interested in planning services than in designing institutions. The King Edward's Hospital Fund is to be congratulated for supporting a project and publication on this important topic.

DEREK CHISWICK, *Senior Lecturer in Forensic*
Psychiatry, University of Edinburgh

Psychotherapy in a New Key: A Guide to Time-Limited Dynamic Psychotherapy. By HANS H. STRUPP and JEFFREY L. BINDER. London: Harper & Row. 1985. Pp. 333. £22.50

In line with the need to specify what psychotherapists actually do, this practical guide does exactly that for a form of time-limited dynamic psychotherapy (TLDP) developed over 15 years by the Vanderbilt team. The book is clinical and practical, the backing research having been reported elsewhere. Brevity, focus, the attempt to understand and to rectify destructive past relationships through a good therapeutic relationship, and the techniques used to achieve this are set out in detail with clinical examples.

There are now a number of clinically soundly-based dynamic psychotherapy systems: Malan, Mann, Davanloo, Sifneos, Luborsky. Whether TLDP has staying power is impossible to know at this point. However, this well-written book is an important contribution, useful both to psychotherapy trainers and to trainees.

SIDNEY CROWN, *Consultant Psychiatrist,*
The London Hospital

Psychological Aspects of Genetic Counselling. Edited by ALAN E. H. EMERY and IAN M. PULLEN. Orlando: Academic Press. 1984. Pp. 236. $14.00/£9.00 (pb), $32.00/£20.00 (hb).

This is a well written and edited book that is specifically aimed at helping genetic counsellors improve their counselling skills. It will also be of interest to other professionals who work with individuals who need counselling. There is not very much information in the book about the genetics of specific diseases, and readers who are seeking this information will need to find it elsewhere.

The book adopts the position that individuals who want counselling will display very different reactions even when faced with the same genetic dilemma. Therefore an awareness of the range of psychological mechanisms that can develop from stress such as depression, guilt, and denial is very important. The authors of the various chapters do however display different approaches to this issue. Falek adopts a relatively hard-nosed attitude, seeking to ensure that counselling is efficacious. The other chapters are concerned with marital pathology, early infant loss, Down's syndrome, mental handicap, physical handicap, Huntington's disease, infertility, sterilisation, artificial insemination, and prenatal diagnosis. All these chapters are thoughtful and humane. Some chapters are delightfully enthusiastic, for example the one on Huntington's disease.

Many psychiatrists might be interested in reading this book because even if they do not provide counselling, it shows how geneticists and others have learnt to describe psychological mechanisms in a practical, non-dogmatic manner.

HUGH GURLING, *Wellcome Senior Fellow in Clinical*
Science, Institute of Psychiatry, London

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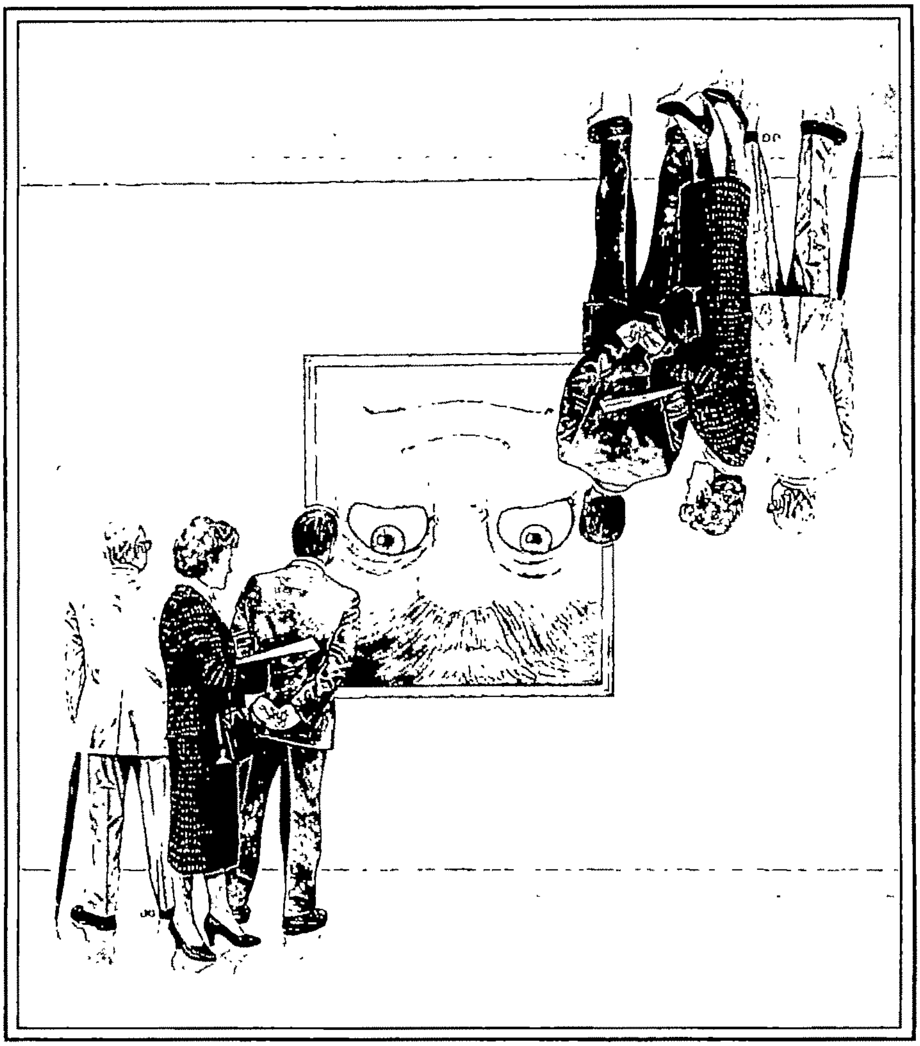

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Review Article

The Burden of Care:
The Impact of Functional Psychiatric Illness on the Patient's Family

G. FADDEN, P. BEBBINGTON and L. KUIPERS

This paper reviews the literature on the effect of severe mental illness on other members of the patient's family. The burdens of caring for a patient at home are considerable. They often affect the caring relative's social and leisure activities, and financial problems arise frequently. Relatives have difficulties in understanding and coming to terms with illness-related behaviour. 'Negative' symptoms are often a particular problem. Despite their burden, relatives do not complain much, although they receive little support, advice or information from the professionals engaged in treating the patient; much is now known about the difficulties relatives face, but we still need to know how they can best be helped. Failure to do this will have bad effects on both relatives and patients.

Most studies of the families of psychiatric patients have been aetiological. Family processes have typically been examined because of an interest in the effect of the family on the patient's illness. The view that relatives may have a deleterious influence on the course of illness is reflected in the behaviour of many mental health professionals in the clinic, who may give an impression of blaming them or of regarding them only as exploitable sources of information.

Comparatively few workers have looked at the impact of a psychiatrically ill member on the rest of the family, or at how they cope with their situation, although the majority of patients now return to live in the community. Indeed, even after many years of illness, as many as 50% of patients remain in contact with relatives (Creer *et al*, 1982). In our view, the existing studies of the effects of mental illness on the family have major implications in the fields both of psychiatric management and of social policy.

The concept of burden

This concept shares characteristics with that of social performance, for one person's poor social performance is another person's burden. Both concepts are relative to social expectations which are likely to be very variable. Thus, as Platt (1981) has emphasised with regard to social performance, measurement can never be entirely satisfactory. Measures of burden likewise remain open to criticism (Platt, 1985).

The existence of a burden indicates the breakdown of the reciprocal arrangements that people maintain in their relationships, such that one person is doing 'more than their fair share'. This may merely result in them taking on greater proportion or number of shared tasks, but it may also restrict their activities outside the relationship. This change in pattern can be assessed against approximate norms. In addition, it is often accompanied by subjective dissatisfaction. At a given level of objective burden, individual levels of distress show considerable variation (Platt, 1985). This further aspect of burden should not be ignored.

The dissection of the concept of burden through the effects on the performance of various roles carried out by the patient's relatives was an approach first used by Mills (1962). Grad & Sainsbury (1963*a*, *b*) advanced the measurement of burden by using a 3-point scale rather than the descriptive sketches given by their predecessors. Hoenig & Hamilton (1966, 1969) made the important distinction between 'objective' burden (e.g. effects on health, financial loss) and 'subjective' burden (the extent to which relatives felt they carried a burden). These techniques were applied by Creer & Wing (1974) in their study of the relatives of patients with schizophrenia. The methods used in the study of burden have now reached a reasonable level of sophistication despite the qualifications concerning measurement (Platt, 1985). A structured account of the behaviour of patients likely to give rise to problems is related to an equally structured assessment of the effects upon relatives and of the ways they have of coping.

Early studies on families of psychiatric patients

The first studies of the demands on the families of those with mental illness were carried out by a group of social scientists in the United States in the mid-1950s (Clausen & Yarrow, 1955). They operated within a theoretical framework developed from studies on deviance, social control and social perception (Schachter, 1951; Bruner & Tagiuri, 1954; Yarrow *et al*, 1955). The impetus for their research was clear:

> Almost no systematically collected data exist to permit an analysis of what happens within the family group when a member develops a mental illness (Clausen *et al*, 1955).

They studied 33 families where the husband was a patient with a diagnosis of psychosis or psychoneurosis. The investigators followed the wife's reaction from the time of her husband's first admission to 6 months after his return home, or to the end of his first year in hospital. This first study highlights a number of important themes which recur in subsequent research and which we will substantiate below.

The extent of burden

Because studies use different criteria for the severity of burden, only the most impressionistic sketch can be given of its extent among the relatives of patients with persistent disorders.

Mandelbrote & Folkard (1961*a*,*b*), in a study of schizophrenic patients, reported the degree to which families were restricted or disturbed by the patient's presence in the home, though it is not clear how they rated these factors. Fifty-five per cent of families were rated as disturbed in some way, though only 2% of relatives reported severe stress. The authors are vague about what they regard as stress on the family, describing typical stress situations as 'those in which the behaviour of the patient upset his family or prevented them from doing certain things'. Another shortcoming of the study is that at follow-up they did not interview the families of those patients (18% of sample) whom it had not been possible to maintain in the community, and who presumably had the most severe effects on their families. The authors found that there were more disturbed families in rural than in urban areas.

In Mills' (1962) study of unselected psychiatric patients, practically all were a source of anxiety to their relatives. More than 50% were described as 'difficult' at home and only 12% caused no practical difficulties. In Grad & Sainsbury's study (1963*a*,*b*) comparing a community-orientated and a traditional service, almost two-thirds of the families had been experiencing hardship because the patient was living at home, and in one-fifth the burden was severe.

Wing and his colleagues (1964) followed the course of 113 schizophrenic patients for a period of 1 year after discharge. Where patients returned to live with their families, social relations were strained in 59% of cases, often to the limit of what would ordinarily be regarded as tolerable.

Waters & Northover (1965), in a 2 to 4-year follow-up of 42 schizophrenic men, similarly reported that many of the patients caused moderate to severe hardship to their relatives in terms of social embarrassment, inconvenience, and behaviour which frightened them or gave rise to tension in the family. Hoenig & Hamilton (1966, 1969) examined both 'objective' and 'subjective' burden. In their study, 76% of patients had some kind of adverse effect on the household.

The impression from these studies is therefore that burden exists and is extensive. This is reflected in the well established finding of high rates of divorce and separation in marriages where one patient is mentally ill. For example, in many cases observed by Brown and his colleagues (1966), the patient's illness had been instrumental in bringing about divorce or separation. The divorce and separation rates quoted in the study were three times the national average for female patients and four times the national average for males. However, in some ways, it is surprising that more marriages do not break up. In the study by Yarrow and his colleagues (1955*a*) several wives had contemplated separation or divorce, but all had decided to give the relationship another try. A similar adherence to the marriage, in the face of considerable difficulties and apparently meagre rewards, was noted by Fadden and her colleagues (1987).

Effects on social relationships

One of the most damaging consequences of living with a relative with a persistent mental illness is the detriment to social and leisure activities. This was noted in the first study of the problem (Yarrow *et al*, 1955*b*): wives consistently believed that mental illness was regarded by others as a stigma and expressed fears regarding social discrimination. In consequence, one-third adopted a pattern of 'aggressive concealment', making drastic changes in order to avoid or cut off former friends, with some going so far as to move to a different part of town. Another third had told only members of the family, or close friends who either understood the problem or had been in a similar situation themselves (Yarrow *et al*, 1955*b*).

A number of other studies have documented the restriction of social activity experienced by those who live with and care for patients with schizophrenia (e.g. Mandelbrote & Folkard, 1961*a*,*b*; Wing *et al*, 1964; Waters & Northover, 1965) and this may be especially marked when the relative is an elderly parent (Leff *et al*, 1982). However, such findings have also been reported for spouses of a group of patients with persistent depressive disorders (Fadden *et al*, 1987). These relatives spent over 60 hours a week in face-to-face contact with the patient and were correspondingly socially isolated. Grad & Sainsbury (1963*a*,*b*) add that the restrictions are not limited to those living with schizophrenic patients. It is not at all clear that the phenomenon of stigma contributes less to this social isolation than it did in 1955.

Financial difficulties

These have been emphasised in a number of studies (Yarrow *et al*, 1955*b*; Mandelbrote & Folkard, 1961*a*,*b*; Mills, 1962; Hoenig & Hamilton, 1966, 1969; Stevens, 1972). To some extent, difficulties may arise because caring for a patient with a persistent psychiatric disorder limits opportunities for an adequate income. The most severe problems, however, occur when the patient was formerly the breadwinner, particularly if circumstances prevent the relative from taking over this role. The extent to which the families of psychiatric patients are impoverished should not be underestimated.

Relatives' attitudes to the illness

Although mental health professionals, by their calling, readily accept that behaviour can be symptomatic of illness, this requires a major shift in attitude for relatives. Clausen and his colleagues (1955) pointed to the difficulties wives experienced in attributing meaning to their husbands' actions. Wives' attitudes about the normality of such unfamiliar behaviour continually altered, probably because of the overlap between the symptoms of mental illness and normal patterns of behaviour. Early interpretations tend to focus on physical difficulties, or character problems such as their husband being weak or lacking in willpower (Yarrow *et al*, 1955*a*). The authors note the psychological impact on the wife of having to consider her own possible role in the development of her husband's disorder, and of contemplating her future as the wife of a 'mental' patient. It is also clear from the study that the wives experienced anxiety, guilt and feelings of rejection towards their husbands as a consequence of the illness.

Most of the relatives described by Creer & Wing (1974) had at various times experienced anger at the way their lives had been spoiled and grief when they recalled what the patient had been like before the onset of illness. In the study of Fadden and her colleagues (1987), many of the spouses of depressed patients expressed a sense of loss, as if they had been physically bereft of the person they had married. Anger and guilt were also prominent.

It is apparent from these accounts and others scattered throughout this literature, that the intrusion of mental illness into the family is a trauma which causes relatives considerable difficulties and with which they are given little help. Moreover, things may not become easier as time passes. Hoenig & Hamilton (1966, 1969) found that the likelihood of some objective burden increased the longer the patient remained ill. Mandelbrote & Folkard (1961*b*) similarly reported that progressively more families were rated as disturbed as time passed during their 4-year follow-up period.

Burdensome symptoms

There is a fairly clear-cut consensus from the available studies regarding the symptoms that relatives find most difficult. A common concern of the relatives in Mills' (1962) study was that patients might be a danger to themselves or others, and problems frequently arose with neighbours as a result of the patients' behaviour. Many relatives complained of disturbed nights, and reported that practical problems caused less difficulties than the patient's 'strange fancies' or 'dumb apathy'. Those patients who did not speak often created more distress than those whose speech was excessive, though the latter caused suffering too. In Grad & Sainsbury's (1963*a*,*b*) study, patients with psychosis presented more of a problem than those with neurotic disorders. The symptoms found to be associated with a rating of severe burden were aggression, delusions, hallucinations, confusion and an incapacity for self-care. However, the problems families complained of most often were not the clear-cut symptoms, such as violent or socially embarrassing behaviour, that are usually associated in the public mind with mental illness, but rather the frustrating, depressive and hypochondriacal preoccupations which the patients exhibited. Brown and his colleagues (1966) found the number of problems and the distress experienced by the relatives of schizophrenic patients were highly related to the degree of disturbed behaviour shown by the patient. Hoenig & Hamilton (1966, 1969) confirmed that relatives most frequently reported both aggressive behaviour and extreme seclusiveness or withdrawal as causing problems.

The symptoms which Creer & Wing (1974) reported as being most problematic for relatives of those with schizophrenia were the 'negative' ones, those associated with social withdrawal such as lack of conversation, underactivity, slowness and having few leisure interests. The other group of symptoms causing problems were socially embarrassing behaviours and the more obviously disturbed behaviours. The fact that relatives find the 'negative' symptoms of schizophrenia most difficult to cope with was again confirmed by Vaughn (1977) who did a content analysis of critical remarks made by relatives. Only one-third of the remarks concerned delusions, hallucinations or other florid symptoms while two-thirds referred to behaviour such as lack of communication, affection, interest and initiative. Relatives typically did not view these deficiencies as part of the illness but as personality attributes which were under the patient's voluntary control. They described the patients in such disparaging terms as 'lazy', 'selfish' and 'useless'.

In depressive illness, there are also symptoms which can in a broad sense be called negative, such as social withdrawal, quiet misery and so on. Fadden and her colleagues (1987) also reported that it was symptoms of this type that relatives found most difficult to deal with, although florid and embarassing behaviour was also hard to tolerate.

A consistent picture emerges from these accounts. Whereas relatives are apprehensive of florid symptoms, it is the suppressive effects of mental illness on behaviour that cause the most problems, and this is partly due to the difficulty which relatives have in attributing such effects to mental illness.

Relatives' burdens and the mental health services

A prominent theme in this literature is the tolerance of relatives, and the relevance of this to the support they receive from the professionals caring for the patient. Mills (1962) noted that relatives accepted their burdens in spite of the great sacrifices involved and, in protecting the patient, endured really difficult behaviour. They sought re-admission only as a last resort. Wing and his colleagues (1964) emphasised that relatives did their best to put up with very disturbed behaviour, complained little and were willing to take on the role of nurse, frequently at the cost of considerable discomfort and distress.

Waters & Northover (1965) also remarked that relatives showed surprising tolerance of disturbed behaviour. Brown and his colleagues (1966) felt strongly that too much was sometimes being asked of relatives who, however, did not complain, either because they were too ashamed to talk about their problems or because they had concluded that no effective help would be offered. It was evident that relatives needed expert aid, which they received only when their difficulties had reached a crisis. Brown and his colleagues (1966) warn against those involved in service provision assuming patients are better off at home because the majority of relatives do not complain: "The fact that there is this lack of complaint cannot be interpreted as a justification of community care".

Hoenig & Hamilton (1966, 1969) found an interesting discrepancy between objective and subjective burden. Almost a quarter of the households carrying a good deal of objective burden made no complaints of subjective burden, reflecting once more the families' tolerance towards the mentally ill member. A more disturbing finding was that, among those people who felt that nothing more could be done for them, three-quarters suffered some objective burden, and almost a half complained of subjective burden. This shows the lack of expectation of help on the part of these relatives, and in fact only 7% of relatives in the study made any complaint about services.

Creer & Wing (1974) reported that the relatives of their schizophrenic group rarely complained about their difficulties for reasons of shame, guilt, denial of problems or unfavourable experiences when they had sought help. Virtually none of the relatives had received advice from professionals on the management of difficult behaviour, and those who had worked out methods of dealing with problems did so by a painful process of trial and error. The authors concluded that there is no general recognition of the fact that relatives were functioning as 'primary care' agents, and suggested the introduction of a counselling scheme that would provide families with information and strategies for dealing with difficult situations.

In their further study of chronic patients of whom over 40% were cared for or supported by relatives, Creer and her colleagues (1982) also looked at how relatives felt about providing support—whether they were content, resigned or dissatisfied. It was clear that the situation reported by Creer & Wing (1974) was largely unchanged. These relatives were coping with very difficult behaviours, for the most part without complaint. The authors were particularly concerned about the failure of professionals to meet relatives' needs, including those for practical assistance and advice, for emotional support, and for providing them with occasional breaks from their demanding task. They pointed out that no professional group was concerned with the problems of relatives in their own right, and that services were almost exclusively patient-oriented.

Wing (1982) reiterated the problems faced by relatives who have no training in dealing with difficult behaviour, who unlike hospital staff are 'on duty' all the time, and whose emotional involvement with the patient makes it difficult for them to remain neutral in their interactions with patients. Once again he advocated that professionals should make themselves aware of the real problems that arise in families.

A number of other authors have drawn clear inferences from their findings for the way services are provided for the families of mental patients. Mandelbrote & Folkard (1961*a*,*b*) recommended that the burden on families should be lightened by arranging facilities to take the patient from the home for part of the day, and that more social workers were needed to deal with interpersonal problems within the families.

Mills (1962) was also of the opinion that relatives needed relief part of the day, at night or during crises, and that these patients remained in the community only at the cost of considerable hardship. She concluded:

> If patients are more often to be treated from their own homes, then their families should not have to bear without help the severe practical problems and strains.

Waters & Northover (1965) regarded the lack of sustained support of patients' families as one of the important shortcomings of the community after-care provided for the patients. These papers were both written over 20 years ago, and there is no evidence that service provision has changed markedly. The implications are stark.

The behaviour of professionals towards relatives

It is clear that professionals do not provide much help for relatives. The early American study described above (Clausen & Yarrow, 1955) examined how this lack of provision was related to the communication between wives and their husbands' psychiatrists, and to the attitudes which each held towards the other (Deasy & Quinn, 1955). An analysis of the requests made of the psychiatrists by wives revealed that most (71%) were efforts to secure information regarding aetiology, diagnosis and prognosis, and advice on how to deal with the patient when he returned home. The remaining requests were either for help with personal problems (9%) or attempts to change the course of hospitalisation (20%). However, in almost two-thirds of cases, the wives expressed dissatisfaction because they were not provided with the information they required or because the doctors were inaccessible. Most psychiatrists considered it reasonable that wives should expect information, and were aware that they did not always fulfil the needs of patients' families, either because of their heavy workload or because the nature of psychiatric illness made it difficult to answer many of the questions posed. Nevertheless the 23 psychiatrists interviewed focussed their attention almost exclusively on their patients, and contacted relatives only at the early stages of hospitalisation to secure information. Although they agreed that wives needed help from some source, they did not see this as their responsibility. When asked to describe the characteristics of a 'good' wife, they used terms such as "she has insight into her husband's condition, lets the doctors alone, co-operates with the hospital's plans for the patient". A 'bad' wife on the other hand was someone who "exhibits signs of emotional distress, tries to thwart the hospital, takes up a great deal of the doctor's time". Deasy & Quinn (1955) reported that the psychiatrists frequently felt they had to protect the patient from his wife as they believed that factors in the husband–wife relationship had contributed to the patient's illness.

It can be imagined that these attitudes, whatever their level of justification, do help to meet the relatives' needs. Although no subsequent study has looked specifically at professional attitudes, these themes continue to arise in reports of relatives' dealings with staff.

Community care and the burdens of relatives

The impetus for many of the studies described here was provided by the move towards community care. It is clear that if more patients are in the community, more relatives will have to care for them and will experience a consequential burden. A worrying finding, however, emerges from the first study specifically designed to examine the differential effects of a community-oriented service and a more traditional hospital-based service on the relatives of psychiatric patients (Grad & Sainsbury, 1963*a*,*b*). Not only were more people caring for relatives in the first scheme, but their degree of burden was actually greater. The authors speculated that this was because the burden on families in the traditional hospital approach was lightened by regular visits to the homes from the social work staff, while this was not the case in the community-based service.

Sex differences

Few investigators have reported on the differential effects of the sex of the relative on the degree of burden. In many cases, this is because studies only looked at male patients.

Rogler & Hollingshead (1965) did examine gender difference in the degree of burden. They reported that schizophrenic illness in a wife had a more disruptive effect on the family than that in a husband. However, the study was carried out in the then rather traditional society of Puerto Rico and the findings have doubtful applicability to other cultures.

Few British studies have paid much attention to sex differences. Mandelbrote & Folkard (1961*a*,*b*) noted that, for male patients, there were more disturbed conjugal than parental families, this situation being reversed when the patient was female. In the study of depressed patients by Fadden and her colleagues (1987), wives were more isolated, possibly because they had fewer outlets to exploit when their spouse became mentally ill. It was also remarkable that wives often found it upsetting to have to take on traditionally male roles, even though they managed to do so competently. Such findings may have more general relevance and should perhaps be anticipated in the clinic.

Relatives' burdens in depressive disorder

The scant information about the specific effects of depression on the family of the patient has been recently reviewed elsewhere (Kuipers, 1987). It deals exclusively with the effects on spouses, although this may not be inappropriate as depressive patients are much more likely to be married and living with the marital partner than are people with schizophrenia. In these marriages there is frequently conflict (Hinchcliffe *et al*, 1978), particularly over role functions (Ovenstone, 1973*b*) and a high level of dependence (Birtchnell & Kennard, 1983). Fewer joint decisions are made with increasing pathology on the part of the husband (Collins *et al*, 1971), and the wives of depressive patients have significantly less independent social activity than controls (Nelson *et al*, 1970).

Fadden and her colleagues (1987) have recently conducted a pilot study of the spouses of persistently depressed patients of specified types, employing techniques of assessing burden developed in the more sophisticated studies of the effects of schizophrenia. Their findings show striking parallels with that work and emphasise the severity of the burden borne by spouses and the noticeably adverse effect upon their mental health.

Depression is not a minor problem for the community: the life-time expectancy of contacting a psychiatric hospital with depression may be as high as 11.9% for men and 20.2% for women (Sturt *et al*, 1984). Nor is it a problem which leads only to short periods of disability. In many cases the problem becomes a chronic one: Mann & Cree (1975) in a study of 'new long-stay' patients aged between 18 and 65 in hospitals found that affective psychosis accounted for 15.8% of patients and formed the largest single group after schizophrenia.

It is clear that more extensive study is required to establish the extent and prevalence of the burdens borne by the relatives of depressed patients.

Implications for further research and the provision of services

Although this review shows that there is now a large body of literature based on the systematic study of relatives' difficulties, much less is known of the best strategies for relatives to use in coping with their problems and of how, even if professionals knew what to advise, they could ensure that their advice was adopted. There is only experiential or anecdotal knowledge in these areas. In their review of research on burden, Kreisman & Joy (1974) describe the 'scatter-shot' approach on the part of researchers who have failed to follow through on promising leads in their own data: "This lack of sustained interest has left us with fundamental pieces of information missing". It must be said that, 13 years later, the situation remains very similar.

The results of this ignorance are likely to be considerable. It is apparent from the earlier studies on 'Expressed Emotion' (Brown *et al*, 1962, 1972; Vaughn & Leff, 1976) that the behaviour of relatives has a significant influence on the patient's likelihood of relapse with schizophrenia. This finding probably applies equally to depression (Vaughn & Leff, 1976; Hooley *et al*, 1986). While not all relatives behave in the same way, it is the nature of the burden placed on them that leads some to resort to ineffective strategies of coping. The intervention studies that have been reported (Liberman *et al*, 1981; Falloon *et al*, 1982; Leff *et al*, 1982; Barrowclough & Tarrier, 1984) and those now proceeding all adopt approaches that broadly aim to enhance the effectiveness of the relative's coping. The results confirm that improving the way relatives deal with the burdens of looking after a schizophrenic patient in the home has a beneficial effect on the course of the illness.

In addition to the indirect effects of burden on the patient's likelihood of relapse, there are direct effects upon the relatives' own mental health. This has been documented particularly for those married to depressed or neurotic partners (Kreitman, 1964;

Kreitman *et al*, 1970; Ovenstone, 1973*a*; Fadden *et al*, 1987) but has been noted for those living with schizophrenic patients (Brown *et al*, 1966; Hamilton & Hoenig, 1966, 1969; Stevens, 1972; Creer & Wing, 1974). The evidence inclines against an explanation in terms of assortative mating, at least in the groups studied.

Conclusion

A number of points can be drawn from this literature on the effects of psychiatric illness upon the families who live with the patient. A more widespread interest was shown in the topic in the late 1950s and early 1960s when community care programmes were first introduced. The first attempts to refine the measurement of burden in families also date from this time. Schizophrenic patients constitute the only group in which interest in family burden has been sustained, although there is evidence that families of other patient groups are also seriously affected by the patient's illness.

It is clear that families typically tolerate a great deal of difficult behaviour and that they frequently find the less clear-cut symptoms the most difficult to bear. They often lack knowledge about the nature of the patient's illness but receive little help from professionals in the management of difficult behaviour except in times of crisis. In spite of all this they rarely make complaints. Coping with their relatives' problems frequently results in adverse effects on their own health, both physical and psychological. From the first studies to the most recent, the point has been made repeatedly by those working in the area that relatives must not be asked to bear these burdens unassisted, and that they should be provided with more help from professionals. Recent studies describing interventions with families have shown that such help can be effective and worthwhile.

The intervention studies that have already been carried out have resulted in reduced relapse rates for schizophrenic patients, and this might also be a realistic aim for other diagnostic groups. A prerequisite of such intervention is knowledge of what problems are in fact faced by relatives. The need for further research is therefore clear.

References

Barrowclough, C. & Tarrier, N. (1984) 'Psychosocial' interventions with families and their effects on the course of schizophrenia: a review. *Psychological Medicine*, **14**, 629–642.

Birtchnell, J. & Kennard, J. (1983) Does marital maladjustment lead to mental illness? *Social Psychiatry*, **18**, 79–88.

Brown, G. W., Monck, E. M., Carstairs, G. M. & Wing, J. K. (1962) Influence of family life on the course of schizophrenic illness. *British Journal of Preventive and Social Medicine*, **16**, 55–68.

——, Bone, M., Dalison, B. & Wing, J. (1966) *Schizophrenia and Social Care*. London: Oxford University Press.

——, Birley, J. L. T. & Wing, J. K. (1972) Influence of family life on the course of schizophrenic disorders: A replication. *British Journal of Psychiatry*, **121**, 241–258.

Bruner, J. & Taguiri, R. (1954) The perception of people. In *Handbook of Social Psychology* (ed. G. Lindzey). Cambridge, Mass: Addison-Wesley.

Clausen, J. A. & Yarrow, M. R. (1955) The impact of mental illness on the family. *Journal of Social Issues*, **11**, 3–64.

——, Yarrow, M. R., Deasy, L. C. & Schwartz, C. G. (1955) The impact of mental illness: research formulation. *Journal of Social Issues*, **11**, 6–11.

Collins, J., Kreitman, N., Nelson, B. & Troop, J. (1971) Neurosis and marital interaction: III Family roles and functions. *British Journal of Psychiatry*, **119**, 233–242.

Creer, C. & Wing, J. K. (1974) *Schizophrenia at Home*. Surbiton: National Schizophrenia Fellowship.

——, Sturt, E. & Wykes, T. (1982) The role of relatives. In *Long Term Community Care: Experience in a London Borough* (ed. J. K. Wing) *Psychological Medicine*, Monograph Supplement 2.

Deasy, L. C. & Quinn, O. W. (1955) The wife of the mental patient and the hospital psychiatrist. *Journal of Social Issues*, **11**, 49–60.

Fadden, G. B., Kuipers, L. and Bebbington, P. E. (1987) Caring and its burdens: a study of the relatives of depressed patients. *British Journal of Psychiatry*. (In press).

Falloon, I. R. H., Boyd, J. L., McGill, C. W., Razani, J., Moss, H. B. & Gilderman, A. M. (1982) Family management in the prevention of exacerbations of schizophrenia. A controlled study. *New England Journal of Medicine*, **306**, 1437–1440.

Grad, J. & Sainsbury, P. (1963*a*) Evaluating a community care service. In *Trends in Mental Health Services* (eds H. Freeman & J. Farndale), Oxford: Pergamon.

—— & Sainsbury, P. (1963*b*) Mental illness and the family. *Lancet*, **i**, 544–547.

Hinchcliffe, M. K., Hooper, D. & Roberts, F. J. (1978) *The Melancholy Marriage*. Chichester: Wiley.

Hoenig, J. & Hamilton, M. W. (1966) The schizophrenic patient in the community and his effect on the household. *International Journal of Social Psychiatry*, **12**, 165–176.

—— & Hamilton, M. W. (1969) *The Desegregation of the Mentally Ill*. London: Routledge & Kegan Paul.

Hooley, J. M., Orley, J. & Teasdale, J. (1986) Levels of Expressed Emotion and relapse in depressed patients. *British Journal of Psychiatry* **148**, 642–647.

Kreisman, D. E. & Joy, V. D. (1974) Family response to the mental illness of a relative: a review of the literature. *Schizophrenia Bulletin*, **10**, 34–57.

Kreitman, N. (1964) The patient's spouse. *British Journal of Psychiatry*, **110**, 159–173.

——, Collins, J., Nelson, B. & Troop, J. (1970) Neurosis and marital interaction: I Personality and symptoms. *British Journal of Psychiatry*, **117**, 33–46.

Kuipers, L. (1987) Depression and the family. In *Coping with Disorder in the Family* (ed. J. Orford) London: Croom-Helm.

Leff, J. P., Kuipers, L., Kerkowitz, R., Eberlein-Fries, R. & Sturgeon, D. (1982) A controlled trial of social intervention in schizophrenic families. *British Journal of Psychiatry*, **141**, 121–134.

Liberman, R. P., Wallace, C. J., Falloon, I. R. H. & Vaughn, C. E. (1981) Interpersonal problem solving therapy for schizophrenics and their families. *Comprehensive Psychiatry*, **22**, 627–630.

Mandelbrote, B. M. & Folkard, S. (1961*a*) Some problems and needs of schizophrenics in relation to a developing psychiatric community service. *Comprehensive Psychiatry*, **2**, 317–328.

—— & Folkard, S. (1961*b*) Some factors related to outcome and social adjustment in schizophrenia. *Acta Psychiatrica Scandinavica*, 37, 223–235.

Mann, S. & Cree, W. (1975) The 'new long-stay' in mental hospitals. *British Journal of Hospital Medicine*, 14, 56–63.

Mills, E. (1962) *Living with Mental Illness: A Study in East London*. London: Routledge & Kegan Paul.

Nelson, B., Collins, J., Kreitman, N. & Troop, J. (1970) Neurosis and marital interaction: II Time sharing and social activity. *British Journal of Psychiatry*, 117, 47–58.

Ovenstone, I. M. K. (1973*a*) The development of neurosis in the wives of neurotic men. Part 1: symptomatology and personality. *British Journal of Psychiatry*, 122, 33–43.

—— (1973*b*) The development of neurosis in the wives of neurotic men. Part 2: Marital role functions and marital tension. *British Journal of Psychiatry*, 122, 711–717.

Platt, S. (1981) Social adjustment as a criterion of treatment success: just what are we measuring? *Psychiatry*, 44, 95–112.

—— (1985) Measuring the burden of psychiatric illness on the family: an evaluation of some rating scales. *Psychological Medicine*, 15, 383–394.

Rogler, L. & Hollingshead, A. (1965) *Trapped: Families and Schizophrenia*. New York: Wiley.

Schachter, S. (1951) Deviation, rejection and communication. *Journal of Abnormal and Social Psychology*, 46, 190–207.

Stevens, B. (1972) Dependence of schizophrenic patients on elderly relatives. *Psychological Medicine*, 2, 17–32.

Sturt, E. S., Kumakura, N. & Der, G. (1984) How depressing life is: lifelong risk of depression in the general population. *Journal of Affective Disorders*, 7, 109–122.

Vaughn, C. E. (1977) Patterns of interactions in families of schizophrenics. In *Schizophrenia: The Other Side* (ed. H. Katschnig). Vienna: Urban & Schwarzenberg.

—— & Leff, J. P. (1976) The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129, 125–137.

Waters, M. A. & Northover, J. (1965) Rehabilitated long-stay schizophrenics in the community. *British Journal of Psychiatry*, 111, 258–267.

Wing, J. K. (1982) Rehabilitation and management of schizophrenia. In *Handbook of Psychiatry III: Psychoses of Uncertain Aetiology* (eds J. K. Wing and L. Wing). London: Cambridge University Press.

——, Monck, E., Brown, G. W. & Carstairs, G. M. (1964) Morbidity in the community of schizophrenic patients discharged from London mental hospitals in 1959. *British Journal of Psychiatry*, 110, 10–21.

Yarrow, M., Clausen, J. & Robbins, P. (1955*a*) The social meaning of mental illness. *Journal of Social Issues*, 11, 33–48.

——, Schwartz, C. G., Murphy, H. S. & Deasy, L. C. (1955*b*) The psychological meaning of mental illness in the family. *Journal of Social Issues*, 11, 12–24.

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The Psychological Well-being of Supporters of the Demented Elderly

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Interviews were conducted with the co-resident supporters of 79 elderly subjects. Forty of these elderly subjects had been diagnosed as being demented (20 mildly, 12 moderately and eight severely) following psychiatric assessment. The supporters were screened for psychological well-being with the 60-item General Health Questionnaire (GHQ) and the Relatives' Stress Scale (RSS). Supporters of demented relatives showed significantly raised levels of stress on the RSS, but no increase in psychiatric morbidity on the GHQ, when compared with the supporters of non-demented relatives. The implications of these findings are discussed.

Community surveys (Kay *et al*, 1964; Parsons, 1965) have established that the great majority of demented elderly people are cared for at home. That is to say, the burden of caring for such patients falls specifically on those relatives and friends who attempt to support them in the community.

It has recently been suspected that these supporters of the demented elderly may be under considerable strain, and indeed they have been referred to as 'hidden patients' (Fengler & Goodrich, 1979). Studies of the families of the demented elderly (Grad & Sainsbury, 1965; Hoenig & Hamilton, 1969; Greene *et al*, 1982; Rabins *et al*, 1982; Gilleard, 1984; Gilleard *et al*, 1984) have found that the supporters experience high levels of stress and psychiatric disturbance. However, these studies have almost exclusively examined the families of patients referred to psychiatric services. As Grad (1971) and Gilleard *et al* (1984) have acknowledged, such patients and their families may be quite atypical of those in the community, and indeed may have been referred by their general practitioners *precisely on account* of the high levels of psychological stress within the family (Eagles *et al*, 1987).

Gilleard (1984) found that the supporters of patients attending a geriatric day hospital are less likely to be psychiatrically disturbed than the supporters of patients attending a psychogeriatric day hospital. This study apart, 'control' families have not been utilised in determining the effects of a demented person on his or her family; it is not known how much stress and psychological illness is precipitated by caring for a *non-demented* elderly relative.

The present study thus examined the psychological well-being of, and the strains perceived by, supporters of demented elderly subjects who had not been identified by referral to psychiatric services. It also investigated the same parameters in the supporters of a group of non-demented elderly subjects.

Method

The study was conducted in a general practice in Aberdeenshire. The practice has just over 14 000 patients of whom about half live in the town in which the practice is centred. The other patients reside in villages and in the country nearby.

A screening investigation of all subjects in the practice who were over 65 years of age had been carried out between August 1980 and April 1982. This survey has been described in more detail elsewhere (Eagles *et al*, 1987). A component of the screening investigation was the completion of the Mental Status Questionnaire (MSQ) as described by Kahn *et al* (1960) and by Wilson & Brass (1973). The MSQ consists of ten questions testing memory and orientation and it was answered by 1765 elderly subjects. Of this cohort, 205 scored 8 or less, indicating the possible presence of dementia. In order to investigate the follow-up validity of the MSQ (results of which will not be reported here) these 205 subjects were matched for age and sex with 205 subjects scoring 9 or 10 on the MSQ, both these groups of subjects being reinterviewed between September 1983 and May 1984. This interview was by a psychiatrist with experience in geriatric psychiatry (J.M.E. or F.R.) and included a repeat MSQ and the administration of the Orientation–Memory–Concentration Test (OMCT) described by Katzman *et al* (1983). This is an abbreviated, six-item version of the Information–Memory–Concentration Mental Status Test devised by Blessed *et al* (1968). The OMCT has the advantage that, unlike other brief screening tests for dementia, it has been positively correlated against cortical plaque counts at post mortem. The psychiatrist's interview, when possible and appropriate, included the gathering of information from relatives, and it concluded with the psychiatrist's assigning the subject to one of four categories: no dementia, mild dementia, moderate dementia or severe dementia.

For the present study, subjects who lived with an identifiable 'supporter' were selected. Supporters were asked, at the time of the psychiatrist's visit, whether they would be willing to receive a further visit from another doctor (A.C.), who would wish to discuss what problems, if any, they experienced in caring for their elderly relative. The first 80 supporters who agreed to this interview were included in the study. Since the order in which elderly subjects were visited depended only on their addresses it was likely that these 80 subjects comprised a fairly equal number of subjects whose 1980–1982 MSQ had been greater than 8 and whose 1980–1982 MSQ had been 8 or less. One supporter subsequently declined to participate on the day of A.C.'s visit. The interview of the supporter comprised:

1. *The 60-item General Health Questionnaire* (GHQ) (Goldberg 1972, 1978), a widely used instrument for the assessment of the presence and the degree of psychiatric morbidity.
2. *Relatives' Mood Scale* (RMS) (Greene *et al*, 1983). Supporters rated their mood on five symptoms—depressed, irritable, tired, tense and worried—using a 100 mm visual analogue scale, the ends of which were labelled 'not at all' and 'extremely'. A total mood score was represented by a mean of these five scores. Some supporters, particularly those who were elderly, found the concept of the visual analogue scale impossible to comprehend, and only 70 of the 79 supporters successfully completed the RMS.
3. *Relatives' Stress Scale* (RSS) (Greene *et al*, 1982). This is a 15-item scale designed to measure the stresses experienced by a relative who is caring for an elderly dependant in the community. The 15 questions relate to such topics as the effect the dependant is having on the relative's social life and mood state, and assesses negative feelings of the relative towards the dependant. Greene *et al* (1982) used a five-point scale of response to each question, but the present study simplified this to a three-point scale. Thus, a typical question reads: "Do you ever get depressed by the situation", and the possible responses were: (a) A great deal of the time, (b) Sometimes, (c) Never. The responses were then summated scoring 2 for the most 'stressed' response, 1 for the moderately 'stressed' response and 0 for the least 'stressed' response. The summation of these responses thus yielded a measure of the overall stress experienced by a supporter in caring for their elderly dependant.
4. *The Behaviour Rating Scale (BRS) of the Clifton Assessment Procedures for the Elderly (CAPE).* (Gilleard & Pattie, 1977). The BRS is an 18-item behaviour rating scale which is completed by an informant, and supporters rated the behaviour of their dependant with this instrument. Each item is rated on a three-point scale, and items cover a range of disturbed and/or dependent behaviour such as incontinence, immobility and night wandering. When the items are summated the total indicates the overall level of behavioural disturbance. The scale has been validated against outcome in elderly psychiatric patients (Pattie & Gilleard, 1978).

Thus, the supporter provided information concerning her psychological well-being (the GHQ and RMS), the stresses she experienced in caring for her elderly dependant (the RSS) and her assessment of the behavioural disturbance exhibited by her dependant (the BRS). In tandem with the data from the psychiatrist's assessments of the elderly subjects, these data from the supporters were analysed to address the principal question: does the presence, and the degree, of dementia affect the supporter's psychological well-being and/or the stresses she experiences in caring for her elderly dependant in the community.

Results

Of the 79 elderly subjects, 40 were male and 39 were female. Their mean age was 79.3 years (s.d. 6.5). The psychiatrist's ratings of the elderly subjects' mental state yielded the following:

No dementia	39
Mild dementia	20
Moderate dementia	12
Severe dementia	8

Thus, 39 subjects were adjudged to be non-demented and 40 to be demented, and these will be referred to as the 'non-demented group' and the 'demented group' respectively.

Of the 79 supporters interviewed, 21 were male and 58 female. Of these supporters, 41 were spouses, 25 were children, seven were siblings and six were a more distant relative or friend. Their mean age was 64.9 years (s.d. 13.9) with an age range of 22 to 88 years.

Table I shows the mean scores of supporters and dependents on each of the screening tests, grouped by the psychiatrist's ratings as to the presence and degree of dementia in the dependants.

Predictably, as global rating of dementia becomes more severe, the subjects perform more poorly on tests of cognitive function (MSQ and OMCT) and have higher levels of behavioural disturbance on the BRS of the CAPE.

The relationship between the mental state of the dependant and the psychological well-being of the supporter was investigated in two principal ways. Firstly the responses to the GHQ, the RMS and RSS were statistically compared for supporters of the demented group of dependants with those of the non-demented group of dependants. These comparisons were made by means of Mann-Whitney U Tests. The results are shown in Table II.

Supporters of demented and of non-demented dependants do not differ significantly in their scores on the 60-item GHQ. The two groups of supporters do differ at a highly significant level in their scores on the RSS ($P<0.001$) and there is a trend towards their scores differing on the RMS ($P<0.1$).

Since dementia increases in prevalence with increasing age it was possible that the 'demented group' would be significantly older than the non-demented group, and that this increased age and resultant infirmity itself, rather than the presence of dementia, might affect the well-being of the supporters of the demented group. The mean age of the demented group was 79.8 years (s.d. 6.4) and that of the non-demented group was 78.7 years (s.d. 6.7). These groups

TABLE I

Dependants' and supporters' mean scores on screening tests by psychiatrist's global rating of dependant (Standard deviations in parentheses)

	All patients ($n=80$)	*No dementia ($n=39$)*	*Mild dementia ($n=20$)*	*Moderate dementia ($n=12$)*	*Severe dementia ($n=8$)*
Dependants	7.18	9.13	7.30	4.50	1.38
MSQ	(2.85)	(0.95)	(1.81)	(1.38)	(1.19)
OMCT	17.15	24.08	14.55	9.08	2.00
	(8.66)	(4.23)	(4.30)	(3.66)	(2.51)
BRS	7.05	3.80	7.50	11.58	15.00
	(5.90)	(3.56)	(4.43)	(6.24)	(6.14)
GHQ	3.85	3.62	2.95	5.25	5.13
Supporters	(6.51)	(5.74)	(4.38)	(11.10)	(6.29)
Mood scale	16.14	12.63	14.61	26.82	21.67
	(18.76)	(17.00)	(13.27)	(26.11)	(23.40)
Stress scale	4.94	2.69	5.50	7.50	10.63
	(5.54)	(3.34)	(5.86)	(6.36)	(6.84)

did not differ significantly when compared by means of a *t*-test ($F=1.08$, $t=0.79$, NS). Furthermore, the correlation between the age of the dependants and the RSS score of their supporters did not reach statistical significance ($\tau=0.098$, NS).

The relationship between supporters' psychological well-being and the *severity* of the dependants' dementia was investigated by comparisons of the groups of dependants arising from the psychiatrist's global ratings as to the presence and severity of dementia. The elderly subjects were grouped as: no dementia ($n=39$), mild dementia ($n=20$), and moderate/severe dementia ($n=20$). Mann-Whitney U Tests were carried out to investigate differences in the supporters' scores on the GHQ, RMS and RSS between supporters of the non-demented group, the mildly demented group, and the moderately/severely demented group.

These analyses showed that, for the three groups of dependants (no dementia, mild dementia, moderate/severe dementia):

1. *GHQ*: there were no significant differences in GHQ scores between the three groups of supporters.
2. *RMS*: the supporters of the mildly demented groups did not differ significantly from those of the other two groups. There was a trend ($P<0.1$) towards the moderately/severely demented group's supporters scoring more highly than those of the non-demented group.
3. *RSS*: there were trends ($P<0.1$) towards differences in the scores of the supporters of the mildly demented group and those of the other two groups in the expected directions. The supporters of the non-demented group had very significantly lower scores ($P<0.001$) than those of the moderately/severely demented group.

The second principal means of testing the relationship between the supporters' psychological well-being and the disability of their dependant was by the calculation of correlation coefficients (Kendall's τ) between the supporters' scores on the GHQ, RMS and RSS and the dependants' scores on the Mental Status Questionnaire (MSQ), the Orientation–Memory–Concentration Test (OMCT) and on the Behaviour Rating Scale (BRS) of the CAPE. The results of these analyses are shown in Table III.

Neither the supporters' GHQ scores nor their RMS scores correlates significantly with the two measures of cognitive impairment in the dependants, the MSQ and the OMCT. The supporters' scores on the RSS however, correlate at a highly significant level ($P<0.001$) with measures of the dependants' cognitive impairment (MSQ and OMCT) and with their level of behavioural disturbance as rated on the

TABLE II

Psychological well-being of supporters of demented and of non-demented dependants

		Demented dependant	*Non-demented dependant*	*Mann-Whitney U*	*z*	*P*
Supporters'	Median	0.90	0.67			
GHQ	Range	0–39	0–25	748.5	0.33	NS
Supporters'	Median	16.00	7.88			
Mood Scale	Range	0–79	0–65	469.5	1.73	0.08
Supporters'	Median	5.75	1.71			
Stress Scale	Range	0–20	0–12	436.5	3.39	0.0007

TABLE III
Supporters' well-being by dependants' disability (Kendall's tau)

	Supporters' GHQ score	*Supporters' mood scale Total*	*Supporters' strain scale Total*
Dependants' MSQ score	−0.03	−0.03	−0.30**
Dependants' OMC score	−0.11	−0.11	−0.32**
Dependants' BRS score	0.25*	0.33**	0.57**

* Denotes $P<0.01$
**Denotes $P<0.005$

BRS of the CAPE. The dependants' BRS scores are also significantly correlated with the supporters' scores on the RMS and on the GHQ. It should be noted that the BRS scores derive from the supporters' (subjective) appraisal of their dependants' behaviour. Thus, psychological morbidity and/or strain in the supporters may result in their rating their dependants' behaviour as more disturbed on the BRS, rather than the morbidity and strain necessarily resulting from the dependants' disturbed behaviour.

The scores of supporters on the RSS did not appear to be independent of their psychiatric morbidity, as measured by the GHQ and RMS scores. Correlation coefficients (Kendall's τ) between the supporters' scores on the three scales were as follows:

RSS with GHQ, $\tau=0.34$
RSS with RMS, $\tau=0.34$
GHQ with RMS, $\tau=0.44$.

All these correlations are highly statistically significant ($P<0.001$).

The two relatively large groups of supporters i.e. spouses ($n=41$) and children ($n=25$), were compared for differences in their levels of psychological morbidity and strain by comparisons of their scores on the GHQ, RMS and RSS with Mann-Whitney U tests. No statistically significant difference was found between these two groups of supporters on any of the three scales.

Discussion

This study has shown that the co-resident supporters of elderly demented patients experience a significantly increased degree of stress, as measured by the RSS, when compared to relatives living with a non-demented old person. The stress experienced escalated significantly with increasing severity of dementia in the elderly dependants. There was no hard evidence, on the other hand, that psychiatric morbidity in the supporter (as measured by the 60-item GHQ) was influenced either by the presence or by the severity of dementia in the elderly dependant. While this finding accords with those of Eagles *et al* (1987), other researchers have almost universally found high levels of psychiatric morbidity in supporters of the demented elderly (Grad & Sainsbury, 1965; Hoenig & Hamilton, 1969; Greene *et al*, 1982; Rabins *et al*, 1982; Gilleard, 1984; Gilleard *et al*, 1984).

The possible reasons for the discrepancy in these findings has been discussed elsewhere (Eagles *et al*, 1987), and will be only briefly rehearsed here. One of the most probable explanations is that of sampling bias. Other studies into the psychiatric well-being of the supporters of demented relatives have investigated families in which the demented person has been the subject of psychiatric referral. These patients may have been selected by the primary care team for psychiatric referral as a direct result of the relatives' psychiatric morbidity, while patients whose families 'cope' without becoming psychiatrically unwell may not be referred to psychiatrists at all.

It is possible that the people of Aberdeenshire are atypical of other areas of the UK. This is partly supported by the finding of Gilhooley (1984), that none of the relatives caring for 37 demented attenders at an Aberdeen Day Hospital had serious psychiatric morbidity. The area in which the present study was based has a high degree of community cohesiveness, and many supporters seemed remarkably stoical and uncomplaining about their caring for a demented relative. Jolley & Arie (1978) summarised one of the major current problems in community care of the elderly when they stated that: "Better standards of living and expectations of the 'good life' do not accord well with caring for a persistently disturbed relative". Perhaps these raised expectations have not yet percolated through to the 'old fashioned' inhabitants of semi-rural Aberdeenshire.

It is interesting to note that the high scores of supporters of demented relatives on the RSS did not 'spill over' into significantly raised scores on the 60-item GHQ. This might be related to the geographical stability and community cohesiveness of the area, making it more likely that the supporters would themselves be visited and supported, a factor which Zarit *et al* (1980) showed to have the effect of relieving supporters' feelings of 'burden'.

On the other hand, the failure to establish a relationship between caring for a demented relative and psychiatric morbidity on the GHQ may indicate that this instrument is inefficient in detecting psychiatric morbidity in the elderly. This possibility is partly borne out by the fact that a weak relationship *was* found between caring for a demented relative and the supporters' scores on the RMS, a very brief and relatively unrefined instrument which many elderly supporters did not fully comprehend. Benjamin *et al* (1982) and Goodchild & Duncan-Jones (1985) have recently drawn attention to the somewhat anomalous GHQ response category 'No more than usual'. These authors argue that for items describing pathology, this response can indicate chronic illness rather than good health, and this may be a particularly cogent criticism in evaluating the responses of elderly people who have been caring for a demented relative over a lengthy period of time.

Correlations of supporters' levels of strain and psychiatric morbidity with their dependants' scores on the CAPE were higher than the correlations with dependants' scores of cognitive impairment on the MSQ and OMCT. While the supporters' rating of their dependants' behaviour on the CAPE is unlikely to be independent of their own feelings of strain and distress, it is not surprising that behavioural disturbance rather than cognitive impairment *per se* appears to give rise to psychiatric morbidity. Indeed, this mirrors the findings of Isaacs *et al* (1972) who, investigating the families of geriatric medical referrals, found that relatives did not suffer 'undue strain' on account of their dependants' confusion *per se*, but that strain did arise from concomitant behavioural disturbances. Other studies (Sanford, 1975; Gilleard *et al*, 1982; Greene *et al*, 1982; Rabins *et al*, 1982) have established that there are specific behavioural problems (e.g. night-time wandering, incontinence, violence and apathy), which supporters find particularly stressful and difficult to tolerate in their elderly dependants.

The findings of this study have service implications. Given that the great majority of the supporters of the demented elderly are 'stressed' rather than psychiatrically disturbed, psychiatrists do not require to become involved with these families. The task of 'supporting the supporters' can appropriately be left in the hands of the voluntary agencies and primary care teams.

The present study indicates that supporting a demented relative who has not been 'selected' by referral to psychiatric services does incur a considerable strain on relatives, but does not in itself lead to increased rates of psychiatric morbidity as measured by the General Health Questionnaire. While this requires to be confirmed in other parts of the country, and while there are doubts about the utility of the GHQ in an elderly population under chronic stress, this evidence suggests that psychiatric screening of these relatives would not be a productive venture. It is worth emphasising that this finding does not diminish the need to provide community support for relatives who are attempting to cope with a demented elderly dependant. Indeed it is quite possible that it is this very support, which is well co-ordinated in the general practice studied, that prevents the transition of relatives from 'stress' to overt psychiatric illness.

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References

Benjamin, S., Decalmer, P. & Haran, D. (1982) Community screening for mental illness: a validity study of The General Health Questionnaire. *British Journal of Psychiatry*, **140**, 174–180.

Blessed, G., Tomlinson, B. E. & Roth, M. (1968) The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry*, **114**, 797–811.

Eagles, J. M., Beattie, J. A. G., Blackwood, G. W., Restall, D. B. & Ashcroft, G. W. (1987) The mental health of elderly couples: I, The effects of a cognitively impaired spouse. *British Journal of Psychiatry*, **150**, 299–308.

Fengler, A. P. & Goodrich, N. (1979) Wives of elderly disabled men: the hidden patient. *Gerontologist*, **18**, 175–183.

Gilhooly, M. L. M. (1984) The impact of care-giving on care-givers: factors associated with the psychological well-being of people supporting a dementing relative in the community. *British Journal of Medical Psychology*, **57**, 35–44.

Gilleard, C. J. (1984) Problems posed for supporting relatives of geriatric and psychogeriatric day patients. *Acta Psychiatrica Scandinavica*, **70**, 198–208.

—— & Pattie, A. H. (1977) The Stockton Geriatric Rating Scale: a shortened version with British normative data. *British Journal of Psychiatry*, **131**, 90–94.

——, Boyd, W. D. & Watt, G. (1982) Problems of caring for the elderly mentally infirm at home. *Archives of Gerontology and Geriatrics*, **1**, 151–158.

——, Belford, H., Gilleard, E., Whittick, J. E. & Gledhill, K. (1984) Emotional distress amongst the supporters of the elderly mentally infirm. *British Journal of Psychiatry*, **145**, 172–177.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire: A Technique for the Identification and Assessment of Non-Psychotic Psychiatric Illness*. London: Oxford University Press.

—— (1978) *Manual of the General Health Questionnaire*. Windsor: NFER Publishing Co.

Goodchild, M. E. & Duncan-Jones, P. (1985) Chronicity and the General Health Questionnaire. *British Journal of Psychiatry*, **146**, 55–61.

Grad, J. (1971) Social causes and social consequences of mental illness in old age. In *Recent Developments in Psychogeriatrics*. Ashford, Kent: Headly Brothers Ltd.

—— & Sainsbury, P. (1965) An evaluation of the effects of caring for the aged at home. In *Psychiatric Disorders in the Aged, W.P.A. symposium*. Manchester: Geigy.

Greene, J. G., Smith, R., Gardiner, M. & Timbury, G. C. (1982) Measuring behavioural disturbance of elderly demented patients in the community and its effect on relatives: a factor analytic study. *Age and Ageing*, **11**, 121–126.

——, Timbury, G. C., Smith, R. & Gardiner, M. (1983) Reality orientation with elderly patients in the community: an empirical evaluation. *Age and Ageing*, **12**, 38–43.

Hoenig, J. & Hamilton, M. W. (1969) *The Desegregation of the Mentally Ill*. London: Routledge & Kegan-Paul.

Isaacs, B., Livingstone, M., Neville, Y. (1972) *Survival of the Unfittest*. London: Routledge & Kegan-Paul.

Jolley, D. J. & Arie, T. (1978) Organisation of psychogeriatric services. *British Journal of Psychiatry*, **132**, 1–11.

Kahn, R. L., Goldfarb, A. I., Pollack, M. & Peck, A. (1960) Brief objective measures for the determination of mental status in the aged. *American Journal of Psychiatry*, **117**, 326–328.

Katzman, R., Brown, T., Fuld, P., Peck, A., Schechter, R. & Schimmel, H. (1983) Validation of a short Orientation-Memory-Concentration Test of cognitive impairment. *American Journal of Psychiatry*, **140**, 734–739.

Kay, D. W., Beamish, P. & Roth, M. (1964) Old age mental disorders in Newcastle Upon Tyne: Part 1, A study of prevalence. *British Journal of Psychiatry*, **110**, 146–158.

Parsons, P. L. (1965) Mental health of Swansea's old folk. *British Journal of Preventive & Social Medicine*, **19**, 43–47. 43–47.

Pattie, A. H. & Gilleard, C. J. (1978) Admission and adjustment of residents in homes for the elderly. *Journal of Epidemiology and Community Health*, **32**, 212–214.

Rabins, P. V., Mace, N. L. & Lucas, M. L. (1982) The impact of dementia on the family. *Journal of the American Medical Association*, **248**, 333–335.

Sanford, J. R. A. (1975) Tolerance of debility in elderly dependents by supporters at home: its significance for hospital practice. *British Medical Journal*, **iii**, 471–473.

Wilson, L. A. & Brass, W. (1973) Brief assessment of the mental state in geriatric domiciliary practice: the usefulness of the mental status questionnaire. *Age and ageing*, **2**, 92–101.

Zarit, S. H., Reever, K. E. & Bach-Peterson, J. (1980) Relatives of the impaired elderly: correlates of feelings of burden. *Gerontologist*, **20**, 649–655.

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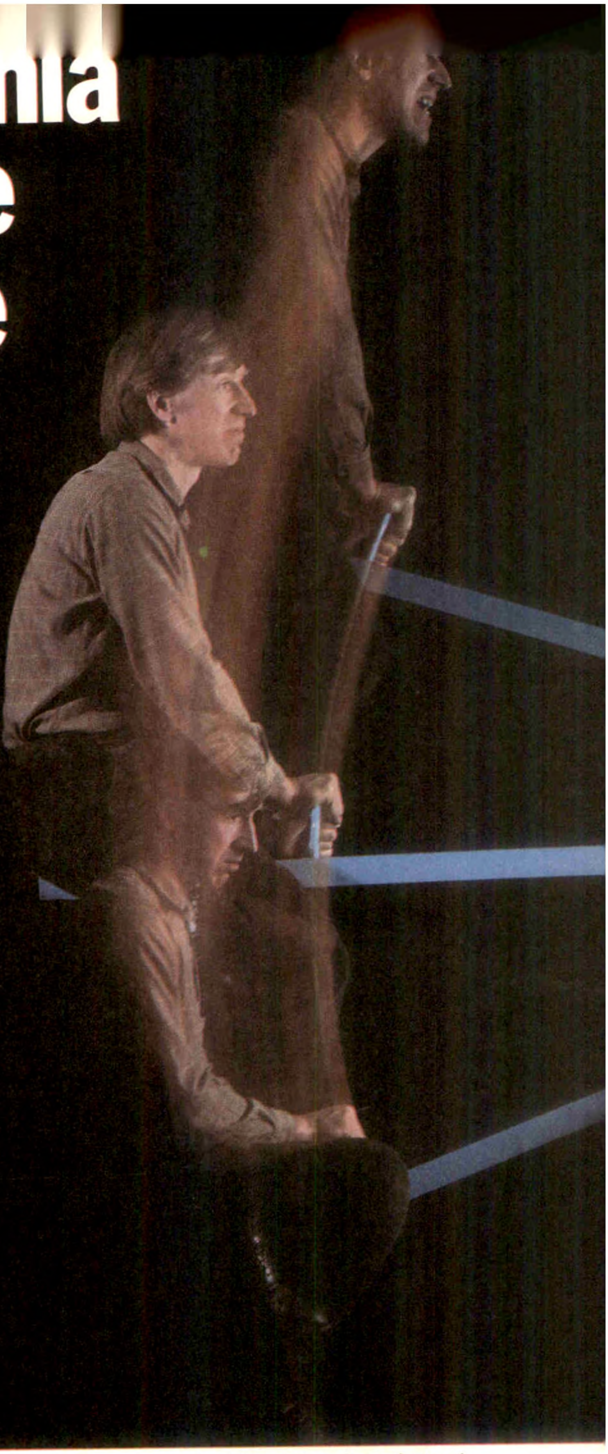

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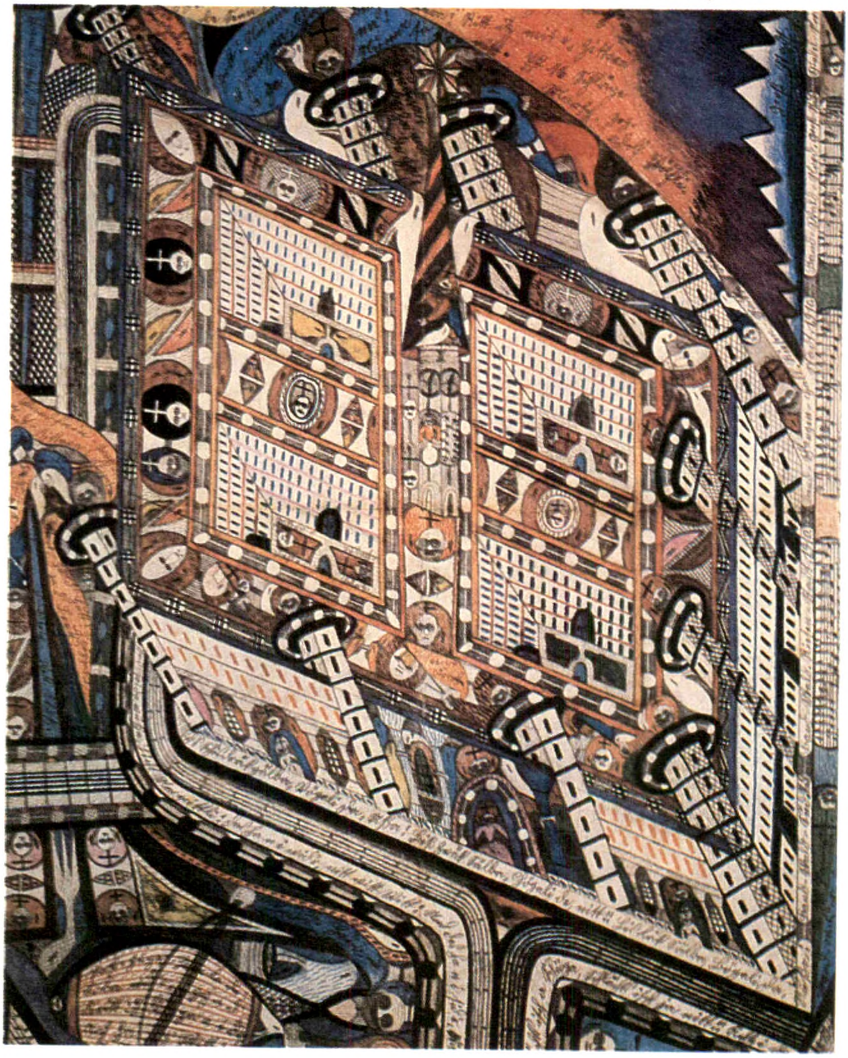

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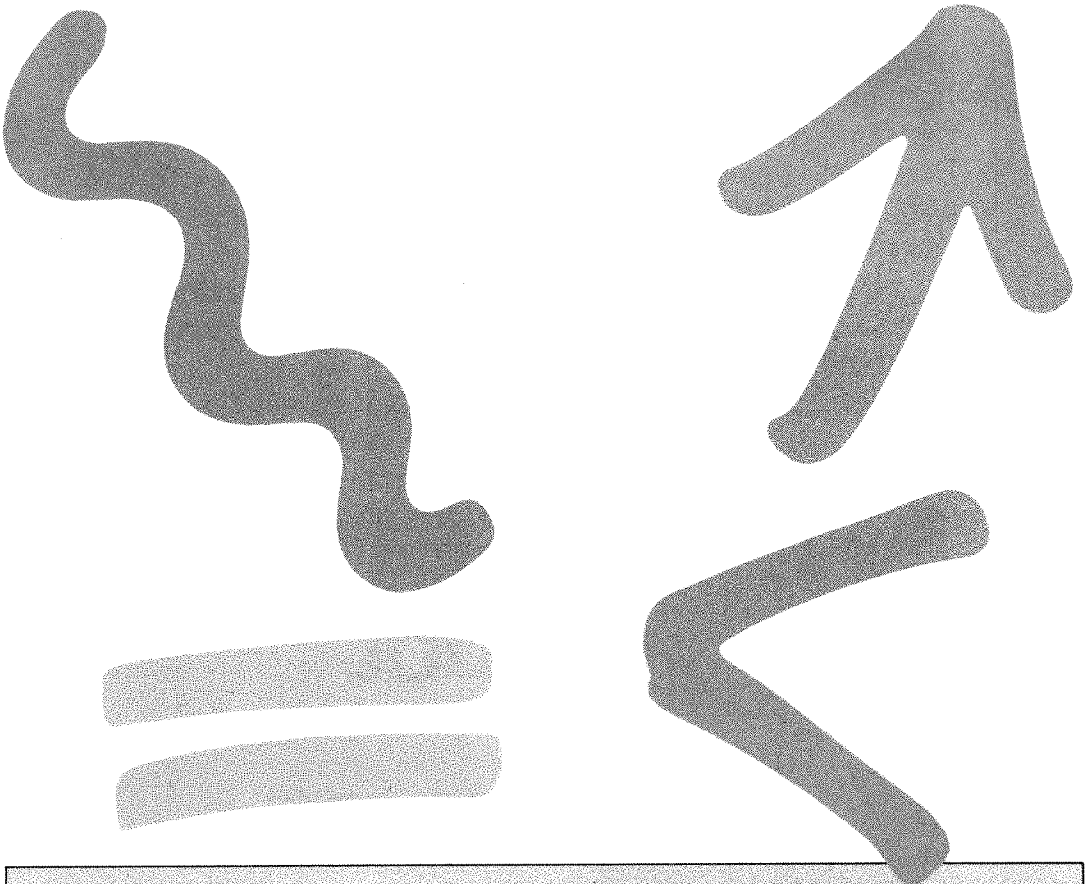

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REFERENCES

1. Johnson D A W and Malik N A. Acta Psychiat Scand 1975; 51: 257-267

2. Pinto R, Bannerjee A and Ghosh N. Acta Psychiat Scand 1979; 60: 313-322

3. Hamilton M, Card I R, Wallis C G and Mahmood M R. Psychopharmacol 1979; 64: 225-229

4. Chowdhury M E H and Chacon C. Comp Psychiat 1980; 21: 135-139

5. Haslam M T, Bromham B M and Schiff A A. Acta Psychiat Scand 1975; 51: 92-100

6. Knights A et al. Brit J Psychiat 1979; 135: 515-523

7. Kelly H B, Freeman H L, Banning B and Schiff A A. Int Pharmacopsychiat 1977; 12: 54-64

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The Mental Health of Elderly Couples

I. The Effects of a Cognitively Impaired Spouse

J. M. EAGLES, J. A. G. BEATTIE, G. W. BLACKWOOD, D. B. RESTALL and G. W. ASHCROFT

In a general practice population, 274 elderly married couples completed the Mental Status Questionnaire (MSQ), the 60-item General Health Questionnaire (GHQ) and the Leeds General Scales for the Self-assessment of Depression and Anxiety. The only relationship detected between cognitive impairment on the MSQ and psychiatric morbidity in the partner was a fairly weak negative correlation between the wives' MSQ and the Leeds Depression score of their husbands. These findings differ from those of previous studies, which have found high rates of psychiatric morbidity in the relatives of demented patients, and the possible reasons for these differences are discussed. Most importantly, other studies have involved the relatives of patients referred to psychiatric services, and these families may be quite different from those in which the demented relative is not referred to a psychiatrist.

In the elderly living at home, the high prevalence rate of dementia is well documented (Kay *et al*, 1964*a*; Parsons, 1965; Bollerup, 1975). The great majority of demented patients are cared for in the community (Kay *et al*, 1964*a*) a situation which is likely to continue and one which will inevitably lead to the main responsibility for their care continuing to fall upon the families of such patients.

It is well recognised that the viability of family support systems is a major determinant of whether or not a demented old person remains in the community (Bergmann *et al*, 1978; Greene & Timbury, 1979). Changes in the age structure of the population are altering the character of such family support systems in that the supporters themselves are tending to become increasingly elderly (Isaacs *et al*, 1972), and it is likely that the more elderly supporters will be those for whom coping with a demented relative will be most difficult. It is thus important to study these elderly supporters in the hope of establishing how their ability to cope might be optimised, for example by attempting to enhance their informal social support network (Zarit *et al*, 1980).

Studies of the stresses involved in caring for an elderly psychiatrically disturbed patient have found these to be considerable (Grad & Sainsbury, 1965; Hoenig & Hamilton, 1969; Gilleard *et al*, 1984). These studies, however, have examined the families of patients referred to psychiatric services, who may be unrepresentative of the families of all elderly people with psychiatric illnesses.

The present study utilised the data from a screening investigation of all those aged over 65 in a general practice population. It was thus possible to examine the psychological well-being of the spouses of an unselected group of cognitively impaired old couples.

Method

The study was conducted in a general practice in Aberdeenshire. The practice has about 14 000 patients of whom about half live in the town in which the practice is centred. The other patients live in smaller towns, in villages, and in the country nearby.

All patients registered with the general practice who were aged 65 and over on the 1 April 1981 constituted the base population for the survey. They numbered 1794 in total. Of these, 16 were unable or unwilling to participate, leaving a total of 1778 subjects who completed the assessment (99.1% of the initial cohort). The subjects were visited by a research worker at their place of residence between August 1980 and April 1982. Interviews were conducted on 1528 of the subjects by a district nurse in the practice and the remaining 250 assessments were carried out by one of two senior medical students. After division of the practice into 13 geographically smaller areas, the subjects were divided by sex and then visited in random order. This was done to avoid the possible 'contaminating' effect of interviewing one neighbour or relative immediately after the other.

At the time of interview the living group of the subject was ascertained in terms of whether they lived alone, with their spouse, or with other relatives. In order to identify all the opposite sexed 'pairs' who lived together and who were both included in the survey population, the lists of subjects visited were scrutinised for males and females of the same name living at the same address. Where households were identified in which there were three or more people of the same name, these households were considered not to contain any 'pairs' for inclusion in the study. The remaining 'pairs' were cross-checked with the information on their living group, and included only if each member of the pair was recorded as living with a spouse. Of the 1778 subjects assessed, 548 were established by this method as living as one of a married couple, of whom both were over 65 and both had been assessed in the course of the survey.

In the context of a comprehensive screening interview, other components of which have been described elsewhere (Restall, 1984), all subjects were tested for cognitive impairment with the Mental Status Questionnaire (MSQ). This is the British version of a questionnaire first developed by Kahn *et al* (1960), consisting of ten questions testing memory and orientation. The MSQ score has been shown by Wilson & Brass (1973) to correlate quite highly ($r = 0.82$) with an independent rating of the presence and degree of dementia in elderly patients. In the study of Wilson & Brass (1973) the mean score of patients considered to have 'mild dementia' was 7.84, and one may thus consider a score of 8 or less to indicate the likely presence of dementia.

Subjects also completed the 60-item General Health Questionnaire (GHQ) (Goldberg, 1972, 1978). The GHQ has been widely used as a screening tool for detecting psychiatric morbidity in the community. It is also of use in comparing different populations, by employing either the proportions of high scorers or differences in mean scores (Goldberg, 1978; Tarnopolsky *et al*, 1979). Of the 548 subjects constituting one of a married couple, 546 completed the GHQ successfully. The other two subjects were considered by the interviewer to be too demented, even with assistance, to complete it satisfactorily.

In the first 300 of the screening interviews, mood was self-rated by visual analogue scale, but many of the elderly subjects had difficulty in grasping the concept of this scale. It was thus abandoned and the subsequent interviewees completed the Leeds General Scales for the Self-assessment of Depression and Anxiety (Snaith *et al*, 1976). These scales constitute a development of the Wakefield Self-assessment of Depression Inventory (Snaith *et al*, 1971), and have been shown to correlate highly with observer rated depression and anxiety (Snaith *et al*, 1976). A total of 453 of the 548 spouses completed both of these scales.

Three methods were adopted to investigate the relationship between cognitive impairment in one member of a couple and the psychological well-being of the other.

(1) Correlation coefficients (Kendall's τ) were calculated between the MSQ scores for the subjects of each sex and the scores on the GHQ and Leeds scales of their spouse.

(2) Those subjects scoring 8 or less on the MSQ were identified, such subjects constituting a 'demented' group. They were matched with a 'non-demented' group who scored 9 or 10 on the MSQ. The matching was done by a rater who was blind to the purpose of the study. She identified 'non-demented' controls matched for age and sex with each 'demented' subject and then identified the spouse of these 'non-demented' controls who was most closely matched in age with the spouse of the 'demented' subject. A married couple, of whom neither was demented, was thus identified as being the most accurate match of a couple in whom one of the partners was demented. Comparisons of the scores on the GHQ and Leeds scales of the spouses of the 'demented' and 'non-demented' subjects in these groups should thus provide accurate information about the effects of living with a demented individual.

(3) Subjects were divided into those scoring 8 or less on the MSQ ('demented group') and those scoring 9 or 10 on the MSQ. The frequency of 'caseness' on the GHQ and Leeds scales was then compared between the spouses of 'demented' subjects and the spouses in all the couples in which neither spouse was demented. 'Demented' subjects themselves, although they lived with a non-demented spouse, were excluded from this analysis since dementia may contaminate responses to the GHQ and Leeds scales.

Results

The characteristics of the total cohort's scores on the MSQ, GHQ and Leeds scales were as follows:

Mental Status Questionnaire

All 548 subjects completed the MSQ. Their mean score was 9.65 (s.d. 0.82). Thirty-one subjects (5.7% of the total sample) scored 8 or less, these subjects constituting the 'demented' group.

General Health Questionnaire (60-item)

This was completed by 546 of the subjects. Their mean score was 3.46 (s.d. 6.41). It is usual to take a health/illness cut-off score of 11/12 in the 60-item GHQ, and 51 subjects (9.3%) scored 12 or more.

Leeds General Depression Scale

This was completed by 453 of the subjects. Their mean score was 2.09 (s.d. 2.64). The advised health/illness cut-off score is 6/7 (Snaith *et al*, 1976) and 37 subjects (8.2%) scored 7 or more.

Leeds General Anxiety Scale

This was completed by 453 of the subjects. Their mean score was 1.83 (s.d. 2.81). The advised health/illness cut-off score is again 6/7, and 44 subjects (9.8%) scored 7 or more.

The mean age of all 548 subjects was 73.0 (s.d. 5.1). The mean age of the 31 'demented' subjects was 76.7 (s.d. 7.3). In comparison with the 'non-demented' subjects by means of a Mann-Whitney U Test, the demented subjects were, predictably, significantly older ($z = 2.81$, $P < 0.005$). Of the 31 'demented' subjects, 20 were male and 11 were female.

The correlation coefficients between the MSQ scores of the subjects of each sex and the spouses' GHQ and Leeds Scales scores are shown in Table I. The number of pairs involved in each analysis is shown in brackets.

If cognitive impairment were associated with psychological disturbance in the subject's living partner, a significant negative correlation between the MSQ scores and the partners' scores on the GHQ and Leeds Scales would have been expected. As Table I shows, this

TABLE I

Correlation coefficients (Kendall's τ) between scores on Mental Status Questionnaire and spouses' scores on General Health Questionnaire, Leeds Depression and Leeds Anxiety Scales

	Spouse's GHQ	*Spouse's Leeds Depression*	*Spouse's Leeds Anxiety*
Male MSQ	−0.032 (274 pairs)	0.042 (227 pairs)	0.083 (227 pairs)
Female MSQ	−0.004 (272 pairs)	−0.129* (224 pairs)	−0.051 (224 pairs)

*$P<0.02$

relationship was detected only between the wives' MSQ and their husbands' scores on the Leeds Depression scale.

The scores on the GHQ and Leeds Scales of the spouses of the 31 'demented' subjects (MSQ 8 or less) and the spouses of the matched 'non-demented' subjects (MSQ 9 or 10) are shown in Table II.

TABLE II

Scores of the spouses of 'demented' and 'non-demented' subjects on the 60-item GHQ and Leeds Scales

	Mean score on GHQ (31 sets of paired data)	*Mean score on Leeds Depression Scale (28 sets of paired data)*	*Mean score on Leeds Anxiety Scale (28 sets of paired data)*
Spouses of demented subjects	3.13 (s.d. 4.51)	2.29 (s.d. 2.62)	1.50 (s.d. 1.67)
Spouses of non-demented subjects	4.48 (s.d. 6.34)	2.04 (s.d. 2.30)	1.57 (s.d. 2.24)

The spouses of the demented subjects thus show a slightly higher mean score on the Leeds Depression Scale, while the spouses of the *non-demented* subjects have higher scores on the GHQ and Leeds Anxiety Scale. Comparison of the scores of the two groups of partners with the Wilcoxon matched-pairs signed-ranks test showed no significant differences for their scores on any of the three scales.

The frequency of 'caseness' on the three scales was compared in the spouses of the 31 'demented' subjects against all the spouses of non-demented subjects, excluding the 31 'demented' subjects themselves. 'Cases' were defined as scoring 12 or more on the GHQ, and as scoring 7 or more on the Leeds Scales. The frequency of 'cases' in the two groups is shown in Table III.

Analysis of differences in the rates of 'caseness' between the two groups, by means of χ^2 tests, found there to be no significant differences on any of the three scales.

TABLE III

Frequency of 'cases' in the partners of demented subjects and in all partners of non-demented subjects

	Cases on GHQ	*Cases on Leeds Depression Scale*	*Cases on Leeds Anxiety Scale*
Spouses of demented subjects	3/31 (9.7%)	3/28 (10.7%)	0/28 (0%)
Spouses of non-demented subjects	49/574 (8.5%)	35/476 (7.4%)	45/476 (9.5%)

Discussion

Data from 274 elderly married couples were analysed to investigate whether a relationship existed between cognitive impairment in one partner and psychological distress or disturbance in the other. Three methods of analysis were employed, but the only such relationship detected was a relationship between wives' impairment on the MSQ and husbands' scores on the Leeds Depression Scale.

The scale of this relationship is surprisingly small, not only at a 'common sense' level, but also in comparison with previous studies in the same area. The study of Gilleard *et al* (1984) investigated the supporters of over 200 elderly patients who attended geriatric or psychogeriatric day hospitals in the Edinburgh area, the majority of these patients being demented. Of the supporters, 67% scored as 'cases' on the 30-item GHQ. Using a global rating scale, Grad & Sainsbury (1965) considered 40% of the families of elderly patients referred to psychiatric services in Chichester and Salisbury to be suffering 'severe burden'. Other authors, without feeling the need for formal testing, have commented on the high levels of psychological distress in such families (e.g. Bergmann *et al*, 1978; Greene & Timbury, 1979). The only study which appears to support the present findings is that of Gilhooly (1984). She studied 37 supporters of demented attenders at Aberdeen day hospitals and found that the majority of supporters were well or only 'mildly mentally impaired'.

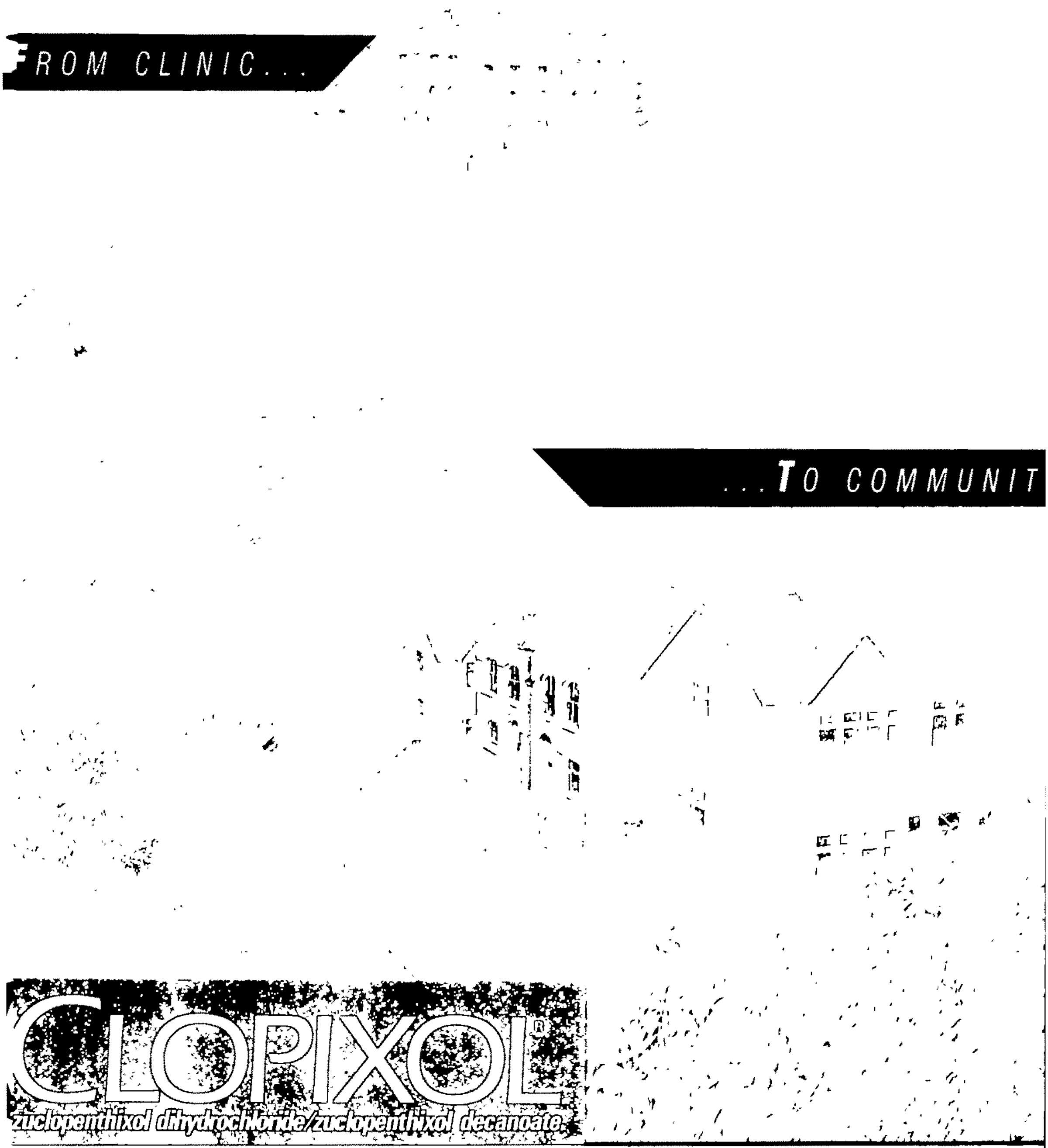
All the studies quoted above, however, share the disadvantage of their being based on the families of elderly patients who have been referred to psychiatric services. Such a referral from the general practitioner may often arise, indeed it may usually arise, not on account of the patient's dementia, but on account of the stresses and psychological disturbances arising in the family as a result of this dementia. Clearly then, studies on the levels of psychological disturbance in such a population of supporters must take into account the fact that the patients may have been 'selected' on account of that self-same disturbance in the relatives.

A weakness of the present study is the diagnosis of 'dementia' on the basis of the Mental Status Questionnaire. The illness/health cut-off point of 8/9 will undoubtedly lead to false negative and false positive classifications. The study of Wilson & Brass (1973), however, showed that a cut-off score of 8/9 would result in more false negatives than false positives. Also, the overall prevalence of dementia with this cut-off point (5.7%) is lower than most estimates of the community prevalence of dementia (e.g. Nielsen, 1962; Kay *et al*, 1964*a*; Williamson *et al*, 1964; Hagnell, 1970), suggesting that a relatively conservative number of 'demented' individuals may have been so classified. However, as well as including a small number of normal old people in the 'demented' group, this will include a relatively large number of individuals with dementia of only mild severity. It may be that supporters develop psychological distress only when their dependant becomes more severely demented, and that this factor may explain the discrepancy between the present study and those quoted above. With this possibility in mind, we compared the partners of patients scoring 6 or less on the MSQ ($n = 10$) with their matched control partners. This comparison was repeated for the partners of the 15 patients scoring 7 or less on the MSQ, this being the point on the 'American MSQ' at which Reisberg *et al* (1982) consider patients usually suffer from 'moderate cognitive decline'. While the groups were admittedly very small, these comparisons between the partners of more severely demented and of normal subjects did not reveal any significant differences in scores on the GHQ and Leeds Scales. Zarit *et al* (1980) also found no relationship between the severity of dementia and the psychological well-being of the supporter.

In diagnosing 'dementia' as we did in the present study, the MSQ has the further disadvantage of measuring only memory and orientation. Other aspects of cognitive decline are not measured and of course there is no account taken of changes in behaviour and personality. While simple decline in memory (as measured by the MSQ) may be quite easily tolerated by relatives, behavioural deterioration is much more stressful (Sanford, 1975; Gilleard *et al*, 1982). Thus, the present study's 'demented' group may be significantly less disturbed behaviourally than those who are referred to psychiatric services and who have comprised the subjects for previous studies on supporters' psychological well-being.

It is also possible that the differences between the findings of this study and those of others arise as a result of special characteristics of the area and the people investigated. The present study found that only 9.3% of the 548 subjects constituted a 'case' on the 60-item GHQ, this rate of 'caseness' being considerably lower than the rate of 30% found in a community sample of women in Dundee by Hobbs *et al* (1983) or the 25% found by Benjamin *et al* (1982) in Manchester. Both of these studies were of younger populations, and the lower rates in our sample may reflect the fact that neuroses become less common with increasing age (Kay, 1976; Post, 1982). That being said, the rates of 'caseness' in the present study are lower than those for neurotic illness found by the community studies of the elderly in Newcastle (Kay *et al*, 1964*a*; Bergmann, 1971). These discrepancies may be partly accounted for by semi-rural/urban differences, and there is certainly evidence that younger patients in rural communities tend to be psychologically healthier than their urban counterparts (Brown & Harris, 1978; Dilling & Weyerer, 1984). The particular area studied is also of low geographical mobility, high community cohesiveness and has a high standard of primary medical care. These factors may predispose to a generally low level of psychiatric morbidity and an increased resilience to stress, notably to the stress of caring for an elderly demented relative. It is possible that the primary care team is identifying stressed relatives at an early stage and is invoking supportive and/or pharmacological measures to alleviate the stress and to prevent or treat overt psychiatric morbidity.

Our results may reflect the fact that the GHQ and Leeds scales are unsuitable screening instruments when seeking to detect psychiatric morbidity in an elderly population. The Leeds scales have not, to the authors' knowledge, been validated in community samples, but the GHQ has been on several occasions (e.g. Goldberg & Blackwell, 1970; Tarnopolsky *et al*, 1979; Benjamin *et al*, 1982; Hobbs *et al*, 1983). While doubts have been expressed about the GHQ's sensitivity as a screening instrument for psychiatric morbidity (Benjamin *et al*, 1982), especially in subjects under chronic stress (Finlay-Jones & Murphy, 1979), it has been validated satisfactorily against psychiatric interview in the supporters of elderly day hospital attenders (Gilleard, 1984; Gilleard *et al*, 1984). However, validation of the



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GHQ in a community sample of elderly people has not yet been carried out.

The present study found a significant association between depression in husbands and cognitive impairment in the wives, but the reverse relationship did not apply. This finding is at odds with other studies (Gilhooly, 1984; Gilleard *et al*, 1984) which found higher levels of distress in females than in male supporters. The supporters in these studies differed from our supporters by being younger, by less often being spouses and by being in contact with psychiatric services. Direct comparisons are thus impossible, and one can hypothesise that these differences may be due to factors relating to age, to the relationship of the supporter to the demented dependant, to sex differences in 'help-seeking behaviour' or to sex differences in benefitting from contact with psychiatric services.

The finding that the partners of demented old people in the community do not, by and large, exhibit overt psychological disturbance should not be taken as an indication that they are free from burden and strain. Indeed, the results of a further study in the same general practice indicate that these families are under considerable strain, even although this is not manifested by overt psychiatric disturbance (Eagles *et al*, 1987).

The principal aim of research into the supporters of the demented elderly is to optimise, and possibly to prolong, these patients' care in the community. In maintaining an elderly demented patient at home one generally seeks to prevent the family from reaching the irreversible stage of 'total rejection' described by MacMillan (1960). How and whether this stage can be avoided is not yet known, and early intervention and support has been sensibly advocated as a means whereby it might be (Kay *et al*, 1970; Godber, 1977; Bergmann *et al*, 1978). Whether or not such support is effective, and whether it should be provided to all supporters, to supporters experiencing significant burden and strain, or only to supporters with overt psychiatric disturbance are all questions requiring further research.

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The Mental Health of Elderly Couples

II. Concordance for Psychiatric Morbidity in Spouses

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A community sample of elderly married couples completed the 60-Item General Health Questionnaire and the Leeds General Scales for the Self-Assessment of Depression and Anxiety. Significant concordance was demonstrated between the spouses' scores on these scales. Concordance was higher for depression than for anxiety. There was little to support previous findings that wives are more likely than husbands to be concordant with an ill spouse. The spouse concordance rates for psychiatric morbidity were similar to those found in studies of younger married couples.

Although almost 50% of the population over the age of 65 live as one of a married couple (Registrar General Scotland, 1982a) little has been written about the relationship between marriage and psychiatric illness in the elderly. At an epidemiological level, it is well documented that the married elderly have a lower risk both of referral to psychiatric services and of being institutionalised with psychiatric illness (Sainsbury *et al*, 1965; Townsend & Wedderburn, 1965; Turner & Sternberg, 1978). However this may well reflect the benefits of social support in general, rather than a specific protective effect of marriage *per se*. While community surveys have found higher rates of functional psychiatric disorders in the elderly who are socially isolated (Kay *et al*, 1964*b*; Bergmann, 1971; Gruer, 1975), the reason for this relationship has not been established. Indeed, it is most probable that lifelong personality traits predispose towards both isolation and the development of neurotic illness in old age (Kay *et al*, 1964*b*; Bergmann, 1971). The epidemiological evidence is thus equivocal regarding the possible influence of marriage *per se* on the mental health of the elderly.

The relative lack of attention given to marital factors in geriatric psychiatry may be justified in that such factors may be very similar to those encountered, and researched, in younger patients. Two sources of evidence, however, suggest that the findings from younger couples are not generalisable and that older couples require to be studied in their own right.

Divorce statistics show that the longer one is married, the less likely one is to divorce (Crown, 1976). In 1981, 48% of all divorces in Scotland took place in those married for less than 10 years, while the corresponding figure for those married over 30 years was 5.5% (Registrar General Scotland, 1982*a*). One might infer from these figures that marriages become progressively stable as the duration of marriage lengthens.

On the other hand research conducted on younger married couples has produced considerable evidence that the concordance for neurotic disorder in spouses exceeds the rate that would be found by chance (Gregory, 1959; Kreitman, 1962; Nielsen, 1964; Merikangas, 1982), and this concordance rate has been shown to increase with increasing duration of marriage both for neurotic illness (Buck & Ladd, 1965; Ovenstone, 1973; Hagnell & Kreitman, 1974) and for neuroticism as measured on the Maudsley Personality Inventory (Kreitman, 1964; Hare & Shaw, 1965). While this relationship has not always been found (Pond *et al*, 1963; Agulnik, 1970), 'received wisdom' has it that 'the longer a marriage continues the more likely it is for both rather than one partner to be neurotic' (Freeman, 1983). If concordance does increase as a function of marital duration, it would follow that the marriages of the elderly would become increasingly polarised into partnerships of two stable or of two very unstable individuals. It is difficult to reconcile this picture with the low divorce rates in the elderly, when one considers the known association between marital disharmony and neurosis (Birtchnell & Kennard, 1983*a*,*b*) and between divorce and mental illness (Briscoe *et al*, 1973; Robertson, 1974; Merikangas, 1984).

Therefore, while the relationship between marriage and mental illness in the elderly has been the subject of little research, such evidence as there is points to conflicting conclusions. The main aim of the present study was to obtain further information on this relationship by using data from the screening investigation of elderly subjects described in the first part of this paper.

Method

Details of the screening investigation carried out on those aged 65 and over in a general practice population are given in the first part of this paper.

The subjects studied in this part of the paper comprised only the 274 married couples. Of the 548 spouses, 546 completed the 60-item General Health Questionnaire and 453 completed the Leeds General Scales for the Self-assessment of Depression and Anxiety.

Two methods were adopted to investigate the pattern of psychological disturbance in married couples. Firstly, within pairs correlation coefficients (Kendall's τ) between male and female scores on the GHQ and Leeds Scales were calculated, these data representing 'score concordance' between spouses. Secondly, 'case concordance' between spouses was examined. Data were analysed separately for males and females. A 'case' was defined as scoring above the cut-off point (described in part I of this paper) on any of the three scales i.e. 'GHQ cases', 'Leeds Depression cases' and 'Leeds Anxiety cases' were identified and analysed separately. 'Case concordance' was defined simply in terms of the spouse of a 'case' scoring above the cut-off point *on any one or more* of the three scales—the sample size did not justify analysing the six scale permutations separately.

Results

The total cohort's mean age and their scores on the GHQ and Leeds Scales are shown in the first part of this paper, and thus they will not be presented again.

The within pairs correlation coefficients between the spouses' scores on the GHQ and Leeds Scales are shown in Table I. The number of husband–wife pairs involved in each analysis is shown in brackets.

Four of the nine correlation coefficients are statistically significant. All correlations are positive, indicating a degree

TABLE I
Correlation coefficients (Kendall's τ) between partners' scores on the GHQ and Leeds Scales

	Husband GHQ	*Husband Leeds Depression*	*Husband Leeds Anxiety*
Wife GHQ	0.070 (272 pairs)	0.108** (224 pairs)	0.134** (224 pairs)
Wife Leeds Depression	0.089* (227 pairs)	0.155*** (218 pairs)	0.075 (218 pairs)
Wife Leeds Anxiety	0.005 (227 pairs)	0.072 (218 pairs)	0.090 (218 pairs)

***$P<0.005$, **$P<0.01$, *$P<0.05$

TABLE II
Spouse concordance with male cases

	GHQ—Husband		*Leeds Depression —Husband*		*Leeds Anxiety —Husband*	
	Cases	*Non-cases*	*Cases*	*Non-cases*	*Cases*	*Non-cases*
Number	21	251	17	207	12	212
Wife concordance	6/21 (28.6%)	41/251 (16.3%)	6/17 (35.3%)	33/207 (15.9%)	3/12 (25.0%)	36/212 (17.0%)
Relative risk of wife concordance	1.75		2.22		1.47	

of concordance for minor psychiatric morbidity. The highest coefficient is between partners' scores on the Leeds General Depression Scale, ($r=0.16$; $P<0.005$).

Tables II and III show the rate of caseness of husband and of wives on each of the three scales. The frequency of spouse concordance is shown in the row below. The bottom rows of these tables show the 'relative risk of spouse concordance', which is derived from the ratio: rate of concordance in spouses of cases/rate of concordance in the spouses of non-cases.

Concordance in the spouses of cases on the three scales is consistently higher than in the spouses of non-cases. However, only for the wives of male Leeds Depression cases and of Leeds Depression non-cases does this difference reach statistical significance ($\chi^2=4.09$, d.f. 1, $P<0.05$).

The 'relative risk of spouse concordance' is consistently higher for women than for men, suggesting that women who have an ill husband may be more at risk of being ill than are men who have an ill wife. However, direct comparisons between husbands' and wives' concordance rates with spouse 'cases' were made using χ^2. For example, 21 wives and 30 husbands had spouses who were 'GHQ cases': spouse concordance was shown by six of the 21 wives and by four of the 30 husbands. These rates of spouse concordance are not statistically significantly different, nor are those of husbands and wives whose spouses are 'cases' on either of the Leeds Scales. There is therefore no statistical evidence to suggest that wives are more likely than husbands to be ill when they have an affected spouse, although the numbers in the analyses are relatively small despite the size of the initial screen.

Discussion

The results provide some support for concordance for minor psychiatric morbidity in elderly spouses. Statistically significant findings were obtained for 'score concordance' on four of the nine combinations of the three scales used. On the other hand, the results failed to provide statistically significant evidence for 'case concordance', although this may be due to the relatively small number of cases identified despite the size of the initial screen. As Tables II and III show, the 'relative risk of spouse concordance' does exceed that to be expected by chance (i.e. exceeds 1) on all three scales for both sexes.

The results of this study are to a large extent dependent upon the validity of the GHQ and Leeds Scales as screening tests for psychiatric morbidity in elderly subjects. Some of the difficulties relating to the use of these instruments, notably the GHQ, are mentioned in Part I of this paper and will not be repeated here.

Other studies of psychiatric morbidity in elderly spouses do not appear to have been undertaken. This may be partly because, as Dominian (1972) has pointed out, increased life expectancy has only recently lead to a relative proliferation of elderly married couples. However the present findings are consistent with those of Fengler & Goodrich (1979) who showed that when elderly men became ill, their 'morale' was interdependent with that of their wives.

Studies of younger married couples have consistently found higher concordance for mental illness than would be expected by chance, and those studies have been the subject of a full review by Merikangas (1982). Both she and Nielsen (1964) have summarised the main hypotheses as to why this should occur, and

TABLE III
Spouse concordance with female cases

	GHQ—Wife		*Leeds Depression — Wife*		*Leeds Anxiety — Wife*	
	Cases	*Non-cases*	*Cases*	*Non-cases*	*Cases*	*Non-cases*
Number	30	244	20	207	32	195
Husband concordance	4/30 (13.3%)	24/244 (9.8%)	4/20 (20.0%)	23/207 (11.1%)	4/32 (12.5%)	23/195 (11.8%)
Relative risk of husband concordance	1.36		1.80		1.06	

four of these hypotheses can be usefully considered in the context of the present study (1) that illness in one partner represents a reaction to illness in the other; (2) that partners may 'infect' each other with abnormal reactions; (3) that individuals with similar constitutions tend to marry one another ('assortative mating'); (4) that after referral for the illness of the first partner, identification of illness in the spouse is facilitated.

With regard to the present study, hypothesis (4) can be discounted since morbidity was not determined by referral to psychiatric services. Hypotheses (1) and (2) both refer to the relationship between husband and wife as constituting the important causative factor, and they may thus be collectively labelled the 'interaction hypothesis'. It is this hypothesis and that of 'assortative mating' which have been most forcefully advocated as determining the raised spouse concordance rates. Merikangas (1982) believes that assortative mating for personality traits (such as 'neuroticism') is sufficient to explain spouse concordance for all types of psychiatric disorder. Kreitman (1968*a*,*b*), on the other hand, concluded that while the findings in the functional psychoses were compatible with an assortative mating hypothesis, the interaction hypothesis provided a better explanation for the patterns observed in the neuroses, notably the pattern of increasing illness concordance with increasing duration of marriage. It is thus instructive to compare the present study's concordance rates with those from studies of younger patients.

The correlations of 'score concordance', as shown in Table I, are not high, the highest correlation (between spouses on the Leeds General Depression Scale) having a Kendall's τ of 0.16, ($P < 0.005$). The data on 'case concordance' (shown in Tables II and III) can be more readily compared with the findings in younger married couples, since it is this approach which has usually been adopted in such studies. The mean of the figures of the 'relative risk of spouse concordance' is 1.6, indicating that, if one has an ill spouse, then one has 1.6 times the risk of being ill than if one's spouse were well. Although there are difficulties in making comparisons with other studies ('cases' are usually defined through contacts with psychiatric services, for example), the figure of 1.6 is strikingly similar to the figures of 'morbidity risk' found in the spouses of younger psychiatric patients, where 'morbidity risk' indicates the ratio of the number of observed to the number of expected cases. The studies of Kreitman (1962) and Negri *et al* (1981) both derived a figure of 1.5 times that expected, while Nielsen's (1964) figure was 1.4. Gregory (1959), studying only psychiatric inpatients, found nine times more concordant spouses than expected by chance. Thus, the figure of 1.6 derived from the present study does *not* support the hypothesis that concordance for psychiatric morbidity steadily escalates into old age with increasing duration of marriage.

There are four main reasons why concordance for psychiatric morbidity in elderly spouses may not differ from that found in younger married couples. Firstly, the relationship between concordance and duration of marriage in younger patients may be an artifact of the methodology used in these studies (Merikangas, 1982). Secondly, marriages of neurotic individuals may be less likely to continue into old age. This could occur due to the increased mortality rates amongst those with neurotic illnesses (Sims, 1978), or because divorce is more common in those who are psychiatrically ill (Robertson, 1974; Merikangas, 1984) and in those who have higher levels of neuroticism on the Eysenck Personality Inventory (Thornes & Collard, 1979). Thirdly, the prevalence of neurotic illness in later life probably declines with increasing age (Kay, 1976; Nilsson & Persson, 1984). Fourthly, older people become more introverted as they age (Nilsson, 1983). This is associated with neurotic illness becoming more inwardly directed so that illnesses which 'forcefully impinge on others' are less common (Post, 1982).

This study does not bear out the findings of the studies of Kreitman (1964) and of Hagnell and Kreitman (1974), who found that wives are more likely than husbands to be concordant with an ill spouse. Partial support does derive from the finding that the 'relative risk of spouse concordance' was consistently higher in wives than in husbands with an ill spouse. However, women are about twice as likely as men to suffer from minor affective disorder (Weissman & Klerman, 1979), a fact confirmed by the present study. It might thus be expected that, given the stress of an ill spouse, a wife would be twice as likely as a husband to become psychiatrically ill. In this light, it might be considered surprising that wives were *not* shown to have significantly higher concordance with an ill spouse.

'Score concordance' between spouses was demonstrated for the Leeds General Depression Scale but not for the Leeds General Anxiety Scale. Adopting an 'interaction hypothesis' model this might be explained on the basis that it is less stressful to have an anxious rather than a depressed spouse, or that one is more likely to respond with depression to illness in one's spouse, than with anxiety. On the other hand, it is possible that assortative mating is more likely between spouses predisposed to developing depression than between those predisposed to becoming pathologically anxious.

Divorce occurs predominantly in the early years of marriage (Crown, 1976), and most of the current marriages between elderly couples did not have the 'advantage' of more liberal divorce laws at a time in the marriage when this 'advantage' was likely to have been utilised. Many marriages of elderly people thus presumably 'weathered the storm' without recourse to the divorce which might nowadays be sought. Studies of such marriages might provide useful background information for those working in the fields of marital therapy and counselling.

With increasing longevity, marriages lasting into old age will almost certainly become increasingly common. The continuing accent on community care for the elderly will thus require enhanced understanding of marital issues for the practice of geriatric psychiatry. Thus, to foster understanding of the marriages of younger people and to optimise the care of elderly patients, further research into the marriages of elderly couples should be given a high priority.

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References

Agulnik, T. (1970) The spouse of the phobic patient. *British Journal of Psychiatry*, **117**, 59–67.

Benjamin, S., Decalmer, P. & Haran, D. (1982) Community screening for mental illness: a validity study of the General Health Questionnaire. *British Journal of Psychiatry*, **140**, 174–180.

Bergmann, K. (1971) The neuroses of old age. In *Recent Developments in Psychogeriatrics* (eds. D. W. K. Kay & A. Walk). Kent: Headley Brothers.

——, Foster, E. M., Justice, A. W. & Matthews, V. (1978) Management of the demented elderly patient in the community. *British Journal of Psychiatry*, **132**, 441–449.

Birtchnell, J. & Kennard, J. (1983*a*) Marriage and mental illness. *British Journal of Psychiatry*, **142**, 193–198.

—— & —— (1983*b*) Does marital maladjustment lead to mental illness? *Social Psychiatry*, **18**, 79–88.

Bollerup, T. R. (1975) Prevalence of mental illness among 70 year olds domiciled in nine Copenhagen suburbs. *Acta Psychiatrica Scandinavica*, **51**, 327–339.

Briscoe, C. W., Smith, J. B., Robbins, E., Marten, S. & Gaskin, F. (1973) Divorce and psychiatric disease. *Archives of General Psychiatry*, **29**, 119–125.

Brown, G. W. & Harris, T. (1978) *Social Origins of Depression: A Study of Psychiatric Disorder in Women*. Cambridge: Tavistock.

Buck, C. W. & Ladd, K. L. (1965) Psychoneurosis in marital partners. *British Journal of Psychiatry*, **111**, 587–590.

Crown, S. (1976) Marital breakdown; epidemiology and psychotherapy. In *Recent Advances in Clinical Psychiatry, 2* (ed. K. Granville-Grossman). Edinburgh: Churchill Livingstone.

Dilling, H. & Weyerer, S. (1984) Prevalence of mental disorder in the small-town-rural region of Traunstein (Upper Bavaria). *Acta Psychiatrica Scandinavica*, **69**, 60–79.

Dominian, J. (1972) Marital pathology: a review. *Postgraduate Medical Journal*, **48**, 517–525.

Eagles, J. M., Craig, A., Rawlinson, F., Restall, D. B., Beattie, J. A. G. & Besson, J. A. O. (1987) The psychological well-being of supporters of the demented elderly. *British Journal of Psychiatry*, **150**, 293–298.

Fengler, A. P. & Goodrich, N. (1979) Wives of elderly disabled men; the hidden patient. *Gerontologist*, **18**, 175–183.

Finlay-Jones, R. A. & Murphy, E. (1979) Severity of psychiatric disorder and the 30-item General Health Questionnaire. *British Journal of Psychiatry*, **134**, 609–616.

Freeman, C. P. (1983) Neurotic disorders. In *Companion to Psychiatric Studies*, 3rd edn. (eds R. E. Kendell & A. K. Zealley). Edinburgh: Churchill Livingstone.

Gilhooly, M. L. M. (1984) The impact of care-giving on care-givers: factors associated with psychological well-being of people supporting a dementing relative in the community. *British Journal of Medical Psychology*, **57**, 35–44.

Gilleard, C. J. (1984) Problems posed for supporting relatives of geriatric and psychogeriatric day patients. *Acta Psychiatrica Scandinavica*, **70**, 198–208.

——, Belford, H., Gilleard, E., Whittick, J. E. & Gledhill, K. (1984) Emotional distress amongst the supporters of the elderly mentally infirm. *British Journal of Psychiatry*, **145**, 172–177.

——, Boyd, W. D. & Watt, G. (1982) Problems in caring for the elderly mentally infirm at home. *Archives of Gerontology and Geriatrics*, **1**, 151–158.

Godber, C. (1977) Planning services for the elderly demented patient. *Age and Ageing*, **6** (Suppl), 100–103.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire: Technique for the Identification and Assessment of Non-Psychotic Psychiatric Illness*. London: Oxford University Press.

—— (1978) *Manual of the General Health Questionnaire*. Windsor: NFER Publishing Company.

—— & Blackwell, B. (1970) Psychiatric illness in general practice: a detailed study using a new method of case identification. *British Medical Journal*, **ii**, 439–443.

Grad, J. & Sainsbury, P. (1965) An evaluation of the effects of caring for the aged at home. In *Psychiatric Disorders in the Aged: A Report on the Symposium Held by the World Psychiatric Association*. Manchester: Geigy.

Greene, J. G. & Timbury, G. C. (1979) A geriatric psychiatric day hospital: a five year review. *Age and Ageing*, **8**, 49–53.

Gregory, I. (1959) Husbands and wives admitted to mental hospitals. *Journal of Mental Science*, **105**, 457–462.

Gruer, R. (1975) *Needs of the Elderly in the Scottish Borders*, Scottish Health Service Studies No. 33. Edinburgh: Scottish Home and Health Department.

Hagnell, O. (1970) Disease expectancy and incidence of mental illness among the aged. *Acta Psychiatrica Scandinavica* (Suppl.) **219**, 83–89.

—— & Kreitman, N. (1974) Mental illness in married pairs in a total population. *British Journal of Psychiatry*, **125**, 293–302.

Hare, E. H. & Shaw, G. A. (1965) The patient's spouse and concordance on neuroticism. *British Journal of Psychiatry*, **111**, 102–103.

Hobbs, P., Ballinger, C. B. & Smith, A. H. W. (1983) Factor analysis and validation of the General Health Questionnaire in women: a general practice study. *British Journal of Psychiatry*, **142**, 257–264.

Hoenig, J. & Hamilton, M. W. (1969) *The Desegregation of the Mentally Ill*. London: Routledge & Kegan Paul.

Isaacs, B., Livingstone, M. & Neville, Y. (1972) *Survival of the Unfittest*. London: Routledge & Kegan Paul.

Kahn, R. L., Goldfarb, A. I., Pollock, M. & Peck, A. (1960) Brief objective measures for the determination of mental status in the aged. *American Journal of Psychiatry*, **117**, 326–328.

Kay, D. W. K. (1976) The depressions and neuroses of later life. In *Recent Advances in Clinical Psychiatry*, No. 2 (ed. K. Granville-Grossman). Edinburgh: Churchill Livingstone.

——, Beamish, P. & Roth, M. (1964*a*) Old age mental disorders in Newcastle upon Tyne (Part 1. A study of prevalence). *British Journal of Psychiatry*, **110**, 146–158.

——, Beamish, P. & Roth, M. (1964*b*) Old age mental disorders in Newcastle upon Tyne (Part 2. Study of possible social and medical causes). *British Journal of Psychiatry*, **110**, 668–682.

——, Bergmann, K., Foster, E. M., McKechnie, A. A. & Roth, M. (1970) Mental illness and hospital usage in the elderly: a random sample followed up. *Comprehensive Psychiatry*, **11**, 26–35.

Kreitman, N. (1962) Mental disorder in married couples. *Journal of Mental Science*, **108**, 438–446.

—— (1964) The patient's spouse. *British Journal of Psychiatry*, **110**, 159–173.

—— (1968*a*) Married couples admitted to mental hospital: I. diagnostic similarity and the relation of illness to marriage. *British Journal of Psychiatry*, **114**, 699–708.

—— (1968*b*) Married couples admitted to mental hospital: II. family history, age and duration of marriage. *British Journal of Psychiatry*, **114**, 709–718.

MacMillan, D. (1960) Preventive geriatrics—opportunities of a community mental health service. *Lancet*, **ii**, 1439–1441.

Merikangas, K. R. (1982) Assortative mating for psychiatric disorders and psychological traits. *Archives of General Psychiatry*, **39**, 1173–1180.

—— (1984) Divorce and assortative mating among depressed patients. *American Journal of Psychiatry*, **141**, 74–76.

Negri, F., Melica, A. M., Zuliana, R., Gasperini, M., Macciardi, F. & Smeraldi, E. (1981) Genetic implications in assortative mating of affective disorders. *British Journal of Psychiatry*, **138**, 236–239.

Nielsen, J. (1962) Geronto-psychiatric period prevalence investigations in geographically delimited population. *Acta Psychiatrica Scandinavica*, **38**, 307–330.

—— (1964) Mental disorders in married couples (assortative mating). *British Journal of Psychiatry*, **110**, 683–697.

Nilsson, L. V. (1983) Personality changes in the aged: a transectional and longitudinal study with the Eysenck Personality Inventory. *Acta Psychiatrica Scandinavica*, **68**, 202–211.

—— & Persson, G. (1984) Prevalence of mental disorders in an urban sample examined at 70, 75 and 79 years of age. *Acta Psychiatric Scandinavica*, **69**, 519–527.

Ovenstone, I. M. K. (1973) The development of neuroses in the wives of neurotic men. Part 1, symptomatology and personality. *British Journal of Psychiatry*, **122**, 35–45.

Parsons, T. L. (1965) Mental health of Swansea's old folk. *British Journal of Preventive and Social Medicine*, **19**, 43–47.

Pond, D. A., Ryle, A. & Hamilton, M. (1963) Marriage and neurosis in a working class population. *British Journal of Psychiatry*, **109**, 592–598.

Post, F. (1982) Functional disorders, I: description, incidence and recognition. In *The Psychiatry of Late Life* (eds R. Levy & F. Post). Oxford: Blackwell.

Registrar General Scotland (1982*a*) *Census 1981 Scotland: Report for Grampian Region, Volume 1*. Edinburgh: HMSO.

—— (1982*b*) *Annual Report 1981*. Edinburgh: HMSO.

Reisberg, B., Ferris, S. H., De Leon, M. J. & Crook, T. (1982) The global deterioration scale for assessment of primary degenerative dementia. *American Journal of Psychiatry*, **139**, 1136–1139.

Restall, D. (1984) Troubleshooting for the elderly. *Community View*, **25**, 6–8.

Robertson, N. C. (1974) The relationship between marital status and the risk of psychiatric referral. *British Journal of Psychiatry*, **124**, 191–202.

Sainsbury, P., Costain, W. R. & Grad, J. (1965) The effects of community service on the referral and admission rates of elderly psychiatric patients. In *Psychiatric Disorders in the Aged: A Report on the Symposium Held by the World Psychiatric Association*. Manchester: Geigy.

Sanford, J. R. A. (1975) Tolerance of debility in elderly dependents by supporters at home: its significance for hospital practice. *British Medical Journal*, **iii**, 471–473.

Sims, A. (1978) Hypotheses linking neuroses with premature mortality. *Psychological Medicine*, **8**, 255–263.

Snaith, R. P., Ahmed, S. N., Mehta, F. & Hamilton, M. (1971) The assessment of the severity of primary depressive illness. *Psychological Medicine*, **1**, 143–149.

——, Bridge, G. W. K. & Hamilton, M. (1976) The Leeds scales for the self-assessment of anxiety and depression. *British Journal of Psychiatry*, **128**, 156–165.

Tarnopolsky, A., Hand, D. J., McLean, E. K., Roberts, H. & Wiggins, R. D. (1979) Validity and uses of a screening questionnaire (GHQ) in the community. *British Journal of Psychiatry*, **134**, 508–515.

Thornes, B. & Collard, J. (1979) *Who Divorces?* London: Routledge and Kegan Paul.

Townsend, P. & Wedderburn, D. (1965) *The Aged in the Welfare State*. London: G. Bell and Sons.

Turner, R. J. & Sternberg, M. P. (1978) Psychosocial factors in elderly patients admitted to a psychiatric hospital. *Age and Ageing*, **7**, 171–177.

Weissman, M. M. & Klerman, G. L. (1977) Sex differences in the epidemiology of depression. *Archives of General Psychiatry*, **34**, 98–111.

Williamson, J., Stokoe, I. H., Gray, S., Fisher, M., Smith, A., McGhee, A. & Stephenson, E. (1964) Old people at home: their unreported needs. *Lancet*, **1**, 1117–1120.

Wilson, L. A. & Brass, W. (1973) Brief assessment of the mental state in geriatric domiciliary practice: the usefulness of the Mental Status Questionnaire. *Age and Ageing*, **2**, 92–101.

Zarit, S. H., Reever, K. E. & Bach-Peterson, J. (1980) Relatives of the impaired elderly: correlates of feelings of burden. *Gerontologist*, **20**, 649–655.

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Suicide in England and Wales, 1975–1984

G. M. G. McCLURE

An investigation of the suicide rate in the decade 1975–1984 in England and Wales revealed an increase in rate for males of all ages, and a small decrease for young females. The increase for males has been greatest in the 25–54 years group. There has been a decrease in the number of suicides by poisoning with solids and liquids (including overdoses) for both sexes. Rates for poisoning by vehicle exhaust gas and by hanging, strangulation and suffocation have increased substantially during this period. In males, suicides by these methods now exceed those due to poisoning by solids and liquids.

Suicide rates in England and Wales showed a steady decline in both sexes between 1963 and the early 1970s, due principally to a decrease in suicide in the older age-groups (Adelstein & Mardon, 1975). Rates for males decreased from 145 per million in 1963 to 95 per million in 1974, and rates for females from 99 to 64 per million during the same period.

The most important change in method of suicide during this period was a decrease in poisoning by domestic gas, which was being detoxified at this time. The total number of suicides from poisoning by domestic gas decreased from 2469 in 1962 to 50 in 1974. Suicide by poisoning with solid or liquid substances increased until 1964 and then remained relatively constant. In 1974 it was the most common method of suicide, with 1877 deaths by this means (794 males, 1083 females).

An investigation of suicide statistics between 1975 and 1980 reported that the downward trend in suicide rates had been reversed (McClure, 1984). 'Undetermined' deaths and 'accidental' deaths by causes comparable to suicide were also examined, because an apparent change in the suicide rate could be due to a change in the classification of deaths in these other categories where there was uncertainty whether death was accidentally or purposely inflicted (Sainsbury & Jenkins, 1982). This paper examines statistics for the decade 1975–1984.

Method

Suicide rates (ICD E950–E958) for England and Wales for the period 1975–1984 were obtained from annual publications (Office of Population Censuses and Surveys 1975–1984). In addition, 'undetermined' and 'accidental' deaths in categories comparable to suicide were also investigated (see appendix for comparable categories of cause of death). Changes by different methods and in different age-groups were examined during this period.

Results

Changes in suicide rate for England and Wales: 1975–1984 (all ages)

Changes in the suicide rate in England and Wales during the decade 1975–1984 are shown in Table I and Fig. 1. The suicide rate increased for males in each consecutive year between 1975–1984 from 91 per million to 118 per million, an increase of 30%. The total number of male suicides increased from 2184 to 2859 during this period. For females there was a small increase between 1975 and 1980, followed by a decrease between 1980 and 1984. The suicide rate for females decreased from 60 per million in 1975 to 57 per million in 1984, a decrease of 5%. The total number of female suicides decreased from 1509 to 1456 during the period 1975–1984.

The total of male and female suicides increased from 3693 (75 per million) to 4315 (87 per million) during the period 1975–1984.

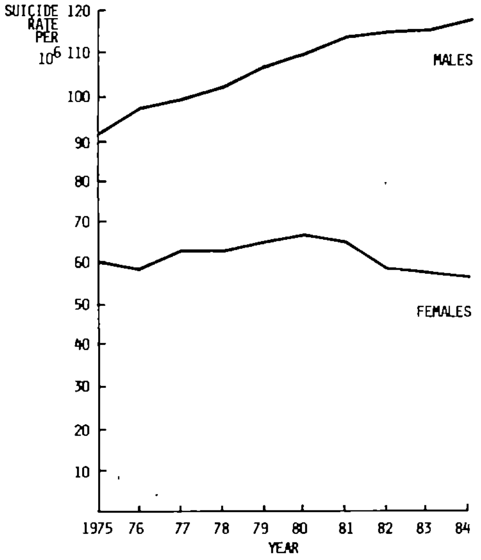

Fig. 1. Suicide rate for England and Wales: 1975–1984 (all ages).

TABLE I

Suicide and self-inflicted injury (ICD E950–E958) in England and Wales (all ages) 1975–1984: total numbers of suicides and rates per million population

		1975	*1976*	*1977*	*1978*	*1979*	*1980*	*1981*	*1982*	*1983*	*1984*
Males	Total	2184	2330	2363	2436	2564	2629	2761	2781	2812	2859
	Rate per 10^6	91	97	99	102	107	110	114	115	116	118
Females	Total	1509	1486	1581	1586	1631	1692	1658	1498	1467	1456
	Rate per 10^6	60	59	63	63	65	67	65	59	58	57

Change in suicide rate by different methods 1975–1984

The changes in suicide rate by different methods for males and females are shown in Table II and Figs 2 and 3. For males, the number of suicides by hanging, strangulation and suffocation (E953) increased from 593 in 1975 (24.7 per million) to 891 in 1984 (36.8 per million). In addition, for males, the number of suicides by poisoning with gases other than domestic gas (E952) increased from 249 in 1975 (10.4 per million) to 773 in 1984 (31.9 per million). The majority of deaths in this category were by motor vehicle exhaust gas (E952.0), comprising 755 of the total 773 deaths for males. There was also a smaller increase in suicide by males using firearms (E955) from a total of 143 in 1975 (6.0 per million) to 205 in 1984 (8.5 per million). For males there was a decrease in the number of suicides by poisoning with solid and liquid substances, including overdoses (E950). The number of males committing suicide by this means decreased from 709 in 1975 (29.6 per million) to 495 in 1984 (20.4 per million).

For females the number of suicides by poisoning with solids and liquids, including overdoses (E950) also decreased during the period under review. The number of female suicides by this method decreased from 969 in 1975 (38.4 per million) to 678 in 1984 (26.6 per million). There was an increase in female suicides by hanging, strangulation and suffocation (E953) from a total of 220 in 1975 (8.7 per million) to 326 in 1984 (12.8 per million). In addition, there was an increase in suicide by gases other than domestic gas (E952) for females, with the number increasing from 27 in 1974 (1.1 per million) to 90 in 1984 (3.5 per million); 84 out of the total of 90 suicides by this method were by motor vehicle exhaust gas (E952.0).

TABLE II

Numbers (and rates per million population) for suicide and self-inflicted injury, classified by different causes, in 1975 and 1984 (all ages)

Cause of death (ICD and classification)		*1975*		*1984*			
		Number	*Rate*	*Number*	*Rate*	*Change in rate 1975–1984*	
E950 Poisoning by solid or liquid substances	Male	709	29.6	495	20.4	− 9.2	−31%
	Female	969	38.4	678	26.6	−11.8	−31%
E951 Poisoning by gases in domestic use	Male	19	0.8	10	0.4	− 0.4	[1]
	Female	4	0.2	1	0.04	− 0.16	[1]
E952 Poisoning by other gases and vapours	Male	249	10.4	773	31.9	+21.5	+207%
	Female	27	1.1	90	3.5	+ 2.4	+218%
E953 Hanging, strangulation and suffocation	Male	593	24.7	891	36.8	+12.1	+49%
	Female	220	8.7	326	12.8	+ 4.1	+47%
E954 Submersion (drowning)	Male	140	5.8	92	3.8	− 2.0	−34%
	Female	144	5.7	141	5.5	− 0.2	−4%
E955 Firearms and explosives	Male	143	6.0	205	8.5	+ 2.5	+42%
	Female	17	0.7	12	0.5	− 0.2	[1]
E956 Cutting and piercing instruments	Male	59	2.5	77	3.2	+ 0.7	+28%
	Female	20	0.8	23	0.9	+ 0.1	+13%
E957 Jumping from high place	Male	110	4.6	114	4.7	+ 0.1	+2%
	Female	46	1.8	77	3.0	+ 1.2	+67%
E958 Other and unspecified means	Male	162	6.8	202	8.3	+ 1.5	+22%
	Female	62	2.5	108	4.2	+ 1.7	+68%

[1] Small numbers: % unreliable

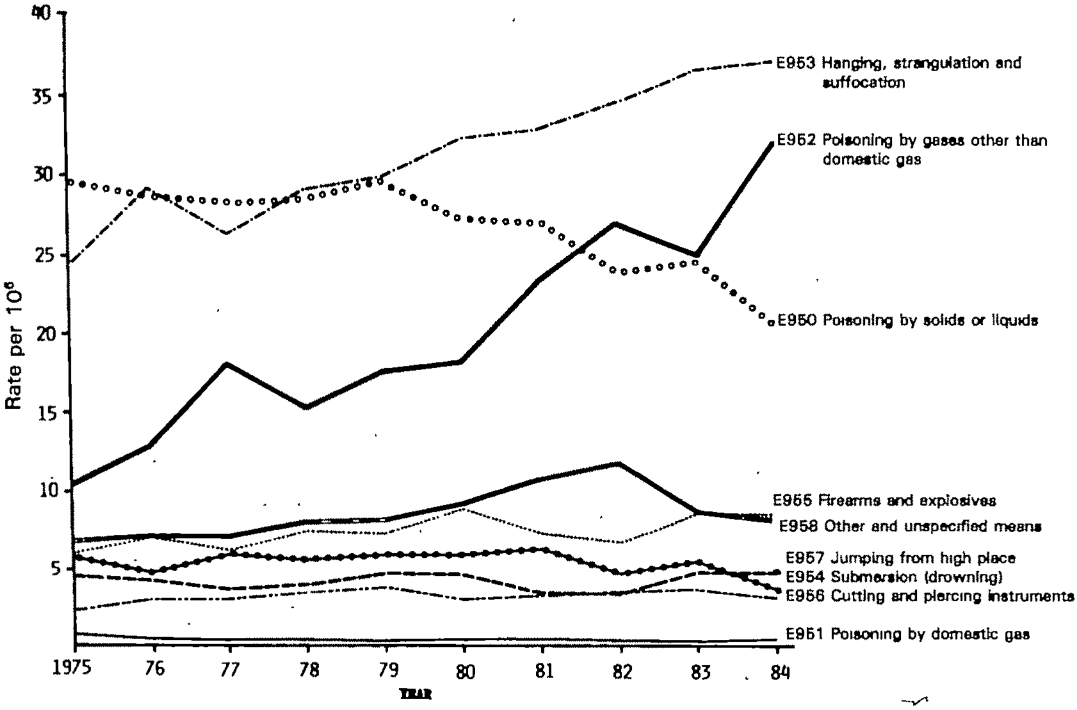

FIG. 2 Suicide rate by different causes (males) for England and Wales: 1975–1984.

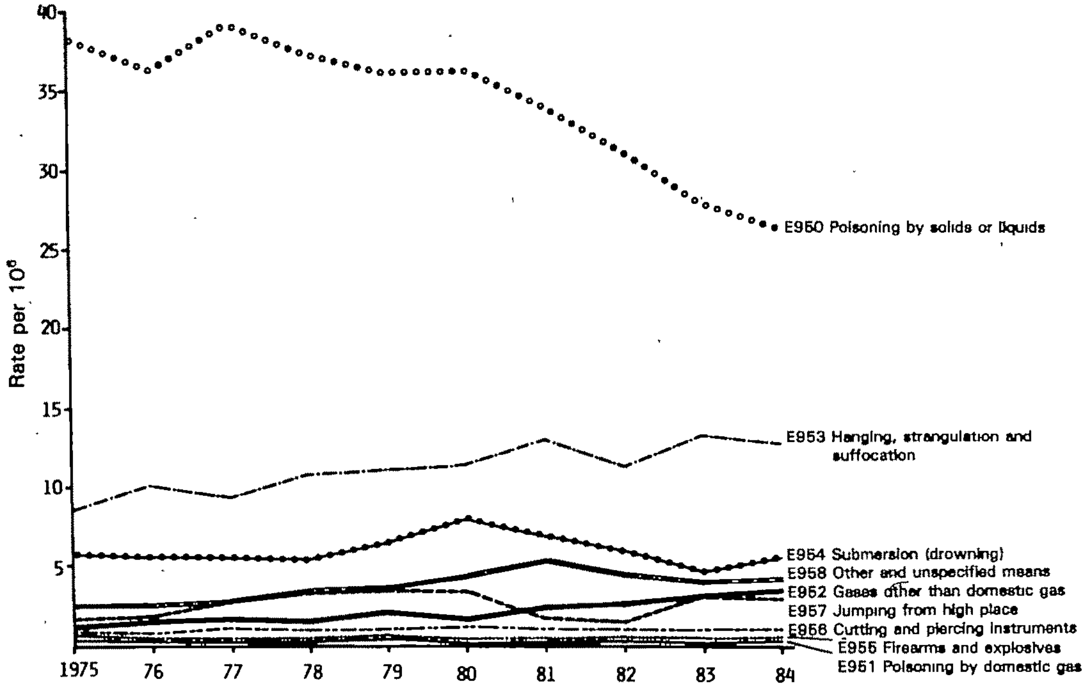

FIG. 3 Suicide rate by different causes (females) for England and Wales: 1975–1984.

TABLE III

Rate (per million population) of suicide and self-inflicted injury in different age-groups in 1975 and 1984

	1975		1984		Change in rate 1975–1984	
Age group	*Males*	*Females*	*Males*	*Females*	*Males*	*Females*
5–14 years	<1	<1	<1	<1	—	—
15–24 years	64	28	75	16	+11(+17%)	−12(−43%)
25–34 years	105	49	140	38	+35(+33%)	−11(−22%)
35–44 years	113	68	165	64	+52(+46%)	−4(−6%)
45–54 years	138	99	182	98	+44(+32%)	−1(−1%)
55–64 years	154	106	168	107	+14(+9%)	+1(+1%)
65+ years	170	106	190	103	+20(+11%)	−3(−3%)

Change in suicide rate in different age-groups: 1975–1984

Table III shows the analysis by age of suicides in 1984, and the change in rate for each group between 1975 and 1984. The suicide rate has increased between 1975 and 1984 for males in all age-groups, particularly in the 25–54 years group. For females, the rate has decreased in the age-group 15–34 years, with little change occurring in females over 35 years.

'Undetermined' and 'accidental' deaths

Categories of 'undetermined' and 'accidental' deaths by causes comparable to suicide are shown in the appendix. Between 1975 and 1984 the 'undetermined' death rate increased by 7 per million to 38 per million for males, and remained unchanged for females at 25 per million. The summation of 'undetermined' death rate plus 'accidental' death rate for causes comparable to suicide showed a small decrease for males of 4 per million to 75 per million in 1984. This is insufficient to account for an increase in suicide rate of 27 per million between 1975–1984. For females, the summation of 'undetermined' death rate plus 'accidental' death rate for causes comparable to suicide decreased by 8 per million to 41 per million in 1984. This change is in the same direction as the small decrease in suicide rate for females between 1975–1984. It is concluded that the change in suicide rate between 1975–1984 is unlikely to be due to a simple change of classification concerning 'undetermined' or 'accidental' deaths.

'Undetermined' and 'accidental' deaths by specific causes

'Undetermined' and 'accidental' deaths by specific causes, comparable to suicide are shown in Table IV. By comparing the changes with those for suicide in Table II, it can be seen that along with the decrease in suicide by poisoning with solid or liquid substances there was also a decrease in 'undetermined' and 'accidental' poisoning by this method. It is therefore unlikely that the decrease in suicide by this method can be explained by a change of classification to 'undetermined' or 'accidental categories'. Similarly, although there was a small decrease in 'undetermined' and 'accidental' poisoning by gases, this is insufficient to account for the increase in suicide by gases other than domestic gas (E952). (In fact, closer examination of the statistics underlines the fact that the increase in suicide by motor vehicle exhaust gas was *not* caused by a change of classification; 'accidental' deaths by this means (E868.2) *increased* for males from 16 in 1975 to 31 in 1984 and decreased for females from 2 in 1975 to 1 in 1984). In addition, the other major change in suicide, by hanging, strangulation or suffocation (E953), cannot be explained by a change of classification to 'undetermined' or 'accidental' death. There was an increase in this category for males, and the small decrease of 0.6 per million for females for 'undetermined' or 'accidental' hanging, strangulation or suffocation is insufficient to account for the increase of 5.1 per million in suicides by this method.

It is also of interest that the smaller changes in suicide by causes E954-E958 (with the minor exception of E955 for females) are not explicable in terms of a change in classification to 'undetermined' or 'accidental' death.

'Undetermined' and 'accidental' deaths by different age-groups

'Undetermined' and 'accidental' deaths for categories comparable to suicide in different age-groups are shown in Table V. The decrease in 'undetermined' and 'accidental' deaths in males under 24 years casts doubt on the reported increase in suicide in this group. For males aged 25–44 years, however, the reported increase in suicide cannot be explained by a change of classification to 'undetermined' or 'accidental' deaths which also increased between 1975–1984. For males aged 45+ years, decreases in 'undetermined' and 'accidental' deaths may account for part, though not all, of the recorded increase in suicide rate.

For females, there was a decrease in 'undetermined' and 'accidental' deaths for categories comparable to suicide in all age-groups except 25–34 years, where there was a small increase. The decrease in suicide recorded in females aged 15–34 years cannot be explained by a change of classification to 'undetermined' or 'accidental' death.

TABLE IV

Numbers (and rates per million population) of 'undetermined' plus 'accidental' deaths, classified by different causes 1975 and 1984 (all ages)

Cause of death (ICD and classification)		*1975*		*1984*		*Change in rate 1975-1984*	
		Number	*Rate*	*Number*	*Rate*		
E980+E850-66 Poisoning by solid or liquid substances	Male	570	23.8	491	20.2	−3.6	−15%
	Female	736	29.1	484	19.0	−10.1	−35%
E981/2+E867/8/9 Poisoning by gases (all gases)	Male	121	5.0	132	5.4	+0.4	+8%
	Female	63	2.5	36	1.4	−1.1	−44%
E983+E913 (except E913.0) Hanging, strangulation and suffocation	Male	133	5.5	137	5.7	+0.2	+4%
	Female	33	1.3	19	0.7	−0.6	−46%
E984+E910 Submersion (drowning)	Male	649	27.1	442	18.2	−8.9	−33%
	Female	243	9.6	238	9.3	−0.3	−3%
E985+E922/3 Firearms and explosives	Male	84	3.5	72	3.0	−0.5	−14%
	Female	5	0.2	11	0.4	+0.2	[1]
E986+E920 Cutting and piercing instruments	Male	20	0.8	38	1.6	+0.8	+100%
	Female	5	0.2	13	0.5	+0.3	[1]
E987+E882 Falling from high place	Male	187	7.8	231	9.5	+1.7	+22%
	Female	68	2.7	87	3.4	+0.7	+26%
E988 Other or unspecified	Male	147	6.1	272	11.2	+5.1	+84%
	Female	77	3.0	163	6.4	+3.4	+113%

1. Small numbers: % unreliable

Table V

Rates (per million population) of 'undetermined' plus 'accidental' deaths[1] in different age groups in 1975 and 1984

Age group	*1975*		*1984*		*Changes in rate 1975-1984*	
	Males	*Females*	*Males*	*Females*	*Males*	*Females*
5-14 years	40	7	23	6	−17(−43%)	−1(−14%)
15-24 years	101	29	79	22	−22(−22%)	−7(−24%)
25-34 years	79	34	94	36	+15(+19%)	+2(+6%)
35-44 years	86	47	88	39	+2(+2%)	−8(−17%)
45-54 years	93	68	84	61	−9(−10%)	−7(−10%)
55-64 years	88	77	87	58	−1(−1%)	−19(−25%)
65+ years	102	86	91	70	−11(−11%)	−16(−19%)

1. For categories comparable to suicide

Discussion

The suicide rate for males increased for each consecutive year during the period 1975-1984, with an overall increase of 30%. During the same period there was little change in the suicide rate for females. Although there was an increase in rate for males of all ages, this was most apparent in the 25-54 years group. There was a decrease in the suicide rate for females in the 15-34 years category.

Suicide by self-poisoning with solids and liquids decreased in both sexes, and there was an increase in poisoning by motor vehicle exhaust gas and by hanging, strangulation and suffocation, particularly in males. Examination of the changes in 'undetermined' and 'accidental' deaths in categories comparable to suicide indicate that the changes observed in the suicide rate could not be explained simply by a change of classification. They are likely to represent real changes in suicide.

The changes observed may not be due to any single cause, but may be multifactoral in origin. It is of interest that the decrease in suicide by poisoning with solids and liquids parallels a decrease in admissions to hospital for adverse effects of chemical substances, which peaked in 1976 and has since been in decline.

(Central Statistical Office, 1982). The greatest decrease in suicide by this method occurred in young females, age 15–34 years—a group which has been most prominent for overdosing. The indications are, therefore, that the popularity of this phenomenon has *decreased* in the last decade, following the 'epidemic' of the 1960s and early 1970s.

The decrease in suicides by poisoning with solids and liquids has been offset by an increase in poisoning with vehicle exhaust gas and by hanging, strangulation and suffocation. An explanation must be sought for the substantial increase in suicide rate, particularly by these methods, in males in the 25–54 years group. Possible factors include unemployment, job insecurity, alcoholism and disrupted family life (as indicated by divorce rates). Unemployment, wine and spirit consumption and the number of divorcees in the population have all shown an increase during the period under review (Central Statistical Office, 1985) and these factors may particularly have affected young male adults.

Hanging has traditionally been employed by males, and may have increased because of decreased availability of domestic gas, and decreased confidence in the lethality of overdosing. Self-poisoning with vehicle exhaust gas has increased, particularly in males, probably because they are more commonly owners and drivers of motor vehicles than females. Both these methods are now more common for males than suicide by self-poisoning with solids or liquids. Hanging, strangulation and suffocation have been at high levels in the past, but the phenomenon of vehicle exhaust gas poisoning is new. The increase in both these methods gives cause for concern, as in neither case can the agents be made less readily available. Hopes for a decrease in the rate must therefore rely on dealing with the causes of suicide and this in turn depends upon a greater understanding of causative factors.

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References

ADELSTEIN, A. & MARDON, C. (1975) Suicides 1961–74. In *Population Trends*, No. 2, London: Her Majesty's Stationery Office.

CENTRAL STATISTICAL OFFICE (1982) *Hospital In-Patient Enquiry*. London: Her Majesty's Stationery Office.

—— (1985) *Annual Abstract of Statistics*. London: Her Majesty's Stationery Office.

McCLURE, G. M. (1984) Trends in suicide rate for England and Wales, 1975–1980. *British Journal of Psychiatry*, **144**, 119–126.

OFFICE OF POPULATION CENSUSES AND SURVEYS (1975–1984) *Mortality Statistics: Cause*. London: Her Majesty's Stationery Office.

SAINSBURY, P. & JENKINS, J. S. (1982) The accuracy of officially reported suicide statistics for purposes of epidemiological research. *Journal of Epidemiology & Community Health*, **36**, 43–48.

Appendix 1

ICD categories of suicide, 'undetermined' deaths and comparable 'accidental' deaths

Cause of death	*Suicide*	*Undetermined death*	*Accidental death*	
			1975–1978	*1979–1984*
Poisoning by solid or liquid substances	E950	E980	E850–E869	E850–E866
Poisoning by gases in domestic use	E951	E981	E870	E867
Poisoning by other gases and vapours	E952	E982	E871–E877	E868–E869
Hanging, strangulation and suffocation	E953	E983	E913 (Excluding suffocation in cot or bed)	E913
Submersion (drowning)	E954	E984	E910	E910
Firearms and explosives	E955	E985	E922–E923	E922–E923
Cutting and piercing instruments	E956	E986	E920	E920
Jumping from high place	E957	E987	E882	E882
Other and unspecified means	E958	E988	—	—

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The Dexamethasone Suppression Test and Prediction of Outcome in Patients Receiving ECT

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Twenty-six in-patients satisfying DSM-III criteria for major depressive episode were assessed using the Newcastle Diagnostic and ECT Predictor Scales and the dexamethasone suppression test (DST), prior to commencing a course of electroconvulsive therapy (ECT). The Newcastle ECT Predictor Scale was successful in predicting both immediate outcome and outcome over the 6 months following ECT; the DST was unsuccessful in predicting either immediate or 6-month outcome.

Abnormalities in the dexamethasone suppression test (DST) have been claimed to have a high degree of specificity for melancholia or endogenous depression (Carroll *et al*, 1981). The DST has also been proposed as a possible guide to the prediction and monitoring of response to electroconvulsive therapy (ECT), with an initial non-suppressor DST predicting a good response to ECT (Albala *et al*, 1981; Coryell, 1982).

The present study compared the ability of the DST to predict ECT response with that of the following clinical scales:

(a) The Newcastle ECT Predictor Scale (Carney *et al*, 1965), which was constructed specifically to identify those patients most likely to respond to ECT.

(b) The Newcastle Scales for the Diagnosis of Depressive Syndromes (Carney *et al*, 1965; Gurney, 1971), which were devised to distinguish between non-endogenous and endogenous forms of depressive illness—the latter generally being considered as the disorder most likely to respond to ECT (Kendell, 1981).

Method

Selection of patients

Twenty-six inpatients satisfying DSM-III criteria for major depressive episode, who had been prescribed ECT by the clinicians responsible for their care, were included, provided that: (i) they did not fulfil the exclusion criteria for the DST suggested by Carroll *et al* (1981); (ii) they showed no evidence of dementia; (iii) they were willing to give informed consent to take part in the trial; and (iv) that it was possible to carry out the pretreatment assessment before the first ECT was administered.

Assessment conditions

The responsible clinicians were kept blind to the researcher's initial assessments, to the Hamilton Ratings and to DST results. Their clinical notes were examined by the researcher assessing immediate clinical outcome and 6-month outcome.

Initial assessment

Patients were interviewed initially by an observer who had access to the case notes; a DSM-III diagnosis was made, and the Newcastle Diagnostic and ECT Predictor Scales completed. Age, sex and concurrent medication were recorded. A separate observer, blind to these assessments, completed the 17-item Hamilton Depression Rating Scale (HDRS) (Hamilton, 1960).

Dexamethasone suppression test

The test procedure was similar to that described by Carroll (1982), with 1 mg of dexamethasone administered orally to each subject at 11 p.m., and blood samples taken at 4 p.m. and 11 p.m. the following day. Plasma cortisol assays were performed with the 'Amerlex' radioimmunoassay kit, using a technique described in detail by Katona & Aldridge (1985).

All the above ratings and tests were carried out between 1 and 7 days before the first ECT; normally, the initial interview was carried out and the dexamethasone administered 2 days before ECT, with the cortisol levels taken and the HDRS completed one day before the first ECT. ECT was administered two to three times weekly (according to clinical judgement), using an Ectron IV machine delivering a high-energy current. The length of the course of treatment was decided by the responsible clinicians.

Assessment during ECT

Wherever possible, the DST and HDRS were repeated the day after the fourth ECT. After the course of ECT, a further DST was carried out and the HDRS completed by the original observer (blind to the original assessments and to DST results) as soon as possible after the clinical decision had been made to give no more ECT. Normally, this was between 1 and 2 weeks after the final ECT. The DST was repeated on the day of the final HDRS. Medication at the end of the course of ECT was recorded, as was the number of ECTs and whether treatment was unilateral or bilateral.

Assessment of immediate outcome

In addition to the final HDRS, a global assessment of outcome was made by another observer, who was blind to the initial assessments, DST results and HDRS Scores. This assessment was based on a scrutiny of the clinicians' notes and nursing notes for the period up to 2 weeks after the final ECT, or until discharge, whichever was the sooner. The ratings were made on a three-point scale: Good = No evidence of depression at the end of treatment; Intermediate = Evidence of improvement but still significantly depressed at the end of treatment; Bad = No improvement, significantly depressed at the end of treatment.

Six-month follow-up

To obtain a limited form of 6-month follow-up assessment, the investigator who had assessed immediate outcome examined the subjects' in-patient and out-patient notes, 6 months after the course of ECT, for unequivocal evidence of deterioration in their mental state, e.g. readmission for further ECT or attempted or completed suicide. General practitioners were contacted about those patients who had been discharged from the out-patient clinic. If there was no evidence of relapse following a good immediate outcome, the 6-month outcome was also classed as good. Patients who were still depressed on discharge or who relapsed after a good immediate outcome were classed as having a poor outcome over 6 months.

Results

Twenty-six patients were studied, ranging in age from 20 to 82 years (mean 54.1). Sixteen patients were women; 24 fulfilled DSM-III criteria for melancholia, and four for bipolar disorder. Their initial assessment data are in Table I.

TABLE I
Initial assessment data (n = 26)

Newcastle 1965 Diagnosis	18 endogenous	8 not endogenous
Newcastle 1971 Diagnosis	20 endogenous	6 not endogenous
ECT Predictor	17 good outcome	9 poor outcome
Initial DST	15 non-suppressor	11 suppressor
Initial HDRS	Range 18–38 (median 25)	

Almost all patients received concurrent psychotropic medication during the course of ECT: 19 received antidepressants, 18 neuroleptics, 12 benzodiazepines and three lithium. Twenty-three patients received bilateral ECT and three unilateral; the number of treatments given ranged between three and 11. Subsequent analysis revealed no significant relationship between either number of ECTs or concurrent medication and outcome or DST results. It will be seen that 15 patients (58% of the total sample and 60% of the DSM-III melancholics) were non-suppressors, according to Carroll's (1982) criteria of 4 p.m. and/or 11 p.m. post-dexamethasone cortisol ≥138 nmol/litre.

Immediate outcome

On case-notes assessment, three patients had a bad immediate outcome, eight an intermediate and 15 a good immediate outcome. The final HDRS rating scores are illustrated in Fig. 1: the final scores fall into three bands, according to the three categories of outcome based on case-note assessment. There were only two discrepant results, both in patients with intermediate outcome on this assessment. The independent HDRS scores thus provide validation of the assessment of immediate outcome based on case notes. We have based further analysis on case-notes outcome assessment, rather than using an arbitrary HDRS cut-off point as the outcome measure. Intermediate and bad immediate outcomes are classed together as 'poor', since both left the patient 'still significantly depressed'.

Prediction of immediate outcome

Of the possible clinical predictors of outcome, the ECT Predictor Scale was strikingly successful (see Table II) being correct in all but four cases. Initial DST non-suppression was not associated with good immediate outcome; indeed, there was a striking though not significant trend for suppressors to have a better immediate outcome than the non-suppressors.

Using final HDRS score ≤6 as the criterion of recovery following Coryell's (1982) criteria, 12 patients had recovered and 14 had not. Repeating the calculations led to the same conclusion — that only the ECT Predictor Scale succeeded in predicting immediate outcome, though with a slightly lower degree of significance.

Prediction of outcome over 6 months

Of the various potential predictors of outcome, only the Newcastle ECT Predictor Scale succeeded in predicting outcome over 6 months.

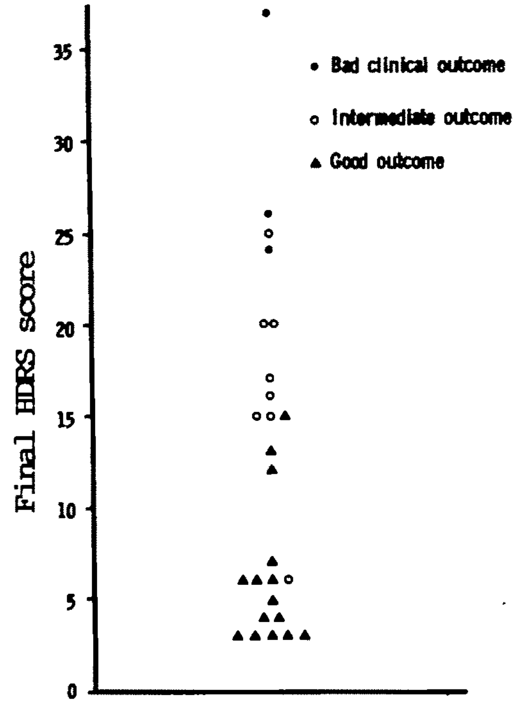

FIG. 1 Final HDRS scores and independent assessment of immediate outcome.

TABLE II
Relationship between predicted and actual outcome

	Immediate outcome			*6-month follow-up*		
	Good	*Poor*	*P*[1]	*Good*	*Poor*	*P*[1]
Initial DST						
Non-suppressor (15)	7	8	NS	3	12	NS
Suppressor (11)	8	3		4	7	
Newcastle 1965 Scale						
Endogenous (18)	11	7	NS	4	14	NS
Non-endogenous (8)	4	4		3	5	
Newcastle 1971 Scale						
Endogenous (20)	11	9	NS	5	15	NS
Non-endogenous (6)	4	2		2	4	
Newcastle ECT Predictor Scale						
Good (17)	14	3	<0.001	7	10	<0.03
Poor (9)	1	8		0	9	

[1] Fisher's Exact Test

Serial DSTs and outcome

Serial DST results and immediate and 6-month outcome are shown in Table III. There was a non-significant trend for persistent non-suppression to be associated with poor immediate outcome, and it should be noted that the nine patients whose initially abnormal DSTs remained abnormal after treatment included three whose DSTs converted to suppression during treatment, only to revert back to non-suppression at the end of treatment.

There was no significant correlation, either before or after treatment, between mean post-dexamethasone cortisol levels and concurrent HDRS scores. However, there was a highly significant correlation (Spearman's rho = 0.55, $P = <0.01$) between changes in HDRS scores and changes in mean post-dexamethasone cortisol levels.

TABLE III
Serial DSTs and outcome

	Immediate outcome		*6-month follow-up*	
	Good	*Poor*	*Good*	*Poor*
Initial DST Suppression (11)				
Persistent suppression (10)	7	3	3	7
Not retested (1)	1	0	1	0
Initial DST Non-suppression (15)				
Suppression after treatment (4)	3	1	1	3
Non-suppression after treatment (9)	3	6	1	8
Not retested (2)	1	1	1	1

Discussion

All the patients in the study had been prescribed ECT on clinical grounds; most had a DSM-III diagnosis of melancholia and most were endogenous depressives on either or both of the Newcastle Diagnostic Scales. In this sample, the pretreatment DST did not predict immediate outcome, and thus our results do not support those of Coryell (1982). In Coryell's study, the DST only predicted outcome according to one of the two methods used to assess outcome, whereas in our series, the DST failed to predict immediate outcome according to either of Coryell's measures.

The Newcastle ECT Predictor Scale was successful at predicting both immediate and 6-month outcome, in contrast to the Northwick Park Study (Clinical Research Centre, 1984), which showed no significant correlation between score on the ECT Predictor Scale and improvement after real or simulated ECT. In our series, the ECT Predictor Scale seemed to be particularly effective in identifying those patients with a poor immediate outcome, and could perhaps be used to help 'screen out' such patients before treatment. In certain clinical situations, such as high suicide risk, even a small chance of improvement might be worthwhile, and the ECT Predictor Scale may best be seen as an adjunct rather than an alternative to clinical judgement.

Diagnosis according to the 1965 and 1971 Newcastle Scales failed to predict outcome. This is perhaps not surprising, in view of the fact that the diagnostic scales were devised in an effort to sharpen the phenomenological distinction between 'neurotic' and 'endogenous' forms of depression, rather than to predict response to a specific form of treatment. The nature, purpose and mathematical structure of scales developed to assist clinical diagnosis and those intended to aid prediction in a specific context are quite different (Roth, 1978).

Like Coppen *et al* (1983), we found a significant relationship between change in mean post-dexamethasone cortisol levels and change in HDRS scores after treatment. Serial post-dexamethasone cortisol levels may perhaps be useful in monitoring recovery from depressive illness. It is suggested that the magnitude of the change in cortisol level may be of greater significance than a knowledge of whether or not the cortisol level has crossed the boundary from non-suppression to suppression, since the latter may represent a relatively small change in post-dexamethasone cortisol level.

Fluctuations in DST status during treatment were observed in three cases; all three patients had a poor immediate outcome, in spite of early normalisation of an initially abnormal DST. This instability in DST status (which has also recently been reported by Bowie (1984)) may limit the extent to which early conversion from non-suppressor to suppressor responses can be taken to predict a good subsequent clinical outcome, as has been claimed by Albala *et al* (1981). Yerevanian *et al* (1983) have suggested that persistent non-suppression after ECT or anti-depressants is a predictor of early relapse after an apparently good immediate outcome. Using our more stringent criterion of recovery, a final non-suppressor DST was associated more frequently with an incomplete recovery than with relapse after a convincingly good immediate outcome.

Conclusions

The findings of this study add to the doubts concerning the clinical value of DST and do not support the use of the DST as a means of predicting immediate response to ECT or relapse within 6 months of ECT. The Newcastle ECT Predictor Scale may be of value in predicting response to ECT, particularly if it is used to 'screen out' patients who are unlikely to make a good response. In those patients with an initially abnormal DST, subsequent DST status during a course of ECT is sometimes unstable, and early normalisation during ECT is not a reliable predictor of a good subsequent outcome. The correlation between mean post-dexamethasone cortisols and concurrent HDRS scores may render the former a useful index of recovery from a depressive illness. We therefore conclude that the DST remains of limited value in the management of patients being considered for ECT, whereas the Newcastle ECT Predictor Scale may be more effective than has hitherto been recognised.

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References

ALBALA, A. A., GREDEN, J. F., TARIKA, J. & CARROLL, B. J. (1981) Changes in serial DSTs among unipolar depressives receiving ECT. *Biological Psychiatry*, **16**, 551–560.

BOWIE, P. C. W. (1984) Dexamethasone suppression test: different categories of response. *British Journal of Psychiatry*, **145**, 218.

CARNEY, M. P. W., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

CARROLL, B. J. (1982) The dexamethasone suppression test for melancholia. *British Journal of Psychiatry*, **142**, 292–304.

——, FEINBERG, M., GREDEN, J. F., TARIKA, J., ALBALA, A. A., HASKETT, R. F., JAMES, N. McI., KRONFOL, Z., LOHR, N., STEINER, M. DE VIGNE, J. P. & YOUNG, E. (1981) A specific laboratory test for melancholia. *Archives of General Psychiatry*, **32**, 15–22.

CLINICAL RESEARCH CENTRE (1984) The Northwick Park ECT trial: predictors of response to real and simulated ECT. *British Journal of Psychiatry*, **144**, 227–237.

COPPEN, A., ABOU-SALEH, M. T., MILLN, P., METCALFE, M., HARWOOD, J. & BAILEY, J. (1983) The dexamethasone suppression test in depression and other psychiatric illness. *British Journal of Psychiatry*, **142**, 498–504.

CORYELL, W. (1982) Hypothalamic-pituitary-adrenal axis abnormality and ECT response. *Psychiatry Research*, **6**, 283–291.

GURNEY, C. (1971) Diagnostic scales for affective disorders. *Proceedings of Vth World Congress of Psychiatry, Mexico City*, 330.

HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

KATONA, C. L. E. & ALDRIDGE, C. R. (1985) The dexamethasone suppression test and depressive signs of senile dementia. *Journal of Affective Disorders*, **18**, 83–89.

KENDELL, R. E. (1981) The present status of ECT. *British Journal of Psychiatry*, **139**, 265–283.

ROTH, M. (1978) Chapter 26. In *Psychiatric Diagnosis: Exploration of Biological Predictors* (eds H. Akiskal & W. Webb), London: SP Books.

YEREVANIAN, B. I., OLAFSDOTTIR, H., MILANESE, E., RUSSOTTO, J., MALLON, P., BACIEWICZ, G. & SAGI, E. (1983) Normalisation of the DST at discharge from hospital: its prognostic value. *Journal of Affective Disorders*, **5**, 191–197.

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Huntington's Chorea:

Some Psychodynamics seen in those at Risk and in the Responses of the Helping Professions

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The autosomal dominant mode of inheritance of Huntington's Chorea (HC) has been established for over 100 years. Strong psychological defences against the acceptance of this knowledge are commonly found both in professionals and in Huntington families and relatives who are at risk. One consequence of maintaining these defences is the continuing transmission of the gene, and therefore the disease, to succeeding generations. The defences of professional persons protect them from the difficult and unsettling task of providing genetic counselling to healthy relatives at risk. An increase in awareness of the psychodynamics involved may lead to constructive thinking about the current deficiencies in care and counselling services provided for these families.

The clinical features of HC have received prominent attention in the literature since George Huntington's lucid description over a century ago (Huntington, 1872; Heathfield, 1973). The fact that the disease is inherited by an autosomal dominant gene has also been recognised since that time. Considerable attention has been directed to scientific investigation of the mechanism by which the abnormal gene causes the degeneration of the central nervous system (Chase *et al*, 1979). This research has not led to any breakthrough in treatment though the development of a genetic marker may well do so (Harper, 1983).

With this in mind it is of considerable interest that so little attention has been paid until recently to the possible benefits of providing skilled genetic counselling services to these families. If those at risk were to be fully informed they might well decide to limit their family size: evidence that this is so has been published (Harper, 1981; Carter *et al*, 1983). Both authors suggest that skilled counselling could very substantially reduce the prevalence of the gene in a few generations. There are about 5-6000 persons with the disease in Britain and a further 50 000 whose lives are burdened by being at risk or married to someone at high risk (Office of Health Economics, 1980).

It seems very important to try to understand why so many of these people receive no counselling even when they have been in contact with otherwise skilled professionals (Martindale & Yale, 1983). It is also important to understand why some families and individuals may not be able to use information that is readily available to them.

The following psychodynamic insights are based on 10 years experience with professionals involved with about 70 HC families as well as direct personal contact with HC patients and those at risk of HC.

Pathological defences seen in some people at risk of HC

I define defence mechanisms as pathological when they are so extensive or rigid that they prevent the individual gradually coming to terms with the possible implications of HC for them and their family's lives. I will emphasise and illustrate the serious problems that follow when these defences are so extensive.

I will also demonstrate the possible developmental background of some of these defences and how the persistence of this disease in many families is related to these defence mechanisms and their interaction with similar mechanisms in professionals.

Denial or disavowal

This term refers to a defence whereby the person refuses to recognise a traumatic perception.

A woman with multiple problems from advanced HC was severely handicapped by her chorea and in a hallucinatory psychotic state. She had recently set fire to her kitchen. Her denial was absolute. "Thank you for coming – we haven't any problems – you don't need to come again". This woman had four children still living with her, aged 8–18, and contact with this family revealed that the older the child, the greater the use of denial of the family problems, especially of the knowledge that mother's and grandfather's

HC had any possible implications for their lives. However, the youngest child was able to talk spontaneously about these worries to the professionals. Prolonged contact with the family led to the firm impression that the family atmosphere of denial, stemming from the parents' denial was a defence mechanism that became increasingly firmly adopted as a personality characteristic as each child grew older and was continually exposed to the family's inability to talk about very painful matters.

It would seem likely that the ability to recognise and come to accept the onset of the disease in oneself might be heavily determined by the pre-morbid defences of the individual.

Reaction formation

This is a defence resembling counter-phobia, whereby an unacceptable feeling or impulse is unconsciously mastered by exaggeration of its opposite.

In some Huntington's families the large number of children can be understood as a result of this defence mechanism. The parents have unbearable feelings of guilt in connection with the knowledge of the suffering that any child could experience from being at risk of the family illness. These feelings cannot be tolerated consciously and in order to defend against them and preserve the wish to have healthy children, the person behaves as if there is *nothing* to worry about – as if to say "Look, I can have an endless number of children without fear for myself or them".

Rationalisation

Rationalisation is the use of logical or acceptable explanations by a person for his attitudes, actions, etc., with the unconscious aim of disguising or not perceiving the true motives.

Whittier gives an excellent example of a mother who consciously knows that her daughter has a 50% risk of developing HC but finds successive rationalisations for keeping this information from her at every stage of her development (for example, "she's too young"; later "I don't want to spoil her chances of dating"; later still "I'm frightened that her engagement will be broken off" etc.). The mother's unconscious use of successive rationalisations was probably to protect herself from her guilt, sadness and bitterness and from her daughter's anger at her possible fate (Whittier *et al*, 1972). The rationalisation is pathological in that it hides the truth of the situation from her daughter and gives her no opportunity to face this reality and decide whether to adjust her life accordingly. In this case, the daughter goes ahead and has a child.

The risk of HC is transmitted to a further generation in ignorance of this risk as a result of mother's defences.

Projection and projective identification

These are very common defence mechanisms used in a variety of ways by those at risk of HC or suffering from the disease. The reason for HC families often avoiding health services are complex and multiple. Often there has been a long history of inadequate if not deleterious responses by professional services. However, in many cases, contact with health services would mean facing the distressing personal problems that Huntington's families would like to hide from. By the processes of projection or externalisation the health services are then felt to be the *source* of the problem and are in turn avoided.

A man trying to avoid his awareness that he was in the early choreiform stages of HC did not attend any health service agency but went to a faith-healer several hours drive away and was reassured that he was not suffering from HC. His mood picked up for a few months (the faith healer having colluded with and reinforced the denial). The HC progressed but he continued to avoid (by denial) allowing his perceptions of his illness becoming fully conscious. Medical services were actively avoided as they were experienced only as useless and/or persecutory. This can be understood as a concrete expression of the feared useless and/or persecuting ill part of himself that he was trying to rid himself of or attribute to an external source. He was, therefore, unable to seek care. His continuing desperate attempts to maintain his denial of the progressive illness and incapacity were successful only at the cost of a further breakdown of his ego in the form of a frank hallucinatory psychotic state. Possibly this served the maintenance of his denial by ensuring that information about his condition from his perceptions was further fragmented.

The defences adopted by some professionals coming into contact with HC families

The previous section briefly illustrated some psychological mechanisms that protect persons at risk from HC from experiencing painful feelings about the disease. It demonstrated that these protective mechanisms paradoxically increase the chances of suffering, especially in the next generation.

Similar defences exist in professionals who come into contact with HC families.

Again, these defences both protect the professional from disturbing feelings and play a very important part in perpetuating the disease, as discussion and counselling with the family about the disease are avoided. It is hoped that increased awareness of these defence mechanisms will alert professionals to their unconscious tendency to avoid providing facilitating counselling for these families. Evidence will be given that this evasion is currently very common and ranges from complete avoidance by the professional of any contact with the HC family to types of involvement that avoid the central issues.

Denial

Denial by professionals of the genetic implications of the disease is common (Martindale & Yale, 1983). It protects them from feeling sadness, hopelessness and horror at the physical and psychological problems of the families. Denial of the genetic implications means that the professional can avoid considering offering genetic counselling.

I have seen neurologists' letters to general practitioners where various details of the disease have carefully been spelled out but reference to the genetic implications for relatives seems to have been selectively omitted. The general practitioner may also then not consider counselling for relatives because of his own denial or from ignorance about the genetic facts.

In one family where the diagnosis was not discussed by professionals with the family, seven children were born in the next three years to relatives who were at risk.

Sometimes, exaggerated attention to a less important detail of the person may have the same function of avoiding the aspects of the disease that the physician finds most uncomfortable.

For example, a neurologist requested a GP's help to get an autopsy because of an unusual clinical feature in a man with probable HC. He did not mention that an autopsy diagnosis would also have been very helpful in counselling the young adult daughters who had never been mentioned by the neurologist in his letters to the general practitioner.

Another hospital specialist told me he had been seeing an HC patient over a number of years mainly for teaching purposes, the emphasis being on demonstrating the movement disorder that would captivate the student audience. The specialist had given no consideration to conveying information about the existence of this genetic disease to those medically responsible for the man's nine siblings and their increasing number of children, now 21 in number (let alone to teaching his students about the psychosocial aspects of the disease).

Displacement of responsibility

Frequently, the professional making the diagnosis will say that he assumed that one of the other disciplines involved had taken on the genetic counselling but it turns out that no arrangements had been made and indeed, other disciplines may have made the same assumption about the diagnostician. Responsibility may well be passed on within a discipline to the most junior member—using the pretext of an interesting case for the junior. I have met several HC families who remember the chain of professionals who were given responsibility for their care for a few weeks only—just enough time to begin to establish a relationship before the professional left.

Projection

A social worker was the legal guardian of three children in care. He said it would be too upsetting for the *children* to see their mother who was in hospital with HC. He had not arranged any visits. When encouraged to do so it transpired that *he* was very anxious about how *he* would cope on this visit, whereas the children were quite keen to visit and coped well with it.

Rationalisation

A doctor explained that he had decided not to counsel the 28-year-old daughter of an affected woman because she was single and therefore the disease had no current relevance to her life!

It is very common for doctors and parents to use conscious explanations to facilitate their denial that children commonly know about the hereditary implications of their parent's illness. If the doctor can allow himself to appreciate this, he will often find the child is full of fantasies and convictions that may be even worse than the reality. They often believe that they will definitely get the illness because of some physical resemblance or personality characteristics, rather than the fact that they have a 50% chance of not getting the disease.

If it is denied that children can be aware of the genetic implications, endless theoretical discussions can occur about why the children should not be told.

When I first worked with an HC family, senior colleagues advised me to spare the children any discussion about HC for a few years yet. It took me some while to realise that I had plenty of clues that informed me that all the five children, aged from 7 to 21, were already distressingly preoccupied with the genetic implications but totally unable to discuss it amongst themselves, believing that the others in the family did not know, or want to know. They were additionally burdened with a professional who also, at this point, did not seem to want to know.

Incurability

Huntington's families are threatening to those professionals who have difficulty working with conditions that cannot be cured. A frequent response is to say or feel that there is nothing that can be done for those families and not to offer them further professional help. It is, of course, true that nothing can be done to reverse the progress of the neurological condition but many disciplines have very useful contributions to make that can considerably ameliorate many of the related problems that can become distressing if unattended to. The stories of Tom told by Gardham (1982) and of Ian Miller by Phillips (1982) are worthwhile reading for anyone who is unaware of either the consequences of professional neglect or the benefits of skilled professional care and facilities.

Furthermore, it is my experience, and that of others, that the quality of care given to HC victims, as perceived by their children, significantly influences the children's likelihood of facing their own possibility of developing the disease and their ability to make use of professional services (Wexler, 1979).

Discussion

Huntingdon's chorea is a disease leading to a very wide range of physical and psychosocial problems. Many different disciplines have areas of expertise that can contribute to the provision of skilled comprehensive care (psychiatrists, neurologists, geneticists, social workers, general practitioners, nurses and occupational therapists as well as the self-help group COMBAT etc.).

However, a previous paper (Martindale & Yale, 1983) described the outcome of a field survey. This demonstrated that opportunities for preventive medicine, especially genetic counselling, were being avoided by professional workers of all disciplines in nearly every Huntington's family uncovered by the survey.

This article emphasises that, whichever discipline is involved, psychodynamic factors can play a very major part in determining whether any care is given and the quality of that care. There is nothing unique about HC in this respect except that the particularly horrific nature of the disease makes it even more likely that psychological defences against involvement will occur. I especially want to emphasise that these defences play a very major part in determining the perpetuation of the disease. They protect families and professionals from accepting the painful reality of the disease and thereby expose them to the consequences of going about matters as if there was nothing disturbing to worry about. For adults at risk, this often takes the form of having children without facing the reality of their own and their children's chances of developing HC. For the professional, it may take the form of not taking any measure to counsel fully those at risk and their relatives. Both family and professional are, therefore, playing a major part in perpetuating the disease, through their understandable difficulties in facing painful realities.

The goals of counselling should not be to make judgements about whether those at risk should or should not have children, but to foster emotional insight so that they can make informed decisions about their future lives. Those who do offer counselling often feel it should only be with *adults* at risk (Harper, 1981). However, if one takes a developmental view of personality organisation, one often finds that those adults at risk of HC have been exposed throughout their formative years to relationships with their families and with professionals in which the evidence of HC has been denied or information about HC has been distorted.

Some of the case material in this paper indicates how the family denial may become internalised and thus become an integral part of the developing personality of children in HC families. If one takes these points into consideration, it may be possible to differentiate, at the time of assessment, those at risk who may be able to make good use of a relatively brief series of information-giving counselling sessions from those who may need a long-term professional relationship, in which they can gradually develop the confidence to face painful matters they have habitually defended against. A further logical extension of my observations would be that families need help in facing the family illness when the children are young, especially when the illness is an integral part of family life. The full co-operation of the parents will need to be elicited to prevent any undoing of the counsellor's work with the children. Premature discussion of the genetic implications before an atmosphere of sufficient trust and support has been achieved may lead only either to denial becoming more entrenched, or to an increase in other forms of resistance to facing the illness. These may include avoidance of contact with the professional (Martindale & Bottomley, 1980).

It is also crucial that the various professionals potentially involved are in good enough communication with each other to clarify who is the best person to take on the counselling in each family. The defences of individual professionals that have been described often prevent this communication and clarification, when no one wants to take on the counselling. It does not seem to me that counselling should be necessarily given by a member of any one profession, but rather by the person who has the best skills and who is in the best position to provide long-term care and counselling. This may be a professional in the primary care situation (social worker, GP, district nurse), if he/she is adequately supported through opportunities for discussion with colleagues with experience of the condition.

In 1981 Harper *et al* published some results of a prospective study of relatives at risk of HC in South Wales. They demonstrated that, coinciding with the introduction of a systematic non-intensive genetic counselling and family support programme, a pronounced fall in the number of births at risk occurred. This contrasted with stable levels before the introduction of the programme. Further support for the effectiveness of even less intensive genetic counselling comes from a smaller study by Carter *et al* (1983).

The services mentioned are very rarely provided elsewhere. Stern & Eldridge (1975) found that medical personnel were cited as the best source of information in only 25% of respondents. Martindale & Yale (1983) found that genetic counselling was rarely offered to those at risk.

The evidence in this paper suggests that a variety of anxieties are aroused in professionals who have contact with both those at risk of HC and those with the disease and that these anxieties inhibit the professional from providing basic counselling services—this mirrors or replicates the difficulties of the families. Therefore, the professionals play a major part in the vicious circle that perpetuates the disease. My findings are similar to those of Britton (1981) who also clearly describes how the pattern of professional care given to other troubled families often unwittingly replicates or mirrors the unacknowledged family problems.

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References

Britton, R. (1981) Re-enactment as an unwitting professional response to family dynamics. In *Psychotherapy with Families, An Analytic Approach* (eds S. Box, B. Copley, J. MacAgna & E. Moustari). London: Routledge & Kegan Paul.

Carter, T. N., Evans, K. A. & Baraitser, M. (1983) Effects of genetic counselling on the prevalence of Huntington's Chorea. *British Medical Journal*, **286**, 281–283.

Chase, T. M., Wexler, N. S. & Barbeau, A. (eds) (1979) *Advances in Neurology*, Vol. 23. New York: Raven Press.

Gardham, F. (1982) *On Nursing Huntington's Chorea*. London: Association to Combat Huntington's Chorea.

Harper, P. S. (1983) A genetic marker for Huntington's Chorea. *British Medical Journal*, **287**, 1567–1568.

——, Smith, S., Tyler, A. & Jones, P. (1981) Decline in the predicted incidence of Huntington's Chorea associated with systematic genetic counselling and family support. *Lancet*, **ii**, 411–413.

Hayden, N. R. (1981) *Huntington's Chorea*, Berlin: Springer-Verlag.

Heathfield, K. W. G. (1973) Huntington's Chorea: A Centenary Review. *Postgraduate Medical Journal*, **49**, 32–45.

Huntington, G. (1872) On Chorea: *Medical and Surgical Reporter, Philadelphia*, **26**, 317.

Martindale, B. V. & Bottomley, V. (1980) The management of families with Huntington's Chorea: A case study to illustrate some recommendations. *Journal of Child Psychology and Psychiatry*, **21**, 343–351.

—— & Yale, R. (1983) Huntington's Chorea. Neglected opportunities for preventive medicine. *Lancet*, **i**, 634–636.

Office Of Health Economics (1980) *Huntington's Chorea*, Pamphlet No. 67. Luton UK: White Crescent Press.

Phillips, D. H. (1982) *Living with Huntington's Disease*, London: Junction Books.

Stern, R. & Eldridge, R. (1975) Attitudes of patients and their relatives to Huntington's disease. *Journal of Medical Genetics*, **12**, 217–223.

Wexler, N. S. (1979) Genetic "Russian roulette": The experience of being "at risk" for Huntington's disease. In *Genetic Counselling: Psychological Dimensions* (ed. S. Kessler) New York: Academic Press.

Whittier, J. R., Heimler, A. & Korenyi, C. (1972) The psychiatrist and Huntington's Chorea. *American Journal of Psychiatry*, **128**, 96–100.

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Cerebral Ventricular Enlargement in Chronic Schizophrenia:
Consistencies and Contradictions

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A study of cerebral ventricular size measured as ventricle to brain ratio (VBR) using computerised tomographic brain scan in chronic schizophrenics provided no support for suggestions that there are significant differences between patients who fall into different clinical subtypes. We found no significant difference in VBR between patients with and without a family history of schizophrenia or between those with or without paranoid symptoms. Applying Crow's classification, contrary to expectations, Type 1 patients had significantly larger ventricles than those with 'mixed' symptomatology (both Type 1 and Type 2 features). We also applied a variety of operational criteria which attempt to define schizophrenia as a whole: of these only Schneider's first-rank symptoms (FRS) yielded conclusive results — FRS-positive patients had significantly larger mean VBR than those without such symptoms. Previously, it has been suggested that ventricular enlargement is more closely associated with 'negative' than with 'positive' symptoms.

Although earlier studies employing lumbar air encephalography produced inconsistent results (Jacobi & Winkler, 1927; Storey, 1966), recent computerised tomographic (CT) scan studies of chronic schizophrenia have been in substantial agreement. Following the first reports by Johnstone *et al* (1976) and Weinberger *et al* (1979) that a proportion of chronic schizophrenics, compared to controls, have enlarged cerebral ventricles, a number of other groups (e.g. Andreasen *et al*, 1982; Nasrallah *et al*, 1982) have confirmed this finding. The percentage of patients with such abnormalities has varied across studies but schizophrenic patient groups consistently show a larger average ventricular size than controls (*Lancet*, 1982), such that this may now be regarded as probably the most replicable biological feature which investigations of the condition have yet revealed.

There is now little dispute about the repeatability and veracity of the finding, but the explanation of ventricular enlargement in schizophrenia is a more contentious issue. The first studies were quickly followed by suggestions that CT scan abnormalities resulted from treatment of schizophrenia or prolonged stays in hospital (Jellinek, 1976; Marsden, 1976). However, subsequent investigations failed to support such a view (Weinberger *et al*, 1979; Tanaka *et al*, 1981). Therefore, most recent interest has centred on more optimistic hypotheses that CT scan abnormalities can tell us something about the aetiology and pathogenesis of schizophrenia, and do not merely reflect secondary consequences of the condition or its treatment.

One of the most provocative hypotheses has been that Johnstone *et al*, 1978). Their observation that ventricular enlargement appeared to be associated with 'negative' clinical features and a degree of cognitive impairment led to the suggestion that CT scan abnormalities provide the structural basis for the 'dementia in dementia praecox'. Subsequently, Crow (1980) proposed that schizophrenia could be divided into two syndromes — a Type 1 characterised by positive symptoms, good response to neuroleptics and probably resulting from abnormalities in brain dopaminergic systems, and Type 2 in which negative symptoms predominate, where response to neuroleptics is less satisfactory and where there is an association with cerebral ventricular enlargement. It was also suggested that in the Type 2 syndrome, ventricular enlargement might be associated with cognitive impairment. Unfortunately, a recent study by members of the same group (Owens *et al*, 1985) failed to show a relationship between ventricular size and cognitive abnormalities. Furthermore, Crow (1980) did not suggest that the two subtypes were necessarily distinct and non-overlapping. Nevertheless there remains considerable interest in such a classification scheme as a starting point for achieving a better understanding of the aetiology of schizophrenia and for deriving an appropriate rationale for the prescription of drugs.

An alternative aetiologically based classification has been put forward by Murray *et al* (1985): the argument is that a genetic contribution is the best established aetiological factor in schizophrenia (Gottesman & Shields, 1982) but a substantial proportion of schizophrenics have no secondary cases among their relatives. In identical twins discordant for schizophrenia, schizophrenic probands have significantly larger ventricles than the unaffected co-twins and there is a marked tendency for large ventricles to be associated with absence of family history in first-degree relatives (Reveley *et al*, 1984). It was therefore suggested that schizophrenia can be subdivided into a familial form, with normal cerebral ventricles and a presumed genetic aetiology, and a 'non-genetic' or sporadic form, associated with absence of genetic loading, enlarged cerebral ventricles and environmental insult to the brain, e.g. perinatal trauma (Murray *et al*, 1985).

Yet another group were unable to find an inverse relationship between presence of family history and enlargement of cerebral ventricles (Nasrallah *et al*, 1982) but did suggest that CT scan abnormalities might be associated with a more 'traditional' subtypology. The division of schizophrenia into hebephrenic and paranoid categories is notoriously imprecise and unreliable but Tsuang & Winokur (1974) have produced operational definitions of hebephrenic, paranoid and 'undifferentiated' schizophrenic subtypes which greatly improve interrater reliability (McGuffin *et al*, 1984). Applying Tsuang & Winokur (1974) criteria, Nasrallah *et al* (1982) found an association between ventricular enlargement and the paranoid category.

In our study on the CT brain scan appearances of chronic schizophrenics, we considered that each of these systems of the sub-classification merited further attention. However, a further problem is that of how to define schizophrenia as a whole. Operational diagnostic criteria provide a potential 'remedy for diagnostic confusion' (Kendell, 1975) for biological research in schizophrenia but there are now many systems of diagnosis available which show poor agreement one with another (Kendell *et al*, 1979). In the face of such difficulties, Kendell (1983) has suggested a 'polydiagnostic' approach, in which a range of commonly used definitions of schizophrenia are applied to the same set of patients, and the results analysed separately for each. There are two arguments in favour of this. Firstly, no clear-cut and conclusive method exists of deciding whether one set of criteria is 'more valid' than another. Secondly, a polydiagnostic scheme may help decrease disparities in the results of different investigators: (e.g. where investigator 'A' using criteria 'X' produces findings which are at odds with those of investigator 'B' using criteria 'Y'). We therefore decided to assess our patients using an operational criteria checklist composed of items extracted from a variety of commonly used operational diagnostic criteria for schizophrenia and which facilitates polydiagnostic investigation while preserving a satisfactory level of inter-rater reliability (Farmer *et al*, 1983; McGuffin *et al*, 1984).

Subjects and methods

Subjects

Thirty-five patients with a hospital diagnosis of schizophrenia (ICD-295) were selected as representing a series of 'typical' cases of chronic schizophrenia, requiring continued neuroleptic medication to ameliorate or prevent recrudescence of symptoms. All underwent computerised tomographic (CT) brain scanning using a 1010 EMI CT Scanner. The mean age at scan was 37.5 years (s.d. 9.3 years, range 22–57 years). The mean length of illness was 13 years (range 4–22 years).

Clinical assessment and diagnosis

Each subject's age at onset of illness (defined as date of first psychiatric onset) and age at scan was recorded. Duration of illness was calculated as the difference between these two dates. An independent investigator, blind to ventricular size measurements, interviewed the patient and at least one close relative informant to establish the presence or absence of a family history of schizophrenia in first-degree relatives. Where possible, hospital notes of relatives were obtained and examined to confirm diagnosis.

Our operational diagnostic criteria checklist was completed on every patient. This was an expanded version of one previously described (Farmer *et al*, 1983; McGuffin *et al*, 1984), which has been shown to have good item by item inter-rater reliability. The checklist (Appendix 1) was completed for each subject from detailed case records of good standard, which included a personal interview with at least one of the authors. The checklist was then scored dichotomously, as previously described — '0' if the item was absent and '1' if ever present. Where there was dubiety about presence or absence of particular checklist items, the patient was re-interviewed by A. E. F. The checklist data were entered into a computer programme SORT to produce a variety of operational definitions of schizophrenia (Schneider, 1959; Feighner *et al*, 1972; Spitzer *et al*, 1978; Carpenter *et al*, 1973) as previously described (McGuffin *et al*, 1984). In addition, the checklist was expanded slightly, enabling the DSM-III definition to be applied (American Psychiatric Association, 1980) (see Appendix 1). Two of us (A. E. F. and P. McG.) were able to achieve very good inter-rater reliability for DSM-III criteria, using the expanded checklist on a separate series of 20 consecutive admissions with psychotic disorders. ($K = 0.840$, $P = 0.00005$).

Subtyping of the patients into hebephrenic, paranoid and 'undifferentiated' categories was also derived from the checklist scores as previously described (McGuffin *et al*, 1984). In addition we applied a more recently derived method of categorising schizophrenics into categories which we have called 'H Type' (hebephrenic-like) and 'P Type' (paranoid-like) (Farmer *et al*, 1984). Lastly, we wished to divide our patients according to the scheme suggested by Crow (1980) descriptions were cast in an operational format and with his helpful advice we arrived at checklist-based algorithms enabling the separation of schizophrenic patients into Type 1, Type 2 and 'mixed' categories (see Appendix 1). Again we were able to establish excellent inter-rater agreement ($K=0.93$, $P=0.00001$) using a separate consecutive series of admissions.

Assessment of CT scans

The maximum ventricular-brain ratio (VBR) was measured for each scan using a semi-automated method (Reveley, 1985) in which the VBR is measured on the slice with the largest ventricular area. Pixels with a value of 0–25 Hounsfield Units (HU) are considered to represent areas of cerebrospinal fluid (CSF) and those with values from 0–99 HU represent areas of brain plus CSF. A computer count is made of pixels in the ventricular area with a value of 0–25 and of the total number of pixels within the inner table of the skull with a value of 0–99. The VBR (maximum slice) is the ratio (× 100) of these counts.

Results

Mean VBR was 5.88 (s.d. 3.16, range 1.0–15.2). The distribution was positively skewed (skewness 0.85) although when the square root transform for VBR was taken an acceptable Gaussian distribution resulted. Square root transformation of the VBR was therefore used in subsequent analyses. Mean root VBR for 25 male subjects was 2.39, and for 10 female subjects was 2.08. There was no significant difference in ventricular size between the sexes ($t=-1.22$, $P=0.11$).

There was no significant correlation between VBR and age of illness onset, age at scan or length of illness ($r=0.15$, 0.13 and 0.01 respectively). Similarly, there was no significant difference in VBR between six (17%) family history-positive subjects and 29 (83%) subjects who had no family history of schizophrenia in first-degree relatives (mean root VBR, family history-positive subjects = 2.17, family history-negative subjects = 2.33; $t=-0.49$, $P=-0.31$).

Table I gives the mean square root VBR and number of subjects in each category for five operational definitions. Schneider's and DSM-III provide dichotomous diagnoses (i.e. 'positive' or 'negative') and non-paired *t*-tests were carried out to compare mean root VBRs for each group. Schneiderian first-rank symptom-positive subjects were shown to have significantly larger mean ventricular size than first-rank symptom-negative subjects ($P=0.008$). However, DSM-III 'positive' cases were not significantly different from DSM-III 'negative' subjects.

Feighner's, Spitzer's and Carpenter's operational criteria provide a grading of diagnosis. Feighner classifies into 'definite', 'probable' and 'negative' categories, Spitzer into 'broad', 'narrow' and 'negative', and the Carpenter system provides '5-cut off', '6-cut off' and 'negative' categories. One-way analysis of variance was carried out to compare

TABLE I

				One-way analysis of variance				
Operational definition	*No. of subjects*	*Mean root VBR*	*Source*	*Sum of squares*	*d.f.*	*Mean square*	*F Ratio*	*P*
Feighner								
Negative	8	2.39	Between	3.38	2	1.69	4.29	0.02
Probable	10	1.82	Within	12.58	32	0.394		
Definite	17	2.54						
Carpenter								
Negative	16	2.13	Between	1.12	2	0.59	1.20	0.31
5 cut off	10	2.32	Within	14.84	32	0.46		
6 cut off	9	2.57						
Spitzer								
Negative	6	3.42	Between	0.65	2	0.32	0.68	0.51
Broad	1	1.50	Within	15.31	32	0.48		
Narrow	28	2.30						

Operational criteria		*No. of subjects*	*Mean root VBR*	*Non paired t-test*		
				T	*d.f.*	*P*
Schneider	Negative	15	1.99	2.53	33	0.0083
	Positive	20	2.54			
DSM-III	Negative	5	2.46	−0.57	33	0.29
	Positive	30	2.27			

mean root VBR for each group, for each of the three definitions. The results for Spitzer's and Carpenter's criteria were not significant. Feighner's 'definite' criteria showed significant difference between groups, but Table I shows that the distribution of mean root VBRs is somewhat odd, in that Feighner 'probable' subjects have the smallest mean root VBR, while Feighner 'negative' subjects are intermediate between these two.

Table II shows the number of subjects and mean root VBR for each subtypology. There proved to be no patients of the pure Crow Type 2, using the operational version of the Crow classification. Tsuang & Winokur non-paranoids, Crow Type 1 and Farmer H-type have the largest mean root VBR (2.41, 2.50 and 2.46 respectively) while Tsuang & Winokur paranoid, Farmer P-type and Crow mixed type have the smaller mean root VBR 1.96, 2.13 and 2.09 respectively). Only in the case of the Crow subtypology does the difference in mean root VBR attain statistical significance ($P = 0.04$); however, it is noteworthy that this is in the opposite of the expected direction in that Type 1 patients have a larger mean VBR than do Type 2 patients.

Discussion

Our aim was not to repeat the now well replicated finding that a proportion of schizophrenic patients show enlarged lateral cerebral ventricles, but rather to examine in detail the clinical features which may be associated with ventricular size, in an attempt to establish whether certain symptoms or clusters of symptoms distinguish patients with and without ventricular enlargement. In particular, we were keen to re-assess the claims that certain subtypologies show a relationship with ventricular size. We also aimed to discover whether any of the commonly used and competing operational diagnostic systems offered significant advantage in detecting a form of schizophrenia with a high rate of CT brain scan abnormality.

TABLE II
Ventricular enlargement and subtypes of schizophrenia

		No. of cases	*Mean root VBR*	*P*
Farmer	H-type	18	2.46	N/S
	P-type	17	2.13	(*t*-test)
Crow	Type I	18	2.50	
	Mixed	17	2.09	0.04
	Type II	0	—	(*t*-test)
Tsuang & Winokur (all subjects)	Hebephrenic	6	2.65	NS
	Paranoid	10	2.21	
	Undifferentiated	19	2.24	(Analysis of variance)
Feighner's subjects ($n = 27$)				
Tsuang & Winokur	Non-paranoid	19	2.41	NS
	Paranoid	8	1.96	(*t*-test)

In common with previous groups we obtained a positively skewed distribution of VBRs in our schizophrenic patients, but taking the square root of the results transformed the data to normality and enabled us to use parametric statistics in our analysis. There was no significant correlation between root VBR and age of onset or duration of illness. There was only a small and non-significant correlation between root VBR and age at scan but this is perhaps not surprising in view of the comparative youth of our sample (mean age 37.5 years, maximum age 57 years).

We found no significant difference between the mean VBRs of patients with or without a positive family history of schizophrenia. We thus found no support for the sporadic/familial classification scheme proposed by Murray *et al* (1985). It seems unlikely that our lack of significant difference between the family history-positive and -negative groups is simply due to a Type 2 error and small sample size, since there is not even a trend towards larger cerebral ventricles in the family history-negative group. Indeed, it is the family history-positive schizophrenics who have a slightly larger mean VBR. The division of schizophrenic patients into 'family history-positive' and 'family history-negative' groups is a difficult and unsatisfactory exercise in a number of respects. Firstly, some authors (e.g. Reveley & Chitkara, 1985) take 'family history-positive' to mean a family history of any major psychiatric disorder. However, since the familial diathesis in schizophrenia appears to be fairly specific for schizophrenia and schizophrenia spectrum disorders (Gottesman & Shields, 1982), it seems better to restrict the term 'family history-positive' to those patients who have secondary cases of schizophrenia in their family. Secondly, family history varies according to the quality of available information and the reliability of informants: certainly in our sample we have somewhat more complete information on some families than others. However, since family history was obtained 'blind' to CT scan results, it is unlikely that any bias could have been introduced which would lessen our chance of finding a negative family history-enlarged VBR association. Although one recent study (Turner *et al*, 1986) does support the findings of Reveley *et al* (1984), it is worth noting that most others (Nasrallah *et al*, 1982; Owens *et al*, 1985), in common with ourselves, do not.

On applying the operational checklist version of Crow's (1980) criteria, we were unable to find any pure Type 2 patients. However, nearly half of our subjects (17 out of 35) fell into the 'mixed' category, showing both Type 2 and Type 1 features. Unexpectedly again, it was the pure Type 1 patients who had the larger mean VBR, with the difference between the two groups (transformed data) being significant at the $P=0.05$ level. It could be argued that the version of the Crow (1980) criteria applied here, although providing good inter-rater reliability, is somewhat oversimplified and we had no specific information about cognitive impairment. The Northwick Park group (Owens *et al*, 1985) and a recent study from Oxford (Kolakowska *et al*, 1985) found no relationship between lateral ventricular size and cognitive impairment. However, it may be that other signs of CNS dysfunction, for example the presence of abnormal movements, are associated with enlarged cerebral ventricles (Owens *et al*, 1985) and such features might properly be incorporated into a revised version of the Type 2 syndrome definition (Crow, 1985).

Our third set of subtypologies were more closely related to traditional categories. Tsuang & Winokur (1975) criteria were applied but our data failed to reveal any significant difference between the hebephrenic, undifferentiated and paranoid categories in respect of ventricular size. Once again, the direction of our findings was the reverse of previously published results. Nasrallah *et al* (1982) found that paranoid schizophrenics had significantly larger ventricles than those who were not of the paranoid subtype. In our series it is the non-paranoid patients who have the slightly larger mean VBR and this applies whether we take the patient group as a whole or only the subset of patients who fulfill the Feighner (1972) criteria for schizophrenia (the latter being the way that Tsuang & Winokur (1975) originally suggested that their criteria be applied). We also used a method of subtyping devised by Farmer *et al* (1984), which although based on multivariate statistical analysis, consists of categories which are somewhat similar to the classical hebephrenic/paranoid classes. There was no significant difference between the groups, but again the H-Type or hebephrenic-like patients showed a slightly larger mean VBR than those of the P-Type or paranoid-like subgroup.

Finally, we examined the VBRs in our patients after classifying them according to a variety of operational diagnostic criteria (Schneider, 1959; Carpenter *et al*, 1973; Feighner *et al*, 1972; Spitzer *et al*, 1978; American Psychiatric Association, 1980). Only the criteria of Feighner *et al* (1972) and Schneider's first-rank symptoms (1959) yielded results which were significant. The Feighner classification allows the separation of patients into definite, probable, and negative (non-schizophrenic) categories. A one-way analysis of variance of root VBR yields significant differences within these groups but the findings are somewhat odd, in that the patients classified as Feighner 'probable' have a lower mean root VBR than the 'negative' or 'definite' groups, whereas, if we were to postulate that the Feighner definition provided a grading either of severity or of certainty of diagnosis, we might have expected the 'probable' category of patient to have a mean VBR which was intermediate between that of the 'negative' and the 'definite' categories. The findings regarding the Schneider criteria are more straightforward, in that there is a highly significant difference between those patients exhibiting first-rank symptoms and those who do not, with the Schneider 'positive' patients having the larger VBRs. Even if we apply a conservative correction factor and multiply the t value by the number of different operational systems used, the difference between the Schneider-positive and Schneider negative patients remains significant at the $P=0.04$ level. This finding is of interest in that several other groups have suggested that large ventricular size is most commonly associated with negative symptoms (Andreasen & Olsen, 1982; Gross *et al*, 1982; Kling *et al*, 1983; Kemali *et al*, 1985; Williams *et al*, 1985) or a Type 2 cluster of symptoms. Here, we have found that it is those patients with classical positive symptoms and those who fall into a Type 1 category who have the larger ventricles.

The subjects described here are in no way an unusual group and were selected specifically as a representative sample of patients with chronic schizophrenia. In the absence of a control group we can make no authoritative statements about which of them are 'normal' or 'abnormal' with regard to ventricular size, and even with access to acceptable controls such separations are somewhat arbitrary (Reveley, 1985). The mean and range of the VBRs in the patients reported here are, however, similar to those in previously described series of schizophrenic patients. Our focus of interest has been entirely upon within-group differences but our analyses provide no support for a consistent relationship between ventricular size and any of a range of clinical subtypes. Our most significant and quite unexpected finding is that in this series ventricular size is associated with the presence of positive symptoms. Inconsistencies and contradictions regarding the clinical features associated with ventricular size are present throughout the recent literature, and it is possible to speculate that technical differences in CT scanning and measurement, and differences in diagnostic criteria or the way that they are applied, play some part. A more fundamental problem may be that most workers have sought to

Coding algorithms[1] for Crow subtypology

Crow Pure Type I	A, B and C required A. One of 26 to 44=1 B. 23=0 C. 20=0
Crow Pure Type II	A, B and C required A. One of 20=1, 22=1, 23=1 B. 26–44=0 C. 48=0
Crow Mixed Type	A and B required A. One of 26 to 44=1 B. One of 20=1, 22=1, 23=1

[1] Based on personal advice provided by Dr Crow

Coding algorithm for DSM-III schizophrenia

A, B, C and D required
A. One of 7 or 8=1
B. 2 and 3=<45
C. 47=1
D. One of 27 to 32, 36 to 39, 41 to 44=1 OR one of 18, 19, 20=1 AND One of 15, 16, 22, 23, 24 26, 35, 40=1

NB Coding algorithms for all other operational definitions previously published (McGuffin *et al*, 1984, Farmer *et al*, 1984)

define categories of patients which differ with respect to a continuous measure of ventricular size such as VBR. No investigators have claimed bi-modality in the distribution of ventricular size and so we might pose the question whether any separation into discrete categories is warranted. However, the presence of bi-modality in itself may be misleading (Murphy, 1964) and a more rational approach to defining subpopulations within schizophrenia with respect to ventricular size might be to carry out an analysis of VBR in patient samples to discover whether the distribution can be more satisfactorily explained by the mixing of two or more curves. The general approach has been outlined (in a somewhat different context) by Everitt (1981) and would seem to be warranted here.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) DSM-III. Washington DC: Division of Public Affairs, APA.

Andreasen, N. C., Olsen, S. (1982) Negative v. positive schizophrenia. Definition and validation. *Archives of General Psychiatry*, 39, 789–794.

——, Smith, M. R., Jacoby, C. G., Dennert, J. W., Olsen, S. A. (1982) Ventricular enlargement in schizophrenia: Definition and prevalence. *American Journal of Psychiatry*, **139**, 292–296.

Carpenter, W. T., Strauss, J. S. & Bartoko, J. J. (1973) Flexible system for the diagnosis of schizophrenia: A report from *Science*, **182**, 1275–1278.

Crow, T. J. (1980) Molecular pathology of schizophrenia: more than one disease process. *British Medical Journal*, **1**, 66–68.

—(1985) The two syndrome concept: origins and current status. *Schizophrenia Bulletin*, **11**, 471–486.

Appendix 1

Operational criteria checklist for schizophrenia

1. Sex
2,3. Age of onset
4. Single
5. Unemployed
6. Illness duration 2 weeks
7. Illness duration 6 months
[1]8. Prodromal/acute/residual symptoms for 6 months
9. Poor premorbid work adjustment
10. Poor premorbid social adjustment
11. Pre-existing personality disorder
12. Alcohol/drug abuse within 12 months of onset
13. Family history of schizophrenia
14. Family history of other psychiatric disorder
15. Bizarre behaviour
16. Catatonia
17. Speech difficult to understand
18. Incoherent
19. Positive formal thought disorder
[1]20. Negative formal thought disorder
21. Affective symptoms prominent
22. Restricted affect
23. Blunted affect
24. Inappropriate affect
25. Rapport difficult
26. Persecutory delusions
27. Well organised delusions
28. Grandiose delusions
29. Delusions of influence
30. Bizarre delusions
31. Widespread delusions
32. Delusions of passivity
33. Primary delusional perception
34. Other primary delusions
35. Delusions and hallucinations lasting one week
[1]36. Persecutory/jealous delusions and hallucinations for one week
37. Thought insertion
38. Thought withdrawal
39. Thought broadcast
40. Thought echo
41. Third person voices
42. Running commentary voices
43. Abusive/persecutory/accusatory voices
44. Other non-affective auditory hallucinations
45. Information not credible
46. Lack of insight
[1]47. Deterioration from premorbid level of functioning
[1]48. Schizophrenic symptoms respond to phenothiazines

[1] Additional items added to checklist to enable DSM-III and Crow operational definitions to be made.

Everitt, B. S. (1981) Bimodality and the nature of depression. *British Journal of Psychiatry*, **138**, 336–339.

Farmer, A. E., McGuffin, P., Spitznagel, E. L. (1983) Heterogeneity in schizophrenia: a cluster analytic approach. *Psychiatry Research*, **8**, 1–12.

——, McGuffin, P., Gottesman, II. (1984) Searching for the split in schizophrenia: a twin study perspective. *Psychiatry Research*, **13**, 109–118.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R., Winokur, G., Munoz, R. (1972) *Archives of General Psychiatry*, **26**, 57–63.

Gottesman, II, & Shields, J. (1982) Schizophrenia. *The Epigenetic Puzzle*. Cambridge: Cambridge University Press.

Gross, G., Huber, G. & Schutter, R. (1982) Computerised tomography studies on schizophrenic diseases. *Archive fur Psychiatrie und Nervenkrankheiten*, **231**, 519–526.

Jacobi, A. & Winkler, H. (1927) Encephalographischen Studien an chronisch Schizophrenen. *Archiv fur Psychiatrie und Nervenkrankheiten*, **81**, 299–332.

Jellinek, E. H. (1976) Cerebral atrophy and cognitive impairment in chronic schizophrenia. *Lancet*, **2**, 1202–1203.

Johnstone, E. C., Crow, T. J., Frith, C. D., Husband, J., Kreel, L. (1976) Cerebral ventricular size and cognitive impairment in chronic schizophrenia.

——, ——, ——, Stevens, M., Kreel, L., & Husband, J. (1978) The dementia of dementia praecox. *Acta Psychiatrica Scandinavica*, **57**, 305–324.

Kemali, D., Mai, S., Galderisi, S., Ariano, M. G., Cesarelli, M., Milici, N., Salvati, A., Valente, A. & Valpe, M. (1985) Clinical and neuropsychological correlates of cerebral ventricular. *Journal of Psychiatric Research*, **19**(4), 587–596.

Kendall, R. E. (1975) The role of diagnosis in psychiatry. Oxford: Blackwell Scientific Publications.

—— (1982) The choice of diagnostic criteria for biological research. *Archives of General Psychiatry*, **39**, 1334–1335.

——, Brockington, I. F., Leff, J. P. (1979) Prognostic implications of six alternative definitions of schizophrenia. *Archives of General Psychiatry*, **36**, 25–31.

Kling, A. S., Kurtz, N. Tachini, K. & Orzeck, A. (1983) Ct scans in sub-groups of chronic schizophrenics. *Journal of Psychiatric Research*, **17**(4), 375–384.

Kolakowska, T., Williams, A. W., Jambor, K. & Ardern, M. (1985) Schizophrenia with good and poor outcome. III Neurological 'soft' signs, cognitive impairment and their clinical significances. *British Journal of Psychiatry*, **146**, 348–357.

Lancet (1982) Editorial: The CT scan in schizophrenia. *Lancet ii*, 968.

Marsden, C. D. (1976) Cerebral atrophy and cognitive impairment in chronic schizophrenia. *Lancet*, **ii**, 1079.

McGuffin, P., Farmer, A. E., Gottesman, I. I., Murray, R. M., Reveley, A. M. (1984) Twin concordance for operationally defined schizophrenia. *Archives of General Psychiatry*, **41**, 541–545.

Murphy, E. A. (1974) One cause? Many causes? The argument from the bimodal distribution. *Journal of Chronic Diseases*, **17**, 301–324.

Murray, R. M., Lewis, S. W., Reveley, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, **ii**, 1023–1026.

Nasrallah, H. A., Charles, G. J., McCalley-Whitters, M. & Kuperman, S. (1982) Cerebral ventricular enlargement in subtypes of chronic schizophrenia. *Archives of General Psychiatry*, **39**, 774–777.

Owens, D. G. C., Johnstone, E. C., Crow, T. J., Frith, C. D., Jagoe, J. R. & Kreel, L. (1985) Lateral ventricular size in schizophrenia: relationship to the disease process and its clinical manifestations. *Psychological Medicine*, **15**, 27–41.

Reveley, M. A. (1985) Ventricular enlargement in schizophrenia. *British Journal of Psychiatry*, **146**, 367–371.

——, Chitkara, B. (1985) Letter to Editor. *Lancet*, June 29, 1503.

Reveley, A. M., Reveley, M. A., Murray, R. M. (1984) Cerebral ventricular enlargement in "non-genetic" schizophrenia: a controlled twin study. *British Journal of Psychiatry*, **144**, 89–93.

Schneider, K. (1959) *Clinical psychopathology*. Translated by M. Hamilton, New York: Grune & Stratton.

Spitzer, R. L., Endicott, J., Robins, E. (1978) *Research Diagnostic Criteria*. Instrument No 58. New York: New York Psychiatric Institute.

Storey, P. B. (1966) Lumbar air encephalography in chronic schizophrenia: a controlled experiment. *British Journal of Psychiatry*, **112**, 135–144.

Tanaka, T., Hazama, H., Kawahara, R. & Kobayashi, K. (1981) Computerised tomography of the brain in schizophrenic patients. *Acta Psychiatrica Scandinavica*, **63**, 191–197.

Tsuang, M. T., Winokur, G. (1974) Criteria for subtyping schizophrenia. *Archives of General Psychiatry*, **31**, 43–47.

Turner, S., Toone, B., Brett-Jones, A. (1986) Computerised tomography scan changes in early schizophrenia — preliminary findings. *Psychological Medicine*. (In press).

Weinberger, D. R., Torrey, F. E., Neophytides, A. N., & Wyatt, R. J. (1979) Lateral cerebral ventricular enlargement in schizophrenia. *Archives of General Psychiatry*, **36**, 735–739.

Williams, A. O., Reveley, M. A., Kolakowska, T., Ardern, M. & Mandelbrote, B. M. (1985) Schizophrenia with good and poor outcome: II. Cerebral ventricular size and its clinical significance. *British Journal of Psychiatry*, **146**, 239–246.

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The Scottish First Episode Schizophrenia Study

I. Patient Identification and Categorisation

THE SCOTTISH SCHIZOPHRENIA RESEARCH GROUP

Forty-nine first episode schizophrenics were identified and considered to be representative of the parent schizophrenic population. Patients were rated before treatment with the Present State Examination. Findings are presented with regard to Catego classes, prevalence of first rank symptoms, conformity to ICD diagnoses, Feighner and Research Diagnostic Criteria and presence of a depressive syndrome. Physical examination revealed isolated neurological signs; in addition two neuroleptically naive patients had abnormal involuntary movements.

In recent years the treatment of schizophrenia has changed considerably, first because of widespread use of neuroleptics as maintenance therapy, and secondly as a result of greater awareness of patients' social needs; most schizophrenics are now cared for throughout most of their illness in the community.

In view of these developments a cohort of first episode schizophrenics has been identified. It is intended to follow them prospectively for 5 years, with detailed assessment at the time of first hospital admission, and at 1, 2 and 5 years after the first admission. The project aims to chart the clinical course of the illness, to compare and contrast different methods of categorising schizophrenia, to assess the efficacy of drug treatment, to examine intellectual and radiological changes, and to measure the impact of the illness on patients' relatives.

This series of papers examines the first episode of illness and this first paper describes the identification and categorisation of patients.

Method

All members of the Group for the Study of Rehabilitation and Community Care of the Scottish Division of the Royal College of Psychiatrists were invited to participate. Consultant psychiatrists from seven hospitals expressed interest and eventually patients from four hospitals entered the study over 2 years. The study was approved by the relevant hospital ethical committees.

Patient identification and categorisation

The criterion for entry was the responsible clinician's diagnosis of patients admitted to hospital with a first episode of schizophrenia. The patient's age, sex, social class, marital status, domiciliary status, and family history of psychiatric illness were recorded. A full medical history was taken and physical examination including the Northwick Park Brief Neurological Assessment (Owens & Johnstone, 1980) and Abnormal Involuntary Movements Scale (AIMS) (United States Department of Health, Education and Welfare, 1976) carried out.

As the study drew on patients from several centres attention had to be paid to comparability of symptom detection and clinical assessment among clinicians taking part. For this reason, within the first week of admission, patients were interviewed using the Present State Examination (PSE) (Wing *et al*, 1974). Several training sessions were held before and during the study. Because of continuing interest in diagnostic criteria for schizophrenia and in their usefulness in predicting outcome (Brockington *et al*, 1978) Feighner and Research Diagnostic Criteria (RDC) were also applied (Feighner *et al*, 1972; Spitzer *et al*, 1978) using information from the PSE interview. The presence of first-rank symptoms as described by Schneider (1974) was also assessed. Finally, the presence of a depressive syndrome was assessed by ignoring schizophrenic exclusion criteria within the RDC description of major depressive illness.

Results

Patient identification

Forty-nine patients entered the study. Knowledge of hospital catchment area populations, inception rates for schizophrenia (Cooper, 1978) and first admission rates in Scottish mental hospitals (Eagles & Whalley, 1985) suggested approximately half of first episode schizophrenics admitted to the participating units entered the study. Of the 49 patients, 47% were male, 53% female. Demographic and social data are shown in Table I. Males tended to be younger and more had never married ($\chi^2 = P < 0.05$). Feighner criteria applied to patients' relatives' case records suggested that four patients (8%) had a first degree relative with schizophrenia.

Twenty per cent ($n = 10$) had a history of possible neurological dysfunction (head injury in eight patients,

TABLE I
Demographic and social data

	Male (n=23)	*Female (n=26)*	*Total (n=49)*
Age: years mean	27.8	33.4	30.6
s.d.	(9.6)	(15.9)	(13.2)
Marital status: %			
Single	82	50	66
Married	9	15	12
Widowed	0	20	10
Divorced/separated	9	15	12
Social class of origin: %[1,2]			
I	0	4	2
II	17	12	15
III	35	50	43
IV	17	15	16
V	17	12	14
Unknown	14	7	10
Current social class: %[1]			
I	0	0	0
II	9	4	6
III	31	31	30
IV	17	23	21
V	17	0	8
Student	0	7	4
Housewife	—	20	10
Unemployed	26	15	21
Domiciliary status			
Lives with parents	52	46	47
Lives with spouse	13	12	11
Lives with other relatives	13	23	20
Lives alone	13	15	14
Other accommodation	9	4	8

1. Office of Population Censuses and Surveys, 1970
2. As assessed by father's most recent occupation

epileptic seizure aged 15 in one and polio as a child in another). Five patients reported sporadic cannabis use; a further patient may have taken LSD one year before admission.

Twenty per cent ($n=10$) had abnormal findings on neurological examination: equivocal or definitely abnormal plantar responses ($n=4$), equivocal difficulty in co-ordination ($n=2$), weakness in lower limbs ($n=1$), diminished tone in lower limbs ($n=1$) and increased ankle and knee jerks ($n=1$). The tenth patient with a history of polio had a mixture of neurological signs: A further three patients had abnormal involuntary movements as assessed by the AIMS global rating—one minimal, one mild and one severe; the two latter had had no antipsychotic medication in the 3 months before admission.

Patient categorisation

Catego system

Catego analysis of the PSE revealed three diagnostic groupings. These were Class S+, which contains central schizophrenic symptoms (disorders of possession of thought, delusions of control and voices discussing patient in third person or commenting on thoughts/actions), Class P+, a paranoid psychosis containing other than first-rank delusions and non-auditory hallucinations, and Class O+, other psychoses where the chief features are catatonic symptoms and behaviour which indicates hallucinations. Thirty-four patients could be given Catego classes with certainty (S+ =30, P+ =1, O+ =3) corresponding to ICD-9 codings of 295.3, 297.9 and 296.2 respectively (WHO, 1978). For 15 patients Catego classification was uncertain (S? =3, P? =5, O? =7).

First-rank symptoms

Of the 49 patients, 26 (52%) had one or more first-rank symptoms. The most common was of delusions of control or of alien forces penetrating the mind, with 13 (26%) patients positively rated, followed by delusional perception (24%), diffusion of thought (24%), and auditory hallucinations in the third person (22%).

American diagnostic criteria

There were 33 definite and nine probable cases of schizophrenia diagnosed by RDC. This contrasts with 12 definite and three probable cases diagnosed by Feighner's criteria. Of the 34 patients who did not fulfil the Feighner criteria, 31 (91%) failed as they had not shown symptoms for at least 6 months.

Presence of a depressive syndrome

When RDC for major depressive disorder were used but criteria excluding patients exhibiting schizophrenic symptoms were ignored, 11 patients (22%) showed a clear-cut depressive syndrome.

Other symptomatology

Other findings culled from the PSE ratings demonstrate the degree to which the study population was distressed and disturbed. Fifty six per cent scored positively on three or more anxiety items and 42% were rated as showing social withdrawal. Almost half expressed irritability, and 18% had attempted suicide. Nearly a quarter were rated as being hostile or suspicious. Over half showed blunted or incongruous affect and a quarter had incoherent speech. There was impairment of insight in 90%. In a third of interviews, the rater noted serious doubt about the adequacy of the interview for rating purposes.

Discussion

The number of patients who entered the study over 2 years was small but was concentrated in four units. As probably about half of all first episode schizophrenics admitted to these units entered the study and as we have no reason to believe there was any systematic bias, the results are likely to be representative of the parent group of first episode schizophrenics. Half the patients were in Social Classes IV and V or were unemployed; perhaps downward social mobility has already begun by the first episode, as suggested elsewhere (Goldberg & Morrison, 1963).

The reliability of information obtained about previous neurological dysfunction is uncertain but a considerable number of patients reported previous head injury. Neurological assessment detected isolated abnormalities in 20% but in only one patient, with a history of polio, were there widespread signs. The significance of the neurological examination is as yet unclear; 'soft' neurological signs can be found in 10% of normal children (reviewed by Shaffer, 1978). If signs persist at future examinations, then they may indeed suggest a significant neurological deficit now believed to exist in chronic schizophrenics (reviewed by Manschreck & Ames, 1984).

If a rating of mild on the AIMS global scale can be taken as definite evidence of abnormal involuntary movements, then two patients had this disorder. Controversy about the aetiology of such movements in schizophrenia continues but as both patients were almost certainly neuroleptically naive, it is most unlikely they were drug induced. Abnormal movements have long been described in schizophrenia (Kraepelin, 1919).

With regard to the PSE, concern about the adequacy of a third of interviews for rating purposes might seem to cast doubt on the reliability of the findings. However, there is ample evidence that this was a disturbed psychotic group of patients. The problem lies in the difficulty of reconciling the cerebral and reliable style of the PSE with the incoherence of psychosis.

The distribution of patients into the three Catego classes is broadly similar to that found in the WHO International Pilot Study of Schizophrenia (WHO, 1973; Wing *et al*, 1974). This suggests that clinicians in the present study entered patients as suffering from schizophrenia on the basis of a clear-cut illness. This is especially true given that the threshold of rating for symptoms in the PSE is high, compared to normal clinical practice.

The proportion of patients exhibiting one or more first-rank symptoms is in keeping with that found in earlier studies (reviewed by Lewine *et al*, 1982). However, there was a difference in frequency of particular symptoms between the present study and that of Lewine *et al*; delusional perception (primary delusions) was more common in the former study, disorders of possession of thought more common in the latter. This may be due in part to the fact that the latter population was an unselected group of schizophrenic admissions, and not first episode patients; primary delusions may become overlaid during subsequent admissions. The difference between the two populations for disorders of possession of thought is less easily explained, but perhaps bears out comments made by Mellor (1982) who criticised a facile approach to the rating of first rank symptoms and the attribution of too much weight to their presence for clinical diagnosis of schizophrenia.

Feighner's criteria are probably excessively strict; one study (Brockington *et al*, 1978) found only 10 out of 119 schizophrenic patients met the criteria; the results reported here would bear this out. While the RDC are less stringent, they are research criteria and would exclude almost a quarter of the study population, already shown to be clearly psychotic. This serves as a reminder that findings from a strictly defined research population may not be immediately generalisable to the clinical population. DSM III (American Psychiatric Association, 1980) is derived in part from these two sets of criteria, and is likely to reduce the number of patients diagnosed as suffering from schizophrenia (Mukherjee, 1983). The present Scottish evidence suggests it will be an excessive reduction.

The nature of 'depression' in schizophrenia has been debated recently (Johnson, 1981; Hirsch, 1982; Galdi, 1983). There is no doubt that symptoms commonly found in depressive illness occur in schizophrenic patients. In the absence of external validating criteria for the presence of depressive illness no study such as the present work can decide how often a 'proper' depressive illness has occurred. However, when criteria required to be met before depressive illness can be diagnosed in other contexts are applied, only a few patients meet the criteria. These patients had not yet started treatment and therefore there is no question of a pharmacogenic depressive process.

In conclusion, it can be said that the patients entering the study were ill, would have been recognised as such by clinicians, and would have been diagnosed as suffering from schizophrenia.

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The Scottish First Episode Schizophrenia Study

II. Treatment: Pimozide Versus Flupenthixol

THE SCOTTISH SCHIZOPHRENIA RESEARCH GROUP

In a randomised double-blind study, 46 first episode schizophrenics were given pimozide or flupenthixol for up to 5 weeks; the mean daily dose at the end was 18.8 mg pimozide and 20 mg flupenthixol. There were no significant between-drug differences in the effect on mental state or behaviour; positive schizophrenic symptoms responded to treatment, negative symptoms did not. Most patients required anti-Parkinsonian medication. Poor response was associated with 'organicity'. A switch to maintenance therapy after 5 weeks, the outcome of successful treatment and patient cooperation, was achieved in only 54%.

There was no relationship between plasma prolactin levels and severity of schizophrenic symptoms in unmedicated patients. Pimozide produced greater elevation of plasma prolactin. Plasma neuroleptic levels showed drug compliance was satisfactory throughout in only 54%.

Pimozide, a member of the diphenylbutylpiperidine group of neuroleptics has been used mainly as maintenance therapy in chronic schizophrenia (e.g. Falloon *et al*, 1978). Little attention has been paid to its possible antipsychotic effect in acute schizophrenics. There have only been four double-blind studies of pimozide in such patients all of which suggest it may be a useful anti-psychotic though two found it less effective than the comparison drug (Haas & Beckman, 1982; Chouinard & Annable, 1982; Pecknold *et al*, 1982; and Silverstone *et al*, 1984). The present study is the largest and most comprehensive to date as it examines not only pimozide's clinical efficacy but also plasma prolactin and neuroleptic levels and drug compliance. It compares pimozide with flupenthixol, a drug shown in a placebo controlled double-blind study to be an effective antipsychotic (Johnstone *et al*, 1978).

The study also examines the relationship in unmedicated patients between severity of schizophrenic symptoms and plasma prolactin levels, to investigate further the dopamine hypothesis of schizophrenia (Carlsson, 1977).

Method

Patients and medication

Of the 49 patients who entered the first episode schizophrenia study (see Part I), 46 took part in the drug study: 21 males, 25 females, mean age 30.6 years (range 16–68 years).

In the 3 months before hospital admission 26% had received small doses of thioridazine or chlorpromazine; the total dose ranged from 50–1400 mg. Up to 7 days after admission patients could receive droperidol for severe behavioural disturbance. Patients were then randomly and blindly allocated to receive in a double-dummy design pimozide or flupenthixol. The starting dose of both was 10 mg daily and could be increased to a maximum of 40 mg pimozide and 50 mg flupenthixol. Treatment lasted 5 weeks after which time, or before if patients had improved sufficiently, they were switched to maintenance therapy. If in the clinician's opinion a patient had not improved by then he was given in addition electroconvulsive treatment (ECT) or another neuroleptic. Also, patients did not receive maintenance therapy if there was no clear rise in plasma prolactin during treatment (see below).

During treatment with pimozide or flupenthixol, patients could receive droperidol for severe behavioural deterioration, an anti-Parkinsonian drug, procyclidine, and an hypnotic, temazepam.

Assessment

On admission to hospital, immediately before starting treatment with pimozide or flupenthixol ('Baseline') and then at weekly intervals, patients' mental state and ward behaviour were assessed by the Krawiecka and Wing Scales respectively (Wing, 1961; Krawiecka *et al*, 1977). One item on the Krawiecka Scale, 'affect', was divided into inappropriate and blunted affect. Parkinsonism and abnormal movements were also measured (Simpson & Angus, 1970; US Department of Health, Education and Welfare, 1976).

Blood samples were taken at regular intervals to measure plasma prolactin, pimozide and flupenthixol levels estimated by specific radioimmunoassay techniques (Prolk-PR commercial kit [CIS UK Ltd]; Michiels *et al*, 1975; FPT-RIA-200 commercial kit [IRE Ltd]).

Differences between the two drug groups were tested by independent t-tests; paired t-tests were used to measure differences within groups over time. Correlations between groups were measured by the Product Moment Correlation Coefficient. The χ^2 test was used to measure different frequencies in discrete categories.

Results

Patients on pimozide and flupenthixol were well matched. There were no statistically significant between-group differences in age and sex distribution, family history of schizophrenia, categorisation of schizophrenia, intelligence quotients, prevalence of neurological signs, and severity of illness at baseline, as measured by the Krawiecka and Wing Scales.

Of the 23 patients who received pimozide, 15 completed 5 weeks of treatment and a further three were switched to maintenance therapy before the end of 5 weeks. Three refused to continue with drug treatment; one discharged himself against medical advice; and one developed a skin rash. The mean daily dose at the end of week 5 was 18.8 mg.

Of the 23 patients who received flupenthixol, 17 completed 5 weeks of treatment and three switched to maintenance therapy before the end of 5 weeks. One patient was discharged well without any significant elevation in plasma prolactin level, one was discharged well but not given maintenance therapy as she left Scotland, and one discharged himself against medical advice. The mean daily dose at the end of week 5 was 20.0 mg.

Mental state (Figs 1 and 2)

In both pimozide and flupenthixol groups there was during the admission period a statistically significant reduction in Krawiecka total scores indicating an improvement in mental state ($t = 3.23$, $P < 0.01$ and $t = 2.51$, $P < 0.02$ respectively). Such improvement continued during drug treatment (baseline compared with subsequent weekly scores: t ranged from 3.67 to 7.99; P from <0.01 to <0.001). In both pimozide and flupenthixol groups, positive schizophrenic symptoms (inappropriate affect, incoherence, delusions and hallucinations) improved significantly during the admission period ($t = 3.40$, $P < 0.01$ and $t = 2.73$, $P < 0.02$ respectively) and drug treatment (t ranged from 3.02 to 7.32 P from <0.01 to $<0.0.001$). Negative symptoms (blunting of affect and poverty of thought) did not change in either group during admission or treatment periods. Non-schizophrenic symptoms did not change in either group during the admission period, but were significantly less when compared with baseline scores at the end of week 1 in the flupenthixol group ($t = 2.30$, $P < 0.05$) and week 5 in the pimozide group, ($t = 2.60$, $P < 0.05$).

There were no significant between-group differences on admission, immediately before, during or at the end of treatment in total, positive schizophrenic, negative schizophrenic, and non-schizophrenic symptom scores (independent t-tests).

Behaviour (Figs 1 and 2)

In both drug groups there was no significant change in total Wing scores during the admission period. In both groups there was a statistically significant reduction in scores during treatment indicating improvement in behaviour, evident in both groups by the end of week 2 (pimozide: $t = 3.56$, $P < 0.01$; flupenthixol: $t = 2.29$, $P < 0.05$). Embarrassing behaviour and social withdrawal had improved significantly by the end of week 2 in the pimozide group ($t = 2.99$, $P < 0.01$; $t = 2.55$, $P < 0.02$) and by weeks 1 and 3 in the flupenthixol group ($t = 2.19$, 2.57; both $P < 0.05$).

There were no statistically significant between-group differences at admission, immediately before, during or at the end of treatment in total, socially embarrassing and social withdrawal scores.

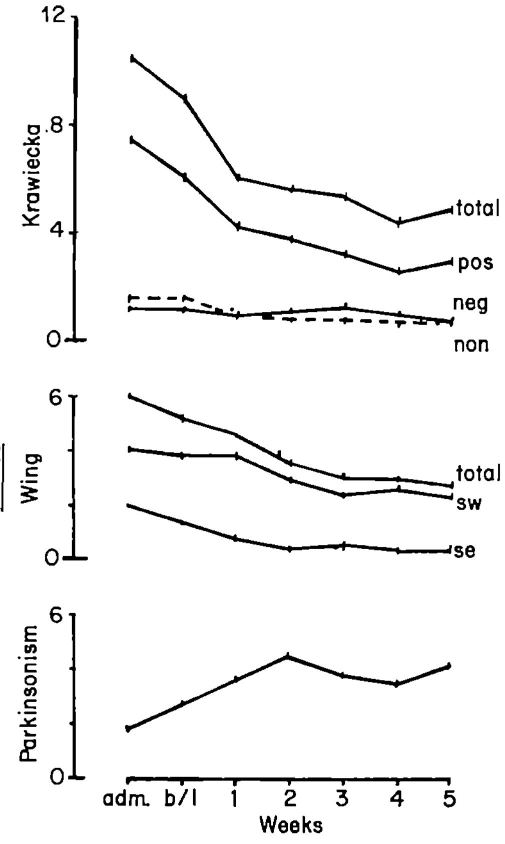

FIG 1. *Pimozide Group.* Weekly mean Krawiecka (mental state), Wing (behaviour) and parkinsonism scores. POS = positive schizophrenic symptom scores; NEG = negative schizophrenic symptom scores; NON = non-schizophrenic symptom scores; SW = social withdrawal scores; SE = socially embarrassing behaviour scores; Adm = admission; b/l = baseline.

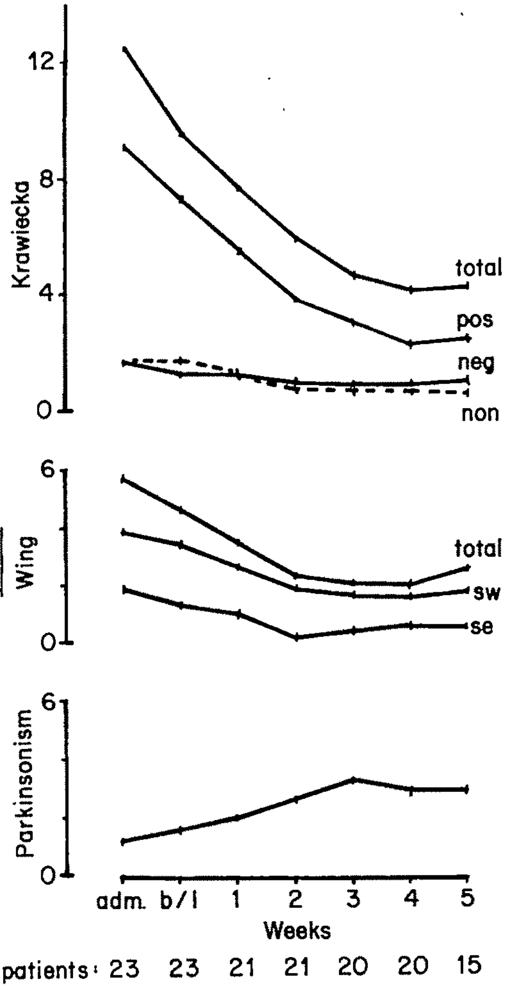

FIG. 2. *Flupenthixol Group.* Weekly mean Krawiecka (mental state), Wing (behaviour) and Parkinonism scores. POS = positive schizophrenic symptom scores; NEG = negative schizophrenic symptom scores; NON = non-schizophrenic symptom scores; SW = social withdrawal scores; SE = socially embarrasing behaviour scores; Adm = admission; b/l = baseline.

Parkinsonism (Figs 1 and 2)

Changes in Simpson and Angus scores are hard to interpret as many patients were quickly prescribed anti-Parkinsonian drugs (see below). In both groups there were no significant changes in the admission period. In the flupenthixol group there was a statistically significant increase in Parkinsonism ($t = 3.11$, $P < 0.01$) by the end of week 2, which persisted throughout the drug period. In the pimozide group scores rose during drug treatment but not significantly.

By the end of the first week of treatment, 57% of patients on pimozide and 35% on flupenthixol were receiving procyclidine; by the end of 5 weeks, these figures had risen respectively to 78% and 85%. There were no significant between-group differences in numbers of patients receiving anti-Parkinsonian medication (χ^2 test).

Abnormal movements

In the admission period three patients had abnormal movements as assessed by the Abnormal Involuntary Movements Scale (AIMS) (see Part I); these movements disappeared during treatment. One patient on flupenthixol and one on pimozide developed respectively mild and moderate movements during treatment.

Droperidol

In the admission period, 43% and 52% of patients in the pimozide and flupenthixol groups respectively required at least one dose of droperidol. Improvement in patients' mental state during this period was not due to droperidol as there were no significant differences between droperidol and non-droperidol groups in mental state scores, at admission or baseline. Numbers of patients receiving droperidol fell sharply during the weeks of treatment and in neither group were they ever greater than 20%. There were no significant between-group differences in numbers of patients requiring droperidol.

Plasma prolactin and neuroleptic levels (Fig. 3)

Virtually complete plasma prolactin and neuroleptic results were obtained in 12 patients on pimozide and 10 on flupenthixol. The pretreatment prolactin level was taken to be the mean of three levels—two during the admission period and one immediately before the administration of the first dose of pimozide or flupenthixol. Pimozide produced greater elevation of plasma prolactin, the level of which was significantly higher than in the flupenthixol group by the end of week 2 ($t = 2.29$, $P < 0.05$). There were no significant between-sex differences in plasma prolactin either at the end of week 1 or 5.

Product moment correlations between plasma prolactin, plasma neuroleptic, and neuroleptic dose were weakly positive but only occasionally reached statistical significance in some weeks when plasma neuroleptic levels were compared with dose and prolactin. Correlations were lowest between dose and plasma prolactin.

Compliance

In the 22 patients in whom virtually full pharmacokinetic data were available, compliance was estimated by examination of weekly neuroleptic levels. When a patient remained at the same dose level for more than 1 week, if subsequent neuroleptic levels were within 10% of the first level, compliance was 'good'. If a subsequent level was 10–50% lower than the first, compliance was 'doubtful' and, if it was less than 50%, it was 'poor'. Using these criteria, compliance in 54% of patients was good throughout the treatment period, doubtful on at least one occasion in 23% and poor on at least one occasion in 23%.

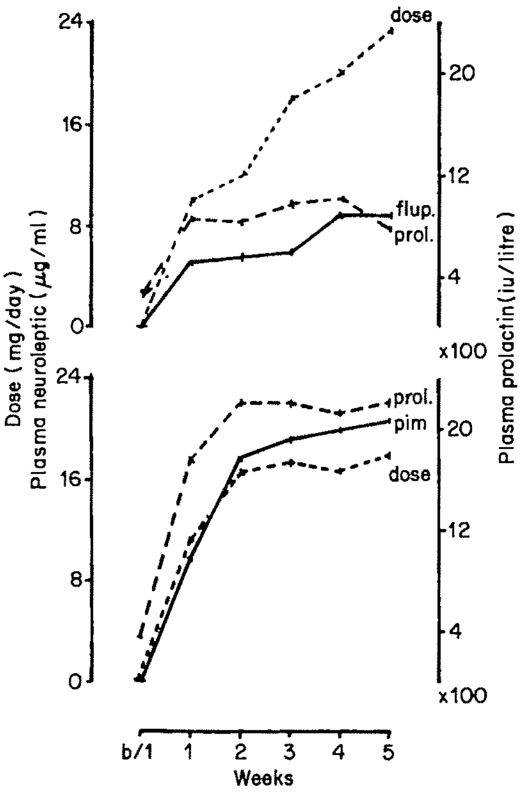

FIG. 3. Weekly mean neuroleptic dose, plasma neuroleptic level, and plasma prolactin level. The upper graph refers to the flupenthixol group, ($n = 10$) the lower to the pimozide group ($n = 12$). FLUP = plasma flupenthixol level; PROL = plasma prolactin level; PIM = plasma pimozide level; b/l = baseline.

Responders vs non-responders

At the end of treatment, patients were divided into three groups; these comprised 'responders' to pimozide or flupenthixol (63%) of whom 25 started but four refused maintenance therapy, 'non-responders' (17%) who received further treatment, either ECT or another neuroleptic, and 'non-completers', who did not proceed to maintenance therapy but could not clearly be said to be 'non-responders' such as those who were discharged prematurely against medical advice (20%). As results with pimozide and flupenthixol were broadly similar, the two neuroleptic groups were combined and 'responders' compared with 'non-responders'. The number of 'non-responders' was too small to produce statistically significant differences but the observation was that more 'non-responders' were male (63% *vs* 31%), and had neurological signs (50% *vs* 17%), a greater mean Progressive matrices/Mill Hill Vocabulary discrepancy (8 points *vs* 3 points) and higher mean negative schizophrenic score (2.1 *vs* 1.2). There was no relationship between lack of response and poor drug compliance.

Prolactin and schizophrenic symptoms

A small group of patients ($n = 9$) fulfilled the following criteria: they had a diagnosis of definite or probable schizophrenia, based on the RDC; had received no anti-psychotic medication in the 3 months prior to admission nor droperidol during the admission week; and had a baseline plasma prolactin level. There were no significant correlations between such prolactin levels and baseline scores on the Krawiecka scale, either total scores ($r = +0.04$) or positive schizophrenic symptom scores ($r = +0.12$).

Discussion

The study was not placebo controlled and no doubt non-drug factors contributed to patients' improvement; during the admission (pre-drug treatment) period there was a significant improvement in positive schizophrenic symptoms. It is unlikely, however, that there was no subsequent drug effect as commercial flupenthixol, used in the present study, contains 50% α flupenthixol, which has been shown to be more effective than placebo in acute schizophrenics (Johnstone *et al*, 1978).

Pimozide is an effective anti-psychotic in acute schizophrenia. It and flupenthixol are broadly similar in efficacy, especially in the speed of reduction of positive schizophrenic symptoms. Neither drug had any appreciable effect on negative schizophrenic symptoms.

The final mean daily dose of pimozide, 18.8 mg, was at the lower end of the dose range in other previously quoted controlled studies, 20–74.5 mg; the mean dose of flupenthixol, 20 mg, was just double that of α flupenthixol (9 mg) in the study quoted above. Probably one reason why clinicians did not prescribe higher doses was the rapid development of Parkinsonism which by the end of the study resulted in most patients in both groups receiving procyclidine.

Neuroleptic dose and plasma prolactin and neuroleptic levels correlated positively but weakly. Prolactin elevation was evident in both groups by the end of the first week but pimozide had a greater effect. This is of doubtful clinical significance as there is no clear relationship between plasma prolactin elevation and clinical efficacy (Kolakowska *et al*, 1980). Plasma neuroleptic estimations however enabled drug compliance to be assessed. In a bare majority of patients was compliance throughout the study period satisfactory. Perhaps the high prevalence of Parkinsonism was a contributory factor to doubtful or poor compliance as such side-effects have been shown in chronic schizophrenics to be associated with poor neuroleptic compliance (Van Putten, 1974).

The number of non-responders was small but 'organicity' as shown by neurological signs, greater performance/verbal discrepancy, and more negative symptoms was associated with a poor response.

The number of unmedicated patients in whom it was possible to correlate schizophrenic symptoms with plasma prolactin was also small, but the present study lends no support to the hypothesis that the schizophrenic process is mediated through dopamine over-activity. This is in contrast to previous work (Kleinman *et al*, 1982) which found an inverse relationship between positive symptoms and prolactin secretion but only in schizophrenics with normal computerised axial tomography (CAT) scans. Other work (Ferrier *et al*, 1984) found such a relationship to be weak. However, many other factors apart from dopamine can affect prolactin secretion.

It is noteworthy that for various reasons, especially lack of response and failure to cooperate, only 54% of patients could be started on maintenance therapy after 5 weeks. The course of the illness in both responders and non-responders after this first acute stage will be reported in later papers but it is already clear that we cannot be complacent about the present day treatment of first episode schizophrenia.

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The Scottish First Episode Schizophrenia Study

III. Cognitive Performance

THE SCOTTISH SCHIZOPHRENIA RESEARCH GROUP

Cognitive performance in 46 first episode schizophrenics was assessed within 1 week of admission to hospital by Progressive Matrices, Mill Hill Vocabulary Scale, Block Design and Similarities subtests of the Wechsler Adult Intelligence Scale, and Digit Copying Test. Patients' intellectual performance was at a dull normal level, just within one standard deviation from the mean. There was an association between the presence of anxiety and depression and lower scores on psychological tests. Patients assessed by the Present State Examination as belonging to the 'uncertain psychosis' category performed more poorly.

Early studies of cognitive functioning in chronic schizophrenics (reviewed by Matarazzo, 1972 and Heaton *et al*, 1978), found no characteristic pattern which differentiated them from patients with organic brain disease. Variables other than intelligence were better at separating the diagnoses. More recent work, however, has found a link in long-stay chronic schizophrenic in-patients between age disorientation, neurological deficit and cognitive impairment (Crow & Stevens, 1978; Owens & Johnstone, 1980; Liddle & Crow, 1984). The few studies of cognitive functioning in acute schizophrenics (reviewed by Payne, 1960, 1973) found them to be duller than average. Payne's conclusion was that psychosis produced a degree of general impairment, which was related to the severity of the illness and was probably progressive if the patient did not recover. Recovery was accompanied by a considerable improvement in general intellectual functioning. Another study, not reviewed by Payne (Foulds & Dixon, 1962*a*,*b*; Foulds *et al*, 1962) concluded that all types of schizophrenics showed intellectual impairment of a reversible kind and that impairment occurred mainly between the onset of illness and first admission to hospital. Any further decrement was brought about by age and was not mediated by being in hospital nor related to length of illness.

A more recent review (Aylward *et al*, 1984) found pre-morbid deficits were more common in men, a trend contrary to Foulds' finding that male schizophrenics had significantly better scores on both Matrices and Mill Hill Vocabulary.

The present study describes the cognitive performance of patients experiencing a first episode of schizophrenia using tests which allow direct comparison with the study of Foulds and reviews of Payne and Aylward. The relationship between cognitive performance and various measures of psychopathology is also examined.

Method

Forty-six out of 49 first episode schizophrenic in-patients (see Part I) were tested within 7 days of admission. Three patients were not tested because of failure to cooperate; some patients did not complete all the test battery. The tests given were Progressive Matrices (Raven, 1938), Mill Hill Vocabulary Scale (Raven, 1948) and the Block Design and Similarities subtests of the Wechsler Adult Intelligence Scale (Wechsler, 1955). A Digit Copying Test (Gibson & Kendrick, 1979) which is only modestly correlated with intelligence was included as a simple test of speed in performance.

Differences between groups were tested by t-tests or analysis of variance as appropriate; differences between means in the latter were tested by Tukey's "honestly significant difference" procedure (Zar, 1984). Correlation between groups was measured by the product moment correlation coefficient.

Results

Table I shows the scores obtained on the tests; for Matrices and Mill Hill Vocabulary means and standard deviations are shown for raw scores and for WAIS-type IQ scores, whilst for Block Design and Similarities they are shown for subtest scaled scores and pro-rated performance and verbal IQ scores respectively. Taken together, the tests indicate that patients performed at a dull normal level just within one standard deviation from the mean. The mean performance on Digit Copying was slightly higher than the mean score of 98.4 obtained by Kendrick's normal old people but was much more variable, the standard deviation being twice that of normals. Women performed better than men on Similarities ($t=2.15$, $P<0.05$) but not on other tests; this sex difference on Similarities ceased to be statistically significant when the presence of Krawiecka negative symptoms was taken into account (see below).

Means, but not standard deviations, for age, Matrices score and Mill Hill Vocabulary score in 90 patients who had been in hospital less than one year at the time of testing were extracted from Fould's study. The present group and this short stay subgroup of Foulds did not differ in mean age or Matrices score but differed on Vocabulary; Foulds' subgroup scored higher. If the present standard deviation is taken as a reasonable estimate of the variability in Foulds' patients the difference between means is statistically significant ($t=3.97$, $P<0.001$).

The four intelligence tests correlated significantly with each other and all except Mill Hill Vocabulary correlated significantly with Digit Copying. The correlations ranged from +0.53 to +0.70 and are similar to those reported in test manuals.

Intellectual status was associated with the Catego class derived from the Present State Examination (Wing *et al*, 1974) (Table II). The mean test scores for the 'Uncertain Psychosis' class were lower than those for 'Definite Psychosis' and 'Possible Neurosis' classes. Analysis of variance showed that only Similarities failed to show a statistically significant difference in the three classes. Tukey's procedure showed that the Uncertain Psychosis group scored significantly worse than Possible Neurosis patients on Vocabulary, Matrices and Block Design and significantly worse than the Definite Psychosis group on Matrices, Block Design and Digit Copying. The Possible Neurosis and Definite Psychosis groups did not differ significantly on any of the tests. The Uncertain Psychosis group had more non-schizophrenic symptoms on admission as assessed by the Krawiecka scale (see Part II) than the other two groups (F=6.78; *d.f.* 2, 45; $P<0.003$).

There was no relationship between positive schizophrenic symptoms on admission as assessed by the Krawiecka Scale and intellectual performance and only Similarities was significantly associated with negative schizophrenic symptoms ($r=-0.43$, $P<0.01$). There was a clear association between non-schizophrenic symptoms, in the main anxiety and depression, and lower scores on psychological tests (r ranged from -0.30 to -0.52; P ranged from <0.05 to <0.01); only Matrices did not correlate significantly with such symptoms.

Patients with significant signs of Parkinsonism on admission, that is, a score of more than 3 on the Simpson and Angus Scale (Simpson & Angus, 1970)

TABLE I
Scores on cognitive tests

Test	*Number of patients*	*Raw scaled scores Mean*	*s.d.*	*IQ scores Mean*	*s.d.*
Matrices	44	29.0	10.9	83.5	15.9
Mill Hill Vocabulary	43	41.4	9.7	90.0	7.8
Block Design	46	7.4	3.3	86.5	21.6
Similarities	46	8.7	2.8	93.2	16.4
Digit Copying	45	102.1	33.3	—	—

TABLE II
Cognitive performance and PSE Catego classes

	COGNITIVE TESTS									
	Matrices		*Vocabulary*		*Block Design*		*Similarities*		*Digit Copying*	
Catego groups	*No. of patients*	*Mean score*	*No. of patients*	*Mean score*	*No. of patients*	*Mean score*	*No. of patients*	*Mean score*	*No. of patients*	*Mean score*
Definite psychosis (S+, O+, P+)	29	86.9	28	90.0	31	8.0	31	9.1	31	109.2
Uncertain psychosis (S?, O?, P?)	11	71.2	11	85.7	11	4.6	11	7.2	11	79.9
Possible neurosis (O?/Various)	4	92.8	4	101.5	4	10.5	4	9.8	3	110.3
F ratio, *P* level	5.65	$P<0.01$	7.94	$P<0.01$	7.99	$P<0.01$	2.28	NS	3.62	$P<0.05$

performed less well on Digit Copying ($t=4.87$, $P<0.001$) and the WAIS subtests of Block Design and Similarities ($t=2.33$, 2.53 respectively: both $P<0.05$); however, when account was taken of the very poor performance on Digit Copying, a series of partial correlations showed that correlations between Parkinsonian symptoms and the two WAIS subtests diminished to near zero.

Some patients had received small amounts of neuroleptics before admission and others droperidol during the first week after admission (see Part II); there were no significant differences in any of the psychometric tests between those who had such drugs and those who had no antipsychotic medication before assessment.

Discussion

The results suggest the intellectual performance of newly admitted schizophrenic patients was below average, to a degree consistent with the studies summarised by Payne and Aylward. Verbal performance was lower than that reported by Foulds, but the reason for this is not clear. There was no marked sex difference in test performance, which is at odds with the trend reported by Aylward and the findings of Foulds. Similarly, there was no strong link between cognitive performance and neurological state. Patients with Parkinsonian symptoms performed very poorly on Digit Copying, probably because of slowness in both thought and action. This impairment also showed itself on Block Design and Similarities but detailed partial correlation analysis showed the correlation was due to slowness rather than intellectual impairment.

Test performance showed interesting links with various mental state assessments. Almost all tests correlated with Krawiecka non-schizophrenic symptom scores, in the main anxiety and depression. Such general emotional upset is known to impair performance over a wide range of psychological tasks. Anxiety and depression were greater in the Uncertain Psychosis group. This might explain the difference between that category and the other two on cognitive assessment; perhaps emotional disturbance interferes with PSE performance, perhaps schizophrenic patients of low intelligence describe less exact symptoms and more diffuse disturbance. Further study of the cohort over time might shed light on this association and point to links with the kind of deficit reported by Crow and colleagues.

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The Scottish First Episode Schizophrenia Study

IV. Psychiatric and Social Impact on Relatives

THE SCOTTISH SCHIZOPHRENIA RESEARCH GROUP

An assessment of 31 main care-giving relatives living with schizophrenics was carried out using the General Health Questionnaire and the Social Adjustment Scale by Self Report.

More than 75% of relatives had a high probability of themselves being a psychiatric case. Relatives also showed social role dysfunction and impairment especially marked in social and leisure activities. Relatives' distress was related to the level of symptoms in patients, as assessed by the Present State Examination.

The effect the family has on the schizophrenic patient has been widely studied (e.g. Brown *et al*, 1972; Vaughan & Leff, 1976; Falloon *et al*, 1982). However, there are few reports which have assessed the impact of the illness on close family members living with schizophrenics. Early studies assessed mainly the impact on the family of new community-based programmes of management by comparing the burden on relatives of schizophrenics treated in the community with those whose illness had been managed in hospital (Grad & Sainsbury, 1963, 1968). Relatives did indeed have high burden scores. Hoenig & Hamilton (1966) followed a cohort of schizophrenics admitted to hospital and subsequently discharged; 42% of relatives reported disruption in family life. In these early studies no reliable information was provided on relatives' psychiatric functioning.

In a recent study (Gibbons *et al*, 1984) almost one-third of relatives of schizophrenics who used the services of a district general hospital psychiatric unit in Southampton scored in the 'caseness' range of the General Health Questionnaire (Goldberg, 1978; Goldberg & Hillier, 1979). This indicates that they had a high probability of being psychiatric cases. The latter study examined schizophrenics at varying stages in their illness. The present study aims to assess the impact of the patient's illness during the first schizophrenic episode and to identify patient characteristics associated with relatives' distress.

Method

Population

Relatives had to live with the patient and be a main care-giver. This was operationally defined as the spouse of a married patient, the parent of a single patient or failing this, an adult offspring. Thirty-nine patients lived with relatives of whom 31 were assessed—24 parents, five spouses and two adult offspring.

Assessment

Relatives were assessed by the General Health Questionnaire (GHQ-28) (Goldberg & Hillier, 1979) and the Social Adjustment Scale Self-Report (SAS-SR) (Weissman & Bothwell, 1976) during the first week of the patients' admission. The GHQ-28 assesses psychological distress; it provides a rating of severity and by means of a threshold score can divide a population into those with a high or low probability of being a psychiatric case (Goldberg, 1978). On the basis of Scottish studies (Hobbs *et al*, 1983; Livingston & Livingston, 1984) respondents who scored five or more were considered cases. The GHQ-28 has four subscales assessing somatic symptoms, anxiety/insomnia, social dysfunction and severe depression. The SAS-SR, a 54-item questionnaire completed by the relative, yields an overall adjustment score and a rating of seven role areas: work, social and leisure activities, functioning within the nuclear family, functioning within the extended family, and marital, parental and economic functioning. A higher score indicates poorer adjustment. Differences between groups were tested by independent *t*-tests; correlations between groups were measured by the product moment correlation coefficient.

Results

The mean score for relatives on the GHQ-28 was 10.81 (s.d. 7.61), well above the caseness threshold. Twenty-four relatives (77%) had scores above the threshold. The relatives' mean score was high on the anxiety/insomnia (9.45, s.d. 5.43), social dysfunction (9.45, s.d. 4.19) and somatic subscales (8.13, s.d. 5.10). GHQ total scores suggest a very high level of psychological distress, about three times the community norm (Goldberg *et al*, 1976; Finlay-Jones & Burvill, 1977).

Social adjustment scores for relatives when compared with the self-assessment of a normal community sample (Weissman *et al*, 1978) (Table I) were higher in all roles except marital and parental functioning. Relatives scored significantly worse on global adjustment ($t = 2.53$, $P < 0.02$) and social and leisure functioning ($t = 3.96$, $P < 0.01$) but better on parental functioning ($t = 3.62$, $P < 0.01$).

TABLE II

Comparison between adjustment of patients' relatives and normal community population

Social adjustment	*Schizophrenics' relatives*			*Community norms*[1]		
	n	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*
Global	31	1.75	0.46*	276	1.53	0.46
Work	25	1.52	1.18	399	1.40	0.46
Social and leisure	31	2.42	0.82**	482	1.83	0.52
Extended family	30	1.47	0.56	475	1.34	0.33
Marital	19	1.27	1.08	361	1.75	0.48
Parental	18	0.77	0.73*	276	1.40	0.42
Nuclear	26	1.59	0.92	464	1.46	0.58
Economic	31	1.58	1.06	Not available		

*$P<0.01$

**$P<0.001$

1. Taken from Weismann *et al* (1978)

The population was next divided into cases (a score of 5 or more on the GHQ-28) and non-cases (a score of 4 or less). Differences in scores between the two groups were statistically significant on all GHQ subscales, cases performing less well (P values ranged from <0.002 to <0.0001); the greatest difference was in anxiety/insomnia ($t=5.22$, $P<0.0001$). Probable psychiatric cases had poorer social adjustment. Difference between the two groups were statistically significant in functioning in the nuclear family ($t=2.44$, $P<0.02$) and in work ($t=2.57$, $P<0.01$).

Relatives' psychological functioning was compared with patients' total scores and subscores on the Present State Examination (PSE) (Wing *et al*, 1974; Wing & Sturt, 1978). The subscores were for the following PSE syndromes: delusional and hallucinatory syndrome, behaviour, speech and other syndromes; specific neurotic syndromes, and non-specific neurotic syndromes. Statistically significant positive correlations were found between total PSE scores and both GHQ and social adjustment total scores ($r=+0.50$, $P<0.005$ and $r=+0.45$, $P<0.01$ respectively). Non-specific neurotic syndrome scores correlated significantly with GHQ total scores ($r=+0.40$, $P<0.03$), delusional and hallucinatory syndrome scores with social adjustment total scores ($r=+0.42$, $P<0.02$).

Relatives' psychosocial distress did not correlate significantly with patients' scores on Krawiecka and Wing Scales (Wing, 1961; Krawiecka *et al*, 1977) nor was it related to Research Diagnostic Criteria or Feighner categorisations (Feighner *et al*, 1972; Spitzer *et al*, 1978). A family history of psychiatric disorder was not associated with relatives' functioning nor was relationship to the patient.

Discussion

We believe this to be the first study to report the impact on relatives of a first episode of schizophrenia. The results demonstrate a very high incidence of psychological distress in relatives with anxiety-based symptoms the most prominent feature. The severity and frequency of such distress were far higher than in the Southampton study (Gibbons *et al*, 1984) but relatives were seen during a period of crisis, the first admission of the patient to a psychiatric hospital. In Southampton, patients had been ill longer; probably relatives had adapted over a longer period of time. Also, self-selection of relatives may indicate bias. For example, the most concerned relative may have visited the patient most frequently after admission and thus found himself identified as the main care-giver. Levels of disturbance in the present study were higher than in other groups of relatives. For example, 3 months after severe head injury 57% of relatives scored within the caseness range of the GHQ but only 10% of relatives of the mildly injured did so (Livingston *et al*, 1985*a*). Approximately 65% of supporters of the elderly mentally infirm had similar GHQ scores (Gilleard *et al*, 1984). The higher levels of disturbance in relatives in the present study may be partly explained by the time of assessment.

Social adjustment was also impaired with global adjustment and social and leisure functioning being significantly worse than in a community sample. Also, this group of relatives had poorer global social adjustment than the relatives of the severely head injured (Livingston *et al*, 1985*b*). However, relatives functioned better in their role as parents or marital partners than the comparison group. Perhaps this is because relatives saw themselves as striving hard in these roles.

Relatives who were cases, as defined by the GHQ-28, had higher scores on all GHQ subscales and performed less well at work and as part of the nuclear family. However, there were marked trends towards poorer functioning in all roles with the exception of economic functioning.

Relatives' distress was related to symptoms in patients as assessed by the PSE. The correlation between total PSE and total GHQ scores was accounted for mainly by neurotic symptoms in the patient; in contrast, correlation with social adjustment total scores was greater in those with florid psychotic symptoms. This might suggest that anxiety in the patient produces anxiety in the relative, whereas psychotic symptoms bring about an adjustment in the relatives' social life. A similar finding, namely that personal distress in relatives was associated with patients' emotional lability rather than active disturbed behaviour was found in demented day patients (Greene *et al*, 1982).

Psychiatric disorder is common in the community (Goldberg *et al*, 1976; Finlay-Jones and Burvill, 1977) but such disorders are usually brief and remit spontaneously. Planned follow-up of relatives in the present study will indicate whether the very high level of psychosocial distress in these patient supporters persists as it did with the relatives of head injured patients for at least 1 year (Livingston *et al*, 1985*b*). The evolution of social functioning is less well known but it is likely it will follow the course of the relatives' psychiatric functioning.

In conclusion, the results in the present study suggest involvement with families should start from the first admission of the patient to hospital, not only for the needs of the patient but also for those of relatives.

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References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders*, Third Revision, Washington DC: American Psychiatric Association.

Aylward, E., Walker, E. & Bettes, B. (1984) Intelligence in schizophrenia: meta-analysis of the research. *Schizophrenia Bulletin*, **10**, 430–459.

Brockington, I. F., Kendell, R. E. & Leff, J. P. (1978) Definitions of schizophrenia: concordance and prediction of outcome. *Psychological Medicine*, **8**, 387–398.

Brown, G. W., Birley, J. T. L. & Wing, J. K. (1972) The influence of family life on the course of schizophrenic disorders: a replication. *British Journal of Psychiatry*, **121**, 241–248.

Carlsson, A. (1977) Does dopamine play a role in schizophrenia? *Psychological Medicine*, **7**, 583–597.

Chouinard, G. & Annable, L. (1982) Pimozide in the treatment of newly admitted schizophrenic patients. *Psychopharmacology*, **76**, 13–19.

Cooper, B. (1978) Epidemiology. In *Schizophrenia: Towards a New Synthesis* (ed. J. K. Wing). London: Academic Press.

Crow, T. J. & Stevens, M. (1978) Age disorientation in chronic schizophrenia: the nature of the cognitive deficit. *British Journal of Psychiatry*, **133**, 137–142.

Eagles, J. M. & Whalley, L. J. (1985) Decline in the diagnosis of schizophrenia among first admissions to Scottish mental hospitals from 1969–78. *British Journal of Psychiatry*, **146**, 151–154.

Falloon, I. R. H., Watt, D. C. & Shepherd, M. (1978) A comparative controlled trial of pimozide and fluphenazine decanoate in the continuation therapy of schizophrenia. *Psychological Medicine*, **8**, 59–70.

——, Boyd, J. L., McGill, C. W., Razani, J., Moss, H. B. & Gilderman, E. M. (1982) Family management in the prevention and exacerbation of schizophrenia: a controlled study. *New England Journal of Medicine*, **306**, 1437–1440.

Feighner, J. P., Robins, E., Guze, S., Woodruff, R. A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–62.

Ferrier, I. N., Johnstone, E. C. & Crow, T. J. (1984) Hormonal effects of apomorphine in schizophrenia. *British Journal of Psychiatry*, **144**, 349–358.

Finlay-Jones, R. A. & Burvill, P. W. (1977) The prevalence of psychiatric morbidity in the community. *Psychological Medicine*, **7**, 474–489.

Foulds, G. A. & Dixon, P. (1962*a*) The nature of intellectual deficit in schizophrenia. Part I: a comparison of schizophrenics and neurotics. *British Journal of Social and Clinical Psychology*, **1**, 7–19.

—— & Dixon, P. (1962*b*) The nature of intellectual deficit in schizophrenia. Part III: a longitudinal study of the sub-groups. *British Journal of Social and Clinical Psychology*, **1**, 199–207.

——, Dixon, M. & McClelland, W. J. (1962) The nature of intellectual impairment in schizophrenia. Part II: a cross-sectional study of paranoid, catatonic, hebephrenic and simple schizophrenics. *British Journal of Social and Clinical Psychology*, **1**, 141–149.

Galdi, J. (1983) The causality of depression in schizophrenia. *British Journal of Psychiatry*, **142**, 621–625.

Gibbons, J. S., Horn, S. H., Powell, J. M. & Gibbons, J. L. (1984) Schizophrenic patients and their families: a survey in a psychiatric service based on a District General Hospital Unit. *British Journal of Psychiatry*, **144**, 70–77.

Gibson, A. J. & Kendrick, D. C. (1979) *The Kendrick Battery for the Detection of Dementia in the Elderly*. Windsor: NFEF-Nelson.

Gilleard, C. J., Belford, H., Gilleard, E., Whittick, J. E. & Gledhill, K. (1984) Emotional distress among the supporters of the elderly mentally infirm. *British Journal of Psychiatry*, **145**, 172–177.

Goldberg, D. (1978) *Manual of the General Health Questionnaire*. Windsor: NFER.

——, Kay, C. & Thompson, L. (1976) Psychiatric morbidity in general practice and the community. *Psychological Medicine*, **6**, 565–569.

—— & Hillier, V. F. (1979) A scaled version of the General Health Questionnaire. *Psychological Medicine*, **9**, 139–146.

Goldberg, E. M. & Morrison, S. L. (1963) Schizophrenia and social class. *British Journal of Psychiatry*, **109**, 785–802.

Grad, J. & Sainsbury, P. (1963) Mental illness in the family. *Lancet*, **i**, 544–547.

—— & Sainsbury, P. (1968) The effects that patients have on their families in a community care and control psychiatric service: a two-year follow-up. *British Journal of Psychiatry*, **114**, 265–278.

Greene, J. G., Smith, R., Gardiner, M. & Timbury, G. C. (1982) Measuring behavioural disturbance of elderly demented patients in the community and its effect on relatives. *Age and Ageing*, **11**, 121–126.

Haas, S. & Beckman, H. (1982) Pimozide versus haloperidol in acute schizophrenia. A double-blind controlled study. *Pharmacopsychiatria*, **15**, 70–74.

Heaton, R., Baade, L. & Johnson, K. (1978) Neuropsychological test results associated with psychiatric disorders in adults. *Psychological Bulletin*, **85**, 141–162.

Hirsch, S. (1982) Depression 'revealed' in schizophrenia. *British Journal of Psychiatry*, **140**, 421–424.

Hobbs, P., Ballinger, C. B. & Smith, A. H. W. (1983) Factor analysis and validation of the General Health Questionnaire on females: a general practice survey. *British Journal of Psychiatry*, **142**, 257–264.

Hoenig, J. & Hamilton, M. W. (1966) The schizophrenic patient in the community and his effect on the household. *International Journal of Social Psychiatry*, **12**, 165–176.

Johnson, D. A. W. (1981) Studies of depressive symptoms in schizophrenia. *British Journal of Psychiatry*, **139**, 89–101.

Johnstone, E. C., Crow, T. J., Frith, C. D., Carney, M. W. P. & Price, J. S. (1978) Mechanism of the anti-psychotic effect in the treatment of acute schizophrenia. *Lancet*, **i**, 848–850.

Kleinman, J. E., Weinberger, D. R., Rogol, A. D., Bigelow, L. B., Klein, S. J., Gillin, C. & Watt, R. J. (1982) Plasma prolactin concentrations and psychopathology in chronic schizophrenia. *Archives of General Psychiatry*, **39**, 655–657.

Kolakowska, T., Gelder, M. G. & Orr, M. W. (1980) Drug-related and illness-related factors in the outcome of chlorpromazine treatment: testing a model. *Psychological Medicine*, **10**, 335–343.

Kraepelin, E. (1919) *Dementia Praecox and Paraphrenia* (translated by R. M. Barclay, ed. G. M. Robertson). Edinburgh: Livingstone.

Krawiecka, M., Goldberg, D. & Vaughn, M. (1977) A standardised psychiatric assessment scale for rating psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

Lewine, R., Renders, R., Kirchhofer, M., Monsour, A. & Watt, N. (1982) The empirical heterogenicity of first rank symptoms in schizophrenia. *British Journal of Psychiatry*, **140**, 498–502.

Liddle, P. F. & Crow, T. J. (1984) Age disorientation in chronic schizophrenia is associated with global intellectual impairment. *British Journal of Psychiatry*, **144**, 193–199.

Livingston, M. G. & Livingston, H. (1984) Emotional distress in nurses at work. *British Journal of Medical Psychology*, **57**, 291–294.

——, Brooks, D. N. & Bond, M. R. (1985*a*) Three months after severe head injury: psychiatric and social impact on relatives. *Journal of Neurology, Neurosurgery and Psychiatry*, **48**, 870–875.

——, Brooks, D. N. & Bond, M. R. (1985*b*) Patient outcome in the year following severe head injury and relatives' psychiatric and social functioning. *Journal of Neurology, Neurosurgery and Psychiatry*, **48**, 876–881.

Manschreck, T. C. & Ames, D. (1984) Neurologic features and psychopathology in schizophrenic disorders. *Biological Psychiatry*, **19**, 703–719.

Matarazzo, J. D. (1972) *Wechsler's Measurement and Appraisal of Adult Intelligence*. Baltimore: Williams and Wilkins.

Mellor, C. S. (1982) The present status of first rank symptoms. *British Journal of Psychiatry*, **140**, 423–424.

Michiels, L. J. M., Heykants, J. J. P., Knaep, S. & Janssen, P. A. J. (1975) Radioimmunoassay of the neuroleptic drug pimozide. *Life Sciences*, **16**, 937–944.

Mukherjee, S. (1983) Reducing American diagnosis of schizophrenia — will the DSM III suffice? *British Journal of Psychiatry*, **142**, 414–418.

Office Of Population Censuses And Surveys (1970) *Classification of Occupations*. London: HMSO.

Owens, D. G. C. & Johnstone, E. C. (1980) The disabilities of chronic schizophrenia — their nature and the factors contributing to their development. *British Journal of Psychiatry*, **136**, 384–395.

Payne, R. W. (1960) Cognitive Abnormalities. In *Handbook of Abnormal Psychology* (ed. H. J. Eysenck). London: Pitman.

—— (1973) Cognitive Abnormalities. In *Handbook of Abnormal Psychology* (ed. H. J. Eysenck) 2nd ed. London: Pitman.

Pecknold, J. C., McClure, D. J., Allan, T. & Wrzesinski, L. (1982) Comparison of pimozide and chlorpromazine in acute schizophrenia. *Canadian Journal of Psychiatry*, **27**, 208–212.

Raven, J. C. (1938) *Guide to Using Progressive Matrices*. London: Lewis.

—— (1948) *Guide to Using the Mill Hill Vocabulary Scale with Progressive Matrices*. London: Lewis.

Schneider, K. (1974) Primary and Secondary symptoms in schizophrenia. In *Themes and Variations in European Psychiatry* (eds S. R. Hirsch and M. Shepherd). Bristol: J. Wright.

Shaffer, D. (1978) Soft neurological signs and later psychiatric disorder — a review. *Journal of Child Psychology and Psychiatry*, **19**, 63–65.

Silverstone, T., Cookson, J., Ball, R., Chin, C. N., Jacobs, D., Lader, S. & Gould, S. (1984) The relationship of dopamine receptor blockade to clinical response in schizophrenic patients treated with pimozide or haloperidol. *Journal of Psychiatric Research*, **18**, 255–268.

Simpson, G. M. & Angus, J. W. S. (1970) A rating scale for extra pyramidal side effects. *Acta Psychiatrica Scandinavica, Suppl. 212*, 11–19.

Spitzer, R., Endicott, J. & Robins, E. (1978) *Research Diagnostic Criteria Instrument, No. 58*. New York: New York State Psychiatric Institute.

United States Department Of Health, Education And Welfare (1976) *Abnormal Involuntary Movements Scale (AIMS)*. In *ECDEU Assessment Manual* (ed. W. Guy), pp. 534–537. Rockville, Maryland: US Dept. Health, Education and Welfare.

Van Putten, T. (1974) Why do schizophrenic patients refuse to take their drugs? *Archives of General Psychiatry*, 31, 67–72.

Vaughan, C. & Leff, J. P. (1976) The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, **129**, 125–137.

Wechsler, D. (1955) *Manual for the Wechsler Adult Intelligence Scale*. New York: The Psychological Corporation.

Weissman, M. M. & Bothwell, S. (1976) Assessment of social adjustment by patients' self-report. *Archives of General Psychiatry*, 33, 1111–1115.

——, Prusoff, B. A., Thompson, W. D., Harding, P. S. & Myers, J. (1978) Social adjustment by self-report in a community sample and in schizophrenic out-patients. *Journal of Nervous and Mental Diseases*, **166**, 317–326.

Wing, J. K. (1961) A simple and reliable sub-classification of schizophrenia. *Journal of Mental Science*, **107**, 862–875.

——, Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

—— & Sturt, E. (1978) The PSE-ID-Catego System: Supplementary Manual. MRC Social Psychiatry Unit, Institute of Psychiatry, London.

World Health Organisation (1973) *The International Pilot Study of Schizophrenia*, Vol. I. Geneva: WHO.

—— (1978) *Mental Disorders: Glossary and guide to their classification in accordance with the Ninth Revision of the International Classification of Diseases*. Geneva: WHO.

Zar, J. H. (1984) *Biostatistical Analysis*, 2nd ed. New Jersey: Prentice Hall.

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Five-year Outcome and Prognosis in Schizophrenia:

A Report from the London Field Research Centre of the International Pilot Study of Schizophrenia

R. PRUDO and H. MUNROE BLUM

A sample of 100 schizophrenic patients admitted to London area psychiatric hospitals were examined as part of the International Pilot Study of Schizophrenia. Clinical and social outcome were variable. At 5-year follow-up, 49% had good symptomatic outcome and 42%, good social outcome. Poor social functioning at inclusion was predictive of poor symptomatic outcome. Illness history (IH), occupational functioning (OF), social relationship functioning (SRF), negative and non-specific symptoms (NNS) at initial evaluation, and their interaction with sex and race accounted for 32% of the 5-year symptomatic outcome variance ($n=84$; $F=3.48$; $P<0.001$). OF, SRF, housing status, NNS and their interaction with sex, race and age accounted for 47% of the social outcome variance ($n=62$; $F=2.62$; $P<0.007$).

This paper examines the 5-year outcome and factors predictive of outcome in 100 schizophrenic patients admitted to London area psychiatric hospitals, reflecting data from the London Field Research Centre of the International Pilot Study of Schizophrenia (IPSS) located at the Medical Research Council (MRC) Social Psychiatric Unit, Institute of Psychiatry, Camberwell, London. The World Health Organisation (WHO) Collaborating Investigators at the London field research centre at inception included Dr J. K. Wing, Dr J. Birley and Dr J. P. Leff. The IPSS represents a WHO-sponsored transcultural psychiatric investigation of 1202 patients in nine countries—Columbia, Czechoslovakia, Denmark, India, Nigeria, Taiwan, the Soviet Union, the United States and the United Kingdom. The IPSS was carried out in three phases: the Preliminary Phase, the Initial Evaluation Plan and the Follow-up Phase. During the preliminary phase, administrative, operational and organisational procedures were established and tested. In the initial evaluation phase approximately 135 patients were selected and examined at each centre from among those patients contacting the centres during the 1-year period from 1 April 1968 to 1 April 1969. The follow-up phase had two major components. Two years following the initial evaluation of patients there was a follow-up evaluation, which was repeated 5 years after patients' inclusion in the study. This report is based on the initial evaluation and 5 year follow-up data. Because of limited resources, a comprehensive second year follow-up at the London centre was omitted.

The broad aims of WHO-sponsored IPSS were to tackle methodological problems relevant to a large scale international psychiatric study and to answer questions about the nature and distribution of schizophrenia. The basic questions were formulated as follows:

(a) In what sense can it be said that schizophrenic disorders exist in different parts of the world? Do they differ in form or content? Does the clinical course differ?
(b) Can other functional psychoses also be recognized and do they run a recognisably different course?
(c) Can techniques be developed for recording and classifying symptomatology reliably?
(d) Can teams of research workers be trained to use those techniques so that comparable observations can be made in both developed and developing countries?

It is beyond the scope of this paper to enlarge on these extensive goals except where relevant to 5 year outcome and prognosis. The reader is referred to Volume 1 of the Report of the IPSS (WHO, 1973) which gives a detailed account of the origins, objectives and design of this study, while the 2-year follow-up report (WHO, 1979) contains a comprehensive account of the overall results. The 5-year follow-up data will be presented in a future IPSS volume.

Although schizophrenia is perhaps generally regarded as an illness with poor outcome, a range of variability in outcome has long been recognized and many attempts have been made to identify various symptomatic, past history and sociodemographic factors that are highly correlated with good and bad outcome. The initial evaluation and the 5-year follow-up phases of the IPSS provide data which make it possible to consider the degree of variance in the outcome of schizophrenic patients that is explained by such predictors. An important advantage of the IPSS data over that used in many other studies of outcome and prognosis is that (a) it was collected in a prospective study; (b) both the initial evaluation and follow-up reassessment of psychopathology were carried out by well-trained investigators with the use of standardised research instruments; (c) the reliability of the clinical evaluation procedures was assessed both within and across research centres; and (d) the data relates to patients from nine different parts of the world, adding a cross-cultural dimension to the investigation of predictors. While the IPSS included for comparative purposes patients with diagnoses other than schizophrenia, this report is concerned with schizophrenic patients alone, since patients with other diagnoses in the UK sample form too small a group to analyse separately. Where results for outcome and factors predictive of outcome in the UK sample differ markedly from that of other IPSS centres, this will be indicated.

Most previous studies on prognosis have operated with unstandardised diagnostic concepts, and predictors and characteristics of outcome whose assessment is not directly replicable, making comparison of results from different studies difficult. Despite these difficulties it is possible to find broad areas of agreement between different studies and to derive hypotheses which could be tested on data from the 5-year follow-up phase of the IPSS London centre. Previous studies have suggested many predictors of outcome drawn from sociodemographic, past history, personality and psychopathology variables.

Many investigators have emphasised the roles of community acceptance, living arrangements, social relationships, and occupational and economic status in influencing how well a person with schizophrenia will do over time (Pasamanick *et al*, 1967; Fairweather *et al*, 1969; Stein *et al*, 1975; Lamb, 1978; Wallace, 1980). In general, findings have indicated that variables predict their like outcome functions. For example, Strauss & Carpenter (1974, 1977) in their analysis of the IPSS follow-up data from the Washington Centre found that employment status and quality of social relationships at inclusion were most predictive of 2 and 5-year follow-up occupational status and social relationships, respectively. This reflects the overall IPSS 2-year follow-up results demonstrating a role for social isolation and marital status in predicting outcome. Illness history has been identified as an important predictor of outcome in schizophrenia by a number of authors (Langfeldt, 1956; Vaillant, 1964). While there has been controversy over the predictive usefulness of subclasses of schizophrenia and presenting psychopathology (Langfeldt, 1956; Strauss & Carpenter, 1974; Hawk *et al*, 1975), there exists some evidence that 'positive' symptoms predict a good response to neuroleptic drugs, while the presence of 'negative' symptoms bodes poorly for long-term outcome irrespective of drug treatment (Mackay, 1980). Positive symptoms included: 'delusions present much of time', 'presence of delusions of grandeur', 'presence of hallucinations', 'hears thoughts aloud'. Negative and non-specific symptoms included: 'uninformed', 'recent change in concentration', 'restricted affect', 'poor rapport with interviewer', 'recent change in interests', 'poor insight'. Extrapolating from these findings one might expect that for a schizophrenic population receiving standard treatment procedures (including neuroleptic drugs), 'positive' symptoms would not be predictive of poor outcome while 'negative' symptoms might be. The results of the 2-year follow-up data from the IPSS, also suggests that negative and non-specific symptoms present at the time of inclusion were predictive of poor outcome (WHO, 1979).

Many prognosis studies tend to ignore evaluation of the interrelationships of multiple predictor variables and their combined influence on outcome. The assessment of potential confounders and interaction factors is often ignored. Investigation of the interrelationships of preselected independent variables and their cumulative effect on outcome with consideration given to potential confounders and interaction terms is indicated as evidenced in the IPSS 2-year follow-up results (WHO, 1979). The hypotheses addressed here include both those in which like inclusion variables were expected to predict like 5-year follow-up functions and those in which specific inclusion variables were expected to predict unlike 5-year follow-up functions. The questions addressed were: (1) What social, illness history and symptom factors will be related to or predictive of symptomatic outcome? (2) What social, illness history and symptom factors will be related to or predictive of social outcome? (3) What are the interrelationships of these predictor and outcome variables? It was hypothesised that poor inclusion social functioning, poor inclusion illness history and the presence of negative symptoms at inclusion would each be positively associated with poor symptomatic and social outcomes at 5-year follow-up. It was further hypothesised that inclusion 'positive' symptoms would not be associated with poor clinical and/or social outcomes at 5-year follow-up.

Method

Patient selection

This report is based on a prospective study of 100 schizophrenic patients examined at the London Field Research Centre. The initial patient sample was obtained by screening the case notes of all subjects with functional psychoses admitted to the Bethlem Royal and Maudsley Hospitals and to Cane Hill Hospital during the study period. The patients selected for the study were between the ages of 15 and 44 years. Exclusion criteria were chosen to screen out chronic patients and those who illness may have been caused or significantly influenced by an organic condition. Chronic patients were defined as those having total periods in hospital of 2 years or more in the previous 5 years, including re-admission, or severe psychotic symptoms in the episode of inclusion, probably continously present for more than 3 years.

Inclusion criteria

Inclusion criteria consisted of symptoms, such as hallucinations, whose presence automatically qualified the patient for inclusion regardless of the degree of symptomatology, and those, such as disorders of form of thinking, considered as a basis for inclusion only if present in severe degree. About one-third of the patients were local residents of Camberwell, a completely urban area, mostly inhabited by working class people but with a sizeable middle class population, while the majority of the remaining patients came from nearby districts. Patients were informed as to the nature of the study and participated voluntarily.

Rating schedules

Patients identified as being potentially suitable for inclusion were interviewed by experienced psychiatrists using a number of standardised ratings instruments. These included a semi-structured sign and symptom interview of established reliability, the 'Present State Examination' (PSE) (Wing *et al*, 1974) and a 'Psychiatric History (PH) and Social Description Schedule' (SD). The individual ratings on the PSE can be grouped to produce 35 syndrome scores. A computer program, CATEGO, has been developed to process the syndrome scores and leads to standardised clinical groupings. Additional information concerning the psychiatric history can also be expressed in a standard form and processed by CATEGO so that it contributes to a final diagnostic category (Wing *et al*, 1974). The PH and SD Schedules contain nine sections of psychiatric history items and twelve sections of social description items. The reliability of the PH and SD Schedules was less extensively documented than the PSE. Four main types of schedules were used at the 5-year follow-up point: the PSE, the Follow-Psychiatric History Schedule (FUPH), the Follow-up Social Description Schedule (FUSD) and the Follow-up Diagnostic Assessment Schedule (FUDA). The main purpose of the FUPH was to describe the course of the patient's illness in the interval between initial evaluation and the 5-year follow-up, while the main aim of the PUSD was to provide an account of the socioeconomic changes occurring to the patient since the initial evaluation. Areas covered included quality of housing, quality and type of work, education and social activities. The FUDA Schedule required the psychiatrist to make an overall diagnosis of the patient at the time of follow-up and to describe the findings. As well as interviews with the patient, information was obtained from family members, health records and health professionals. Following the initial screening assessment, suitable patients were given a clinical diagnosis by the interviewing psychiatrist based on all the available information at the initial evaluation and using the description and categories provided by the 'International Classification of Diseases—Eighth Revision (ICD-8)' (WHO, 1965). Of the 100 patients given a clinical diagnosis of schizophrenia at inclusion, 77% of the diagnoses were paranoid schizophrenia, 10% were hebephrenic schizophrenia, and 8% were schizo-affective. Using the CATEGO program, 79% of the patients given a clinical diagnosis of schizophrenia fell into the important 'Class S'. This class contains patients with at least one of Schneider's first-rank symptoms, most of which are considered to be highly discriminating for a diagnosis of schizophrenia. Follow-up interviews were conducted 5 years after admission to the study by one of the authors (RP). At this time the four follow-up schedules were administered.

Outcome criteria

The criteria used to assess outcome in schizophrenia have varied widely from study to study. Some investigators report 'global' assessments, while others stress either symptomatic improvement or social outcome. Recent studies have emphasized the need to view outcome as a multidimensional construct. Brown *et al* (1966), for example, pointed out that improvement within one individual area of functioning does not necessarily correlate with improvement in other areas; patients with, for example, extensive delusions may be able to work. In our paper both symptomatic and social outcome scales have been utilised as measures of outcome.

The symptomatic measures of outcome are based on the type, severity and duration of symptoms during the 5-year follow-up period. Four symptomatic outcome categories have been created, based on the total time patients experienced either florid episodes of major mental disorder or marked residual symptoms or defect state, warranting an ICD diagnostic label over the 5 years (Table I). Only disorders subsumed under the category 'Psychosis' (ICD-8) were included as major mental disorders. Mental disorders subsumed under the 'Neurosis' category and minor or occasional residual symptoms, such as delusional fragments or hallucinations, have not been taken into account in this outcome measure, with the exception of the best outcome category (Table I). While a variety of overall symptomatic outcome scales have been utilised in previous research, including length of hospitalisation during the follow-up period or symptom profiles in the month prior to follow-up, this new scale was preferred because it seemed to reflect a more complete picture of the symptomatic course during the 5-year follow-up period and more fully utilised the comprehensive information available to this study.

Social outcome categories have been created based on the assessment of three distinct areas utilising the IPSS social schedules: occupational functioning, social functioning and housing status in the year prior to follow-up. Eleven specific social outcome variables subsumed under these three categories were utilised and score 0 for good functioning and 1 or more for poor functioning, and these items summed to create major variables, such that a low score reflects good functioning and a high score poor functioning (Appendix I).

Patients

At the time of inclusion into this study the patients were predominantly male (64%), young (71% in the age range 15–34 years), and unmarried (81%). Thirty-four percent had no psychiatric history prior to their inclusion in this study, whilst about half (53%) of the remaining 66 patients had a psychiatric history of less than 1 year. Thus, the majority of the patients (70%) had either no prior psychiatric history or a history of less than 1 year.

Nineteen percent of the patient population was non-white (12% of West Indian origin and 7% other), the great majority of whom were born overseas. Comparison of white and non-white subjects by age, sex and marital status revealed no significant differences. The proportion of the Camberwell population made up of West Indian immigrants (the only group large enough to justify a separate analysis in this study) was estimated in a full census in 1971 to be 4.9% while a 10% census in 1966 indicated 4.7% (Leff *et al*, 1976). It would appear that West Indian patients were over-represented in this UK sample (12% *vs* 4.9%). This is what Leff and his colleagues found when screening all psychiatric admissions in Camberwell as part of an epidemiological study of mania (Leff *et al*, 1976). Whilst there are a number of possible explanations for this over-representation, many studies have reported higher rates of psychiatric morbidity among immigrants than non-immigrants, including West Indian immigrants (Hemsi, 1967). In the 25–44 age-group, Rwegellera (1977) has calculated that the inception rates for schizophrenia are significantly increased among West Indian Camberwell residents as compared with Camberwell United Kingdom residents (0.80 vs 0.17—inception rate per thousand of the population at risk per annum). The findings of our study, as described later, indicate non-white race to be associated with poor prognosis and outcome.

Follow-up

At the 5-year follow-up, strenuous efforts were made to locate and interview each of the 100 schizophrenic patients. Apart from those who were in hospital (13%), patients were usually interviewed at home. It was possible to fully interview and evaluate 65 subjects after 5 years. This evaluation included a patient interview, where possible an additional interview with a relative, a computer search of the Department of Health and Social Security 'Mental Health Enquiry Record', which provided details of all UK hospital admissions during the follow-up period, and extensive perusal of all relevant hospital and other records. These 65 patients are included in all the analyses.

Comprehensive clinical information (other than personal interview) was available for a further 23 patients, using the same data gathering strategy. Most of these 23 patients had moved out of the country or well away from London, and it proved impractical to interview them. This group included six patients who had died during the follow-up period, five by suicide. Since these patients could be classified with regard to overall clinical outcome they have been included along with the 65 fully evaluated subjects in some analyses relating to clinical outcome. However, they have been omitted from analyses of social outcome as this information was incomplete. Twelve further patients were regarded as having limited follow-up information and these have been omitted altogether from any analyses.

A number of variables were considered to determine whether the non-interviewed patients were representative of the entire cohort. The non-interviewed group ($n=35$) were more frequently non-white than the interviewed ($P=0.001$). Moreover, of the 23 patients who could be classified with regard to clinical outcome but had not been interviewed at follow-up, 74% had a poor clinical outcome compared with 40% of the interviewed subjects ($P=0.03$). There was no difference by age, sex, marital status, or diagnosis at inclusion. Of the 35 non-interviewed subjects, 14 had moved to other countries (10 had returned to their country of origin), six had moved to distant parts of Britain, four lived a vagrant life style, five patients could not be located at all and one person refused to be interviewed. Six remaining patients were deceased. The excess of non-white patients who were in the 'not fully interviewed' category appears to be explained mainly by the repatriation of those patients with chronic schizophrenia to their countries of origin (to their families and extended families). Repatriation may also account for the excess of patients with poor clinical outcome found in the 'not fully interviewed group'.

Because of the large amount of information collected on each subject (for example, there are 360 items in the PSE alone), it was necessary to reduce and group the data. Items of information were grouped using a combination of statistical procedures and clinical judgement. In this study, it was anticipated that while particular configurations of variables would not always demonstrate strong intra-group statistical cohesion, these items would be meaningfully grouped in terms of their additive influence on outcome. Several constructs were selected as representing the prediction and outcome pictures of interest. These were: illness history (mental illness in family, history of physical illness in subject, obvious precipitating event, prior psychiatric history and episode preceded by lengthy symptoms), social functioning and 'negative' and 'positive' symptoms as predictors, and clinical and social functioning as outcomes. Social functioning was made up of occupational, social relationship and housing variables. For example, the inclusion occupational factors involved, 'number of job changes in the past year', 'financial dependence on family', 'ever had a full time job', 'had job in year prior to inclusion'. The inclusion social relationship factor involved, 'social isolation', 'marital status', 'lives alone', 'sees friends on a regular basis', 'sees relatives on a regular basis'. Inclusion housing status included, 'number of moves compared with culture', 'number of moves in past year', 'number of rooms available for personal use', 'length of time at current address' and 'home ownership'. Thus, the major predictor and outcome variables were created by summing the sets of items reflecting each major construct. The major predictor variables were created to reflect mutually exclusive constructs, as were the major outcome variables.

Correlation and multiple regression analyses comprised the primary analytic approaches to the examination of the study hypotheses. Correlation analyses were used to examine the associations between variables of interest, whilst multiple regression analyses were utilised in assessing the predictive value of specific variables while controlling for the potential confounding or interaction influences of age, race and sex.

Results

Symptomatic outcome

Eighty-eight schizophrenic patients could be classified across the four symptomatic outcome categories. Details are given in Table I.

Nineteen percent of the patients were in the best outcome category. These patients experienced no further episodes of major mental illness following the episode of inclusion nor any residual psychotic symptoms. The mean time spent in the episode of inclusion for these patients was 22 weeks. A further 30% of the patients were in the next best group having had further episodes of major mental disorder though, in total, for less than one year of the 5-year follow-up period. Half the patients, then, could be said to have had a good symptomatic outcome. Of the remainder, the great majority had experienced major mental disorder for at least half the 5-year follow-up period (including 10% who had never recovered symptomatically from the episode of inclusion).

TABLE I

Percentage distribution of subjects by symptomatic outcome at 5 years

	Good outcome		*Bad outcome*	
Number of patients	*1*	*2*	*3*	*4*
88	19%	30%	10%	41%

Outcome categories: (1) No further episodes of major mental disorders following episode of inclusion. No residual psychotic symptoms. Mean time spent in episode of inclusion was 22 weeks. (2) Further episodes of major mental disorder in addition to episode of inclusion. Total time in episodes less than 1 year. This category includes three subjects who did not have further episodes of major mental disorder but did have psychotic residual symptoms and have therefore been excluded from Group 1. (3) Between 1 and 2½ years spent in episodes of major mental disorder. (4) More than 2½ years in episodes of major mental disorder (including six subjects deceased at follow-up).

The 17 patients in the best outcome category (Group 1) were compared to the remaining 71 patients along a variety of parameters. Whilst these 17 patients were slightly younger than the other patients (78% *vs* 67% aged 15–34 years), they did not differ significantly on comparisons of sex, marital status, prior psychiatric history or level of social functioning at inclusion. Similarly, the 36 patients in the worst outcome category (Group 4) were compared to the remaining patients. Sex and inclusion social functioning were significantly associated with poor outcome ($P = 0.025$ in both cases). Males and those with poor inclusion social functioning were more likely to fall into the worst outcome group.

In order to control for the possible role of previous psychiatric history on overall symptomatic outcome, 20 of the 65 fully assessed patients who had no prior history at inclusion were compared in terms of symptomatic outcome with those who did. Fifty-three per cent of those with no prior psychiatric history had a poor symptomatic outcome compared with 45% of those who did ($P = \text{NS}$). This surprising result may reflect the fact that chronic patients were excluded from the study so that most of the patients with a prior illness had a history of less than one year and had been in hospital for less than 6 months in the preceding 5 years. In other words, those patients with a psychiatric history prior to inclusion in this study had psychiatric disorders of relatively recent origin with short hospitalisations, thus tending to minimise clinical differences between the two groups. In addition, psychiatric 'illness history' refers only to the presence or absence of prior episodes and does not include other important illness history characteristics such as length of the psychotic episode, premorbid personality or family history of mental illness. This broader concept of illness history is examined later in the paper in the section dealing with prognosis.

In addition to the data contained in Table I, information was collected to provide diagnostic details of the various episodes of psychiatric disorder which contributed to the overall symptomatic outcome result. The 65 fully assessed schizophrenic patients between them experienced 115 episodes of major mental disorder during the 5-year period; 84% of these episodes were schizophrenic psychoses (ICD 295) and 16%, affective psychoses (ICD 296). In other words, one in six episodes experienced by patients with an established schizophrenic disorder were of a pure affective type. The episodes of schizophrenic psychoses were predominantly of the paranoid type (295.3) with a small number of schizo-affective (295.7) and hebephrenic types (295.1). At 5-year follow-up, of the 65 patients for whom we had a PSE, 29% had delusions or hallucinations in the month prior to interview, 32% had behavioural disturbances which were probably related to their schizophrenic illness, 48% had "neurotic" symptoms and 32% were asymptomatic.

Social outcome

Two overall social outcome categories were created on the basis of total scores taking into account functioning in social relationships, housing status and occupational performance in the year prior to interview. Of the 65 fully interviewed patients who could be classified with respect to social outcome, approximately two-thirds (42/65) were categorised as having no severe impairment at 5-year follow-up as determined by a total social functioning score of 0–3, and one-third as showing evidence of severe social impairment with social functioning scores ranging from 4–7 (Table II). None of the patients in the 0–3 or 'no severe impairment' group had a score of 3 in any one of three social outcome areas, i.e. there was no major deficit in any one area. The mean score was 1.5. Twenty-five of the 42 patients in this social outcome group had handicap scores of only 0 or 1. Social outcome was not significantly associated with age or marital status.

TABLE II

Percentage distribution of subjects' outcome social functioning scores[1]

Score	*No severe impairment*[1] *0–3*	*Severe impairment*[2] *4–7*	*8–11*	*Total*
n	42	22	1	65
%	66	33	1	100

1. Social functioning scores were based on assessment of occupational performance, social functioning and housing status.
2. Severe social impairment defined as social functioning scores ranging from 4–11. A patient with a score in this range would have a deficit in each of the areas of assessment or would have a major deficit in at least one of these areas. None of the patients in the 0–3 group had a score of 3 in any one of the three social outcome areas.

Of these 65 patients, 40% (26) worked a full 12 months during the final year of the 5-year follow-up and another 12% (8) worked more than 6 months of the final year. At follow-up 74% (48) were socially active within their households to the extent expected of a person without a psychiatric disorder and 43% (28) socialised outside of the household and were involved with community activities at this same level of expectation, while only 35% (23) experienced satisfactory sexual functioning. Sixty-nine per cent of these patients (45) lived in housing conditions equal to or better than the norm for British culture.

Combined social and clinical outcome

Clinical and social outcome were significantly correlated ($P=0.0001$). Forty-nine per cent of the 65 patients with complete social and clinical outcome information had good outcomes in both areas of functioning (i.e. category 1 or 2 on symptomatic outcome (Table I) and a score of 0–3 on social functioning (Table II). It should be remembered, however, that the good symptomatic outcome group included some patients with disorders subsumed under ICD-8 'Neurosis' category as well as some patients with mild schizophrenic residual symptoms. The good social outcome category also included patients with some deficits, though none were severe. For example, a housewife with children at home (social functioning score = 1, gained in the occupational area) was unable to manage all the housework in the way she had prior to her illness because she felt 'slowed up' and could not 'do things as fast as I could before'. Her relatives helped out with the household duties, and with this support her household ran well. Another patient (social functioning score = 2, gained in social relationships) had an excellent work record as a schoolteacher and supported himself in independent lodgings. However, he was unable to establish and maintain peer or heterosexual relationships. Another patient (social functioning score = 2, gained in occupational functioning) lived in a youth hostel, was socialising well both in his household and in the community but had been employed in a menial job well below his level of education since his illness. These examples are intended to give both a flavour of the extent of the social handicaps included in the 'good' social outcome group and also to illustrate how relatively focused some of the social handicaps may be in the wake of schizophrenic illness.

Twelve per cent of the patients were found to have a good social outcome but a bad symptomatic outcome while 6% had a good symptomatic outcome, but a bad social outcome.

Prediction of symptomatic outcome

A majority of the hypotheses concerned with symptomatic outcome were confirmed. Specific illness history factors at inclusion into the study (particularly 'no precipitating event' and 'lengthy symptoms') were positively and significantly associated with poor symptomatic outcome at 5-year follow-up. Somewhat unexpectedly, poor social functioning at inclusion was also positively and significantly associated with poor symptomatic outcome. Positive symptoms did not demonstrate any relationship of significance with poor symptomatic outcome. Negative and non-specific symptoms did not demonstrate any significant relationship with

TABLE III

Predictor variables which correlate significantly with symptomatic outcome*

Predictor variable	*Clinical outcome*	*n*
	Pearson's R	
1. Illness history	0.22	84
2. Total social functioning	0.29	88
Occupational functioning	0.28	88
Social relationship functioning	0.21	88
Housing status	N.S.	88
3. Sex	0.22	88
4. Race	0.23	88

*Significant at $P<0.05$ level.

symptomatic outcome and therefore did not support the related hypothesis. Male gender was positively and significantly correlated with poor clinical outcome, as was non-white racial status, though age did not demonstrate a significant relationship with symptomatic outcome. Table III gives details.

Subclasses of the variable categories were introduced in a stepwise regression analysis, a procedure that sequentially adds predictor variables in order of their contribution to outcome, combining them to increase the overall correlation with outcome. Inclusion illness history, occupational functioning, social relationship functioning and negative and non-specific symptoms were the only variables which entered the model at an acceptable level of significance; these accounted for 23% of the variance in symptomatic outcome. When first order interaction terms were added to this model, three interaction terms met the significance criteria for acceptance. These interaction terms were such that non-white subjects with poor inclusion illness history has poorer symptomatic outcome than white subjects with poor inclusion illness history; male subjects with inclusion positive symptoms had poorer symptomatic outcome than females with inclusion positive symptoms; and, non-white subjects with poor inclusion social functioning had poorer symptomatic outcome than white subjects with poor inclusion social functioning. This full model including interaction terms explains 32% of the variance in symptomatic outcome. Given that interaction is present, the assessment of confounders is questionable. When interaction terms are excluded from the model however, sex and race enter in a significant fashion.

These results support the hypotheses identifying inclusion illness history, social functioning and negative and non-specific symptoms as predictors of symptomatic outcome and underscore the usefulness of including interaction terms in the analyses. Table IV summarises the significant findings of the symptomatic outcome regression analyses.

TABLE IV

Multiple regression analyses with symptomatic outcome as dependent variable (n = 84)

Dependent variable:

Clinical outcome

Independent variables:

i) Illness history
ii) Occupational functioning
iii) Social relationship functioning
iv) Negative and non-specific symptoms
—Variance in outcome accounted for: 23%
—F of this equation: 5.69 ($P < 0.0005$)

Interaction term added:

v) Race × illness history
vi) Sex × positive symptoms
vii) Race × social functioning
—Variance in outcome accounted for: 32%
—F of this equation: 3.48 ($P < 0.001$)

Prediction of social outcome

While certain inclusion psychopathological items were predictive of symptomatic outcome they were not predictive of social outcome. Inclusion social factors were however, predictive of social outcome. Poor social functioning at inclusion was positively and significantly associated with poor social outcome at 5-year follow-up. Male gender was positively and significantly correlated with poor social outcome, as was non-white racial status. Inclusion illness history, negative and non-specific symptoms, positive symptoms and age were not significantly associated with social outcome. Table V further describes the inclusion variables which were significantly correlated with social outcome.

TABLE V

Predictor variables which correlate significantly with social outcome (n = 64)*

Predictor variable	*Pearson's R*
1. Total social functioning	0.44
Occupational functioning	0.23
Social relationship functioning	0.38
Housing status	0.28
2. Sex	0.24
3. Race	0.36

*Significant at $P < 0.05$ level.

Subclasses of the variable categories were introduced in a stepwise regression analysis. Inclusion occupational functioning, social relationship functioning, accommodation status and negative and non-specific symptoms accounted for 21% of the variance in social outcome. When first order interaction terms were added to this model, four interaction terms met the significance criteria for acceptance into the model. Thus, being older and having positive symptoms at inclusion or, being older and having a poor illness history at inclusion led to poorer social outcome. Further, being male and having a poor illness history at inclusion or being non-white and having a poor illness history at inclusion also led to poorer social outcome. This full model including interaction terms explains 47% of the variance in social outcome. Again, given that interaction is present the assessment of confounders is questionable. When the interaction terms are excluded from the model however, sex, and race enter at acceptable significance levels.

These results do not support the hypotheses which predicted significant relationships between inclusion illness history and social outcome and inclusion negative and non-specific symptoms and social outcome. The hypothesis concerning a relationship between inclusion social functioning and social functioning at 5-year follow-up was, however, supported. Social factors appear to be the best predictors of social functioning with illness history explaining only a very small part (2%) of the variance in social outcome. These results support the inclusion of interaction terms in the analysis. Table VI summarizes the significant findings of the social outcome regression analysis.

TABLE VI
Multiple regression analyses with social outcome as dependent variable (n = 62)

Dependent variable:

Social outcome

Independent variables:

i) Occupational functioning
ii) Social relationship functioning
iii) Housing status
iv) Negative and non-specific symptoms
—Variance in outcome accounted for: 21%
—F of this equation: 5.13 ($P < 0.004$)

Interaction terms added:

v) Age × positive symptoms
vi) Age × illness history
vii) Race × illness history
viii) Sex × illness history
—Variance in outcome accounted for: 47%
—F of this equation: 2.62 ($P < 0.007$)

Discussion

A striking finding is the variability in outcome for this group of stringently diagnosed schizophrenic patients. About one-fifth did not experience any further episodes of major mental disorder after the episode of inclusion while, at the other extreme, one-tenth never recovered from their episode of inclusion; more than one-third of the patients spent over half the follow-up period in episodes of major mental disorder. In the month prior to follow-up about one-third experienced delusions or hallucinations, one-third behavioural disturbances and one-third were asymptomatic. Overall, about half the patients could be considered to have both good symptomatic and social outcomes, even though our findings are biased in the direction of poor outcome because of an excess of non-white patients who appear to have a more unfavourable course. It should be noted that even among those considered to have a good outcome many patients, as we have documented, continue to experience some degree of symptomatic or social handicap. The London data suggest that schizophrenic patients who are initially relatively clinically homogenous and who correspond to the strict notion of schizophrenia assumed in the CATEGO Program have a wide variety of outcomes in the 5-year follow-up period. This finding is not necessarily due to variations in diagnostic criteria or other methodological difficulties. The variability in outcome evident at the London centre was one of the major findings in the IPSS and 2-year follow-up study as a whole. Moreover, there also appear to be clear differences in the course and outcome of different centres, with patients in centres in developing countries having a better course and outcome than those in centres in developed countries, suggesting that the course and outcome of schizophrenia is affected by sociocultural conditions (WHO, 1979).

With respect to prediction, our results identify specific illness history factors (especially 'no precipitating event' and 'lengthy history') as predictors of symptomatic outcome. However, the major prediction results described here suggest an important role for occupational and social factors in predicting symptomatic as well as social outcome. These findings reflect results of the IPSS 2-year follow-up generally, which found positive correlations between social isolation and marital status and all outcome measures. Strauss & Carpenter (1974), in a correlational analysis of the Washington IPSS 2-year follow-up data, reported positive correlations between social relations' and 'employment' with symptomatic outcome ($r = 0.28$ and $r = 0.28$ respectively, $P < 0.01$). These results are very similar to those reported here ($r = 0.21$ and $r = 0.28$ respectively, $P < 0.06$). Their 5-year follow-up results (Strauss & Carpenter, 1977) confirmed the importance of these predictors and showed that adding 'previous hospitalisations' to 'previous social contact' and 'previous employement' provided the most efficient way of anticipating 5-year outcome. In these results from the London centre, the relationships between inclusion illness history and symptomatic outcome and between inclusion social functioning and symptomatic and social outcome were demonstrated in correlational analyses. They persisted in multiple regression analyses controlling for the impact of age, race and sex. Reflecting the IPSS 2-year follow-up findings, social variables and illness history variables are about equal with respect to their ability to predict symptomatic outcome (WHO, 1979). (Unlike the 2-year follow-up results, symptomatic variables contribute somewhat less to prediction of outcome.) The variables examined in this UK study accounted for only 32% of the variance in the symptomatic outcome of schizophrenia and 47% of the variance in social outcome. This underscores the conclusion of other prognostic studies that no single predictor variable and no combination of key predictor variables is capable of explaining a majority of the variance in the outcome of schizophrenia, though utilising combinations of predictor variables clearly leads to a more complete picture of the prediction process. Nevertheless, the majority of the variance in outcome prediction remains unexplained. This may be because of methodological issues to do with this study or because certain recognised (as well as yet unrecognised) variables that have a bearing on the outcome of schizophrenia have been omitted. No attempt was made to control for treatment in this study nor to take into account such diverse factors as the quality of the home environment of patients (Brown *et al*, 1962), the impact of intervening life events (Leff & Vaughn, 1980), or the role of organic dysfunction (Kolakowska *et al*, 1985). Future research which addresses such additional intervening variables may bring new insights into the prediction of outcome in schizophrenia.

In conclusion the main results of interest in this study include the variability in clinical outcome and the finding that poor social functioning at inclusion is predictive of poor symptomatic outcome at 5-year follow-up. Thirty-two percent of the variance in symptomatic outcome was explained and to a great extent by social factors. There are a number of possible explanations for this finding. Poor premorbid social functioning may itself represent 'negative' type symptoms of schizophrenia, these symptoms tending to be associated with chronicity, though in this study 'negative' symptoms were not found to be positively associated with outcome. Poor social adjustment may also be seen to augur poorly for the patient's management of subsequent schizophrenic illness in general. Another possibility is that the social deficit identified at inclusion in this study as predicting subsequent poor symptomatic outcome is interacting with other established risk factors for schizophrenia thus giving rise to chronicity. For example, poor social functioning, e.g. work impairment in the patient, is known to engender the formation of critical, hostile and over-involved attitudes and behaviours by relatives towards the patient (Vaughn & Leff, 1981) and this to deleteriously affect the subsequent course of the schizophrenic illness (Vaughn & Leff, 1976). In support of this notion Vaughn and Leff reported that two-thirds of the patients with work impairment were found in those homes characterised by critical or over-involved relatives, and that furthermore 55% of the work-impaired patients from these homes relapsed in the subsequent 9 months compared with none from other homes. In other words, poor social functioning in the setting of schizophrenia may gain its status as a predictor of outcome by way of its interaction with the patient's family members. A variety of social factors such as inadequate housing or social isolation may also serve as chronic life stressors playing a role similar to that of life events in provoking further episodes of schizophrenia (Brown & Birley, 1968) while lack of social activities outside the home and social isolation may increase patient contact with critical or over-involved relatives to a degree which may be harmful (Brown *et al*, 1972). The overall results need to be interpreted with caution since no special attempts were made at inclusion to ensure a representative group of patients. The finding that social factors predict symptomatic outcome in schizophrenia may suggest emphasising particular therapeutic strategies since it has been demonstrated that maladaptive social functioning is amenable to change (Leff *et al*, 1982; Stein *et al*, 1975; Lamb, 1978; Wallace, 1980).

Appendix 1
Social outcome variables

Variables	*Possible range scores*[1]
1. Total Occupational Functioning	0–3
a) Months worked in past year	0–1
b) Occupation consistent with education	0–1
c) Occupational status compared with culture	0–1
2. Total Social Relationship Functioning	0–5
a) Active community involvement	0–1
b) Satisfactory sexual functioning	0–1
c) Socialises in community as normal	0–1
d) Socialises in household as normal	0–1
e) Currently socialises as did prior to onset of illness	0–1
3. Total Housing/Accommodation Status	0–3
a) Overcrowded	0–1
b) Unsatisfactory in other ways	0–1
c) Compared with culture	0–1

*Low score = good functioning

References

Brown, G. W. (1962) The influence of family life on the course of schizophrenia. *British Journal of Preventive Social Medicine*, **16**, 55.

—— & Birley, J. L. T. (1968) Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behaviour*, **9**, 203–214.

——, Bone, M., Dalison, B. & Wing, J. K. (1966) *Schizophrenia and Social Care*. London: Oxford University Press.

——, Birley, J. L. T. & Wing, J. K. (1972) Influence of family life on the course of schizophrenic disorders: a replication. *British Journal of Psychiatry*, **121**, 241–258.

Fairweather, G. W., Sanders, D. H., Maynard, H., *et al* (1969) *Community Life for the Mentally Ill*. Chicago: Aldine.

Hawk, A. B., Carpenter, W. T., Jr. & Strauss, J. S. (1975) Diagnostic criteria and five year outcome in schizophrenia: A report from the International Pilot Study of Schizophrenia. *Archives of General Psychiatry*, **32**, 343–347.

Hemsi, L. K. (1967) Psychiatric morbidity of West Indian immigrants. *Social Psychiatry*, **2**, 95–100.

Kolakowska, T., Williams, A. O., Ardern, M., Reveley, M. A., Jambor, K., Gelder, M. G. & Mandelbrote, B. M. (1985) Schizophrenia with good and poor outcome. I. Early clinical features, response to neuroleptics and signs of organic dysfunction. *British Journal of Psychiatry*, **146**, 229–246.

LAMB, H. R. (1978) *Community Survival for Long-Term Patients.* Washington: Jossey-Bass.

LANGFELDT, G. (1956) The prognosis of schizophrenia. *Acta Psychiatrica Neurologica Scandinavia*, Suppl. 110.

LEFF, J. & VAUGHN, C. (1980) The interaction of life events and relatives' expressed emotion in schizophrenia and depressive neurosis. *British Journal of Psychiatry*, **136**, 145–153.

——, FISHER, M. & BERTELSEN, A. (1976) A cross-national epidemiological study of mania. *British Journal of Psychiatry*, **129**, 428–442.

——, KUIPERS, L., BERKOWITZ, R., EBERLEIN-VRIES, R. & STURGEON, D. (1982) A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, **141**, 121–134.

MACKAY, A. V. P. (1980) Positive and negative schizophrenia symptoms and the role of dopamine. *British Journal of Psychiatry*, **137**, 379.

PASAMANICK, B., SCARPITTI, F. & DINITZ, S. (1967) *Schizophrenics in the Community.* New York: Appleton-Century-Croft.

RWEGELLERA, G. G. C. (1977) Psychiatric morbidity among West Africans and West Indians living in London. *Psychological Medicine*, **7**, 317–329.

STEIN, L. I., TEST, M. A. & MARX, A. J. (1975) Alternative to the Hospital: A Controlled Study. *American Journal of Psychiatry*, **132**, 517–522.

STRAUSS, J. S. & CARPENTER, W. T. JR. (1974) The prediction of outcome in schizophrenia: II. Relationships between predictor and outcome variables. *Archives of General Psychiatry*, **31**, 37–42.

—— & —— (1977) The prediction of outcome in schizophrenia: III. Five year outcome and its predictors. *Archives of General Psychiatry*, **34**, 159–163.

VAILLANT, G. (1964) Prospective prediction of schizophrenia remission. *Archives of General Psychiatry*, **21**, 509–515.

VAUGHN, C. E. & LEFF, J. P. (1976) The influence of family and social factors on the course of psychiatric illness: A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, **129**, 125–137.

—— & —— (1981) Patterns of emotional response in relatives of schizophrenic patients. *Schizophrenia Bulletin*, **7**, 43–45.

WALLACE, C. J. (1980) A review and critique of social skills training with schizophrenic patients. *Schizophrenia Bulletin*, **6**, 42.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Description and Classification of Psychiatric Symptoms: An Instruction Manual for the PSE and Catego System.* London: Cambridge University Press.

WORLD HEALTH ORGANIZATION (1965) *International Classification of Diseases—Eighth Revision.* Geneva: WHO.

—— (1973) *International Pilot Study of Schizophrenia.* Vol. 1, Geneva: WHO.

—— (1979) *Schizophrenia: An International Follow-up Study, Vol. II.* Geneva, WHO.

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Clomipramine and 'Drive' in People with Anorexia Nervosa:
An In-patient Study

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Drugs can sometimes be helpful within the overall treatment and management of anorexia nervosa. In this study we have examined the impact of clomipramine on food and activity in anorectics engaged in a treatment programme enabling them to regain normal body weight. Consumption of the drug was associated with increased appetite, hunger and calorie consumption during the early stages of treatment. There was no impact on ultimate outcome. In this study it was evident that the drug was unnecessary for weight gain, but it may be helpful in other programmes, both short and long term, where the patient has sufficient trust (to tolerate the enhanced hunger experience) but is not involved in the same behavioural constraints. However, its possible value in such programmes has not been proven here. Meanwhile, we take our findings as further evidence that clomipramine increases drive behaviour and appetite in humans and may be beneficial in the treatment of affective disorders because of this effect.

It has been argued elsewhere (Crisp, 1967; 1978; 1984) that the best treatment for anorexia nervosa is a controlled intake of normal amounts of carbohydrate and calories in general; moreover, that drugs aimed at facilitating appetite can be counterproductive. Phenothaizines can sometimes be helpful through their tranquillising and immobilising effects but such consequences may induce a further reactive panic or hostility in anorectics who then feel trapped. Minor tranquillisers can occasionally be helpful, but also invite self-poisoning behaviour. The potential sedative effect of amitriptyline may be nullified by its occasional extreme carbohydrate-craving potential (Paykel, 1977), so alarming for the anorectic. Other hypnotics should be avoided at all costs. Otherwise they tend to become used by the anorectic in the same way that she sometimes abuses alcohol.

In 1977 we decided to study the impact of clomipramine on patients within our established in-patient treatment programme (Crisp, 1980*b*). It had been claimed that clomipramine, a tricyclic with some resemblance also to chlorpromazine, was potentially useful in the treatment of severe obsessional and phobic disorders. To the extent that there may be primary phobic and secondary avoidance behaviour and obsessional features within anorexia nervosa, it seemed justified to explore the effects of the drug within the syndrome.

Method

Patients are admitted to the anorectic unit at Atkinson Morley's Hospital after having committed themselves to treatment involving restoration of body weight to matched population mean levels consistent with the anorectic's age at onset of the condition (Crisp, 1980*a*). Patients eat 3000 calories prescribed per day within a normal diet and meal pattern. This generates a weekly weight gain of about 1.3 kg. At the time of the study (1977–1978) patients in the programme remained in bed without exception until they had reached 'target weight'. Patients are weighed routinely twice weekly. This behavioural programme is complemented by intensive individual and family psychotherapy involving the skills of a multidisciplinary team. Otherwise, of course, patients would not stay in it. On achieving target weight patients become progressively more mobile in a planned way with a view to discharge within a further 6 weeks. Out-patient follow-up involves monitoring of body weight and related behaviour, coupled with continuing psychotherapy.

For the purposes of the present study 16 female patients, successive admissions to the unit, all of whom understood the nature of the study and had given their informed consent in respect of involvement in it, were randomly allocated to consumption of 50 mg clomipramine or else placebo identical in appearance at 20 h each day.

Patients were also required to complete a series of analogue scales four times a day: before breakfast, before lunch, before dinner and just before retiring. These scales measured aspects of mood and appetitive behaviour, monitoring the daily fluctuations of hunger, appetite, anxiety, anger, sadness, tension, excitement, irritability and restlessness.

On one day each week the patients dispensed with the prescribed diet of the refeeding programme and chose whatever diet they wished. The food taken on this 'free day' was quantified. In addition, dietary histories were completed by the patients on other days of the refeeding programme selected at random. This allowed the dietary intake on the unsupervised 'free day' to be compared with the usual prescribed diet.

Overactivity is a common feature of anorexia nervosa. Any effect of the drug on activity would affect energy balance and hence weight gain. Therefore each week throughout the study, each patient spent 24 hours on a bed which was specially constructed to record and quantify movement (Crisp *et al*, 1970).

Results

The two groups—formed by random allocation of consecutive admissions—were well matched for mean duration of illness [clomipramine group 5 years (range 2-10 years), placebo group 3.7 years (range 1-7 years)] and mean age at onset [clomipramine group 15.9 years (range 13-20 years), placebo group 17.7 years (range 14–22 years)]. As populations they were mildly obese before the onset of illness and the social classes were represented equally in the two groups. At the time of admission the mean age of the clomipramine group was 20.9 years and that of the placebo group 21.4 years. One subject from each group presented with persistent vomiting and prolonged bulimia, whilst the other seven had lost weight and maintained low weight by abstaining from food.

Three patients in our sample, two of whom were found subsequently to be taking active substances, asked to leave the project before reaching their target weights. One, on clomipramine, left during her ninth week in the study, when she had reached 95% of her target weight. This appeared to be mainly due to psychodynamic 'transference' difficulties. The other two—one on placebo, the other on clomipramine—stopped their tablets during their third and fourth weeks in the study, when they had reached 82% and 80% respectively of their target weights. Both (including the patient on placebo) complained of side-effects usually associated with tricyclic antidepressants.

The mean body weights, at admission, of the clomipramine and placebo groups were not significantly different, though the placebo group were slightly lighter (Table I). Mean target weight was the same for the two groups. All the patients, including those who dropped out of the study, reached their target weights. The two groups showed no significant differences in weight gained during the refeeding programme, or in the time taken. Those who took the clomipramine spent a mean of 76 (s.d. 24) days to reach their target weights, whilst the control group spent a mean of 72 (s.d. 28) days. Those taking active medication gained, on average, less body weight each day than the placebo group, but this difference was not statistically significant. Both groups were highly active in bed both by day and by night. There were no signficant differences between the groups in these respects but a tendency for the placebo group to be more active at night (Table II).

TABLE I

Weight change in anorectic patients given clomipramine or placebo

	Clomipramine group		*Placebo group*	
	mean	*s.d.*	*mean*	*s.d.*
Mean body weight on admission: kg	40.6	4.6	37.7	5.2
Mean target weight: kg	53.93	4.22	52.4	6.15
Mean weight gain: kg	13.33	5.32	14.7	4.60
Rate of weight gain (kg/day)	0.17	0.038	0.21	0.031

TABLE II

Mean hourly activity (units of movement on motility bed)

	Nocturnal (23:00 to 7:00)	*Diurnal (7:00 to 23:00)*	*For 24 hours*
Clomipramine	97.1 ± 41.9	249.9 ± 127.1	171.5 ± 80.1
Placebo	125.2 ± 40.7	270.7 ± 100.0	197.9 ± 65.9

Mean daily energy intake on the refeeding programme was 2650 calories. When patients were allowed their own choice of food (free day), those on placebo demonstrated marked weekly variation (Fig. 1). For five such free days their mean intake was below the mean of the refeeding programme.

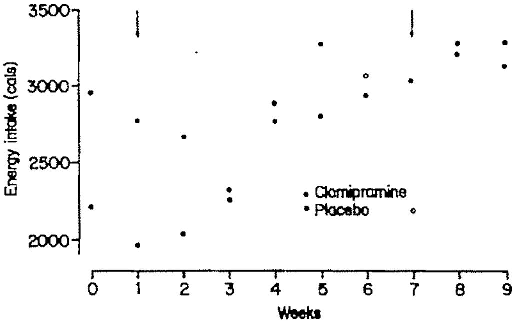

FIG 1. Mean 'free day' energy intake for clomipramine and placebo groups in each week of study, showing the statistically significant differences at weeks 2 and 8 (arrowed).

Patients in the clomipramine group were more stable in their eating habits and, except for 1 week, their mean energy intake was above the mean for the prescribed diets given on the other days.

During the second and eighth weeks of the study, the difference in caloric intake between the two groups was statistically significant (standard *t*-test), and in the first week it just failed to reach significance (Fig. 1).

Table III shows the mean analogue scale scores (and s.d.s) for the eight feelings measured. Only one—hunger—significantly differentiated overall between the two populations. For the first 2 months those taking clomipramine were significantly more hungry (standard

TABLE III
Mean analogue scale scores

	Clomipramine		*Placebo*	
	mean	*s.d.*	*mean*	*s.d.*
Hunger	48	8.3	19	6.1
Anxiety	30	5.2	26	4.7
Anger	21	6	23	5
Excitement	32	9	25	10
Sadness	38	16	51	17
Tension	42	12	31	11
Appetite	51	5	49	6.4

t-test), whilst during the last 3 weeks the two populations felt hunger similarly (Fig. 2).

Although the overall appetite scores were not statistically different, a more detailed examination (Fig. 3) of daily and weekly recordings indicates that the appreciation of appetite by the two groups was quite distinct. For the first 5 weeks those on the drug felt an increased appetite. This was statistically significant for the first 3 weeks and the fifth week (Student *t*-test). Thereafter the scores of the two groups were similar. The clomipramine group appreciated appetite as a more stable phenomenon and their reports lack the major fluctuations of those who did not take the medication.

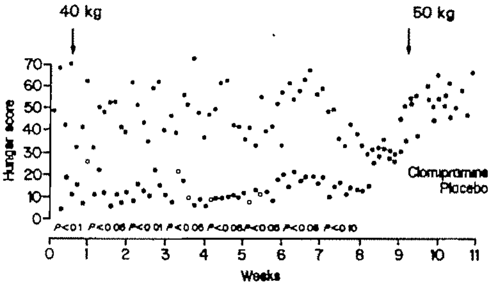

FIG. 2. Mean daily hunger scores of the two populations. Figures above tracings give mean body in kg.

At 1-year follow-up (Lacey & Crisp, 1980) there was no significant difference in body weight between the groups, though the clomipramine group (at 95.6% of target weight) was somewhat heavier than the control group (89.9% of target weight). At 4-year follow-up (1981–1982), involving personal interviews, extensive assessment was made using the original Morgan & Russell measures of outcome (Morgan & Russell, 1975; Hsu *et al*, 1979). The average score on this measure is the sum of the outcomes assessed in four areas: nutritional status, sexual adjustment, socio-economic adjustment and mental state. Ratings refer to the 6 months before assessment. There was no difference between the groups. The clomipramine group was at a mean 94.1% of target weight, the placebo group 92.7%. Mean sexual adjustment scores (which include a measure of menstruation) were 2.4 for each group while mean social adjustment scores were 3.0 and 2.5 respectively.

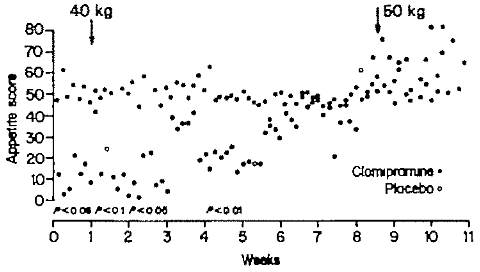

FIG. 3. Mean daily appetite scores of the two populations. Figures above tracings give mean body weight in kg.

Discussion

In this study the additive effect of clomipramine has been studied within an established in-patient programme. The latter, with its complementary behavioural and psychodynamic treatment elements is itself sufficient to enable anorectics to achieve target weight in a predictable way. However, when the behavioural constraints were occasionally relaxed in the present study the clomipramine consumption was, especially in the earlier stages of treatment, sometimes significantly associated with continued ingestion of calories sufficient to maintain such weight gain whereas, without clomipramine, this was not always the case.

Consumption of the drug was also associated with increased reported experience of hunger and appetite during the early stages of treatment. Such experience also arose in the placebo group after the half-way stage of treatment, by which time the average weight of each group would have been about 45–47 kg. This body weight is of a pubertal level and kind and associated in normal growth with a spurt in both appetite and weight gain. The first author has argued elsewhere that this experience is often the principal seed bed of anorexia nervosa in those who associate it with loss of control in respect of new-found sexuality and its consequences, and who identify this unwanted and frightening impulse with their increasing and newly constituted 'fatness'. It is therefore likely that it is experienced as frightening once again by anorectics gaining weight in the way described. Indeed, it is at this level of weight that an anorectic is most likely to drop out of treatment, especially if she and her family have not now developed some additional and psychological resources to enable her and them to live with the experience and to cope with and adjust to it.

Anorectics are known to be hyperactive and this population was probably no exception, although we do not have normative data concerning the activity of non-anorectic subjects confined to bed throughout

24 hours. Activity levels, as measured, were not affected by clomipramine.

In this study the drug was stopped at the point of reaching target weight. Up until that time it had seemed to be potentially helpful in terms of achieving weight gain, albeit unnecessary for this purpose within the established treatment programme. However, of course, the problem with treating anorexia nervosa is not so much this short term treatment as the longer term one enabling the anorectic to maintain herself at normal body weight and thereby begin to work through its consequences in a psychological sense. If we had continued to prescribe clomipramine following mobilisation and relaxation of the behavioural constraints of the in-patient programme in respect of food intake, then perhaps it would have been helpful during the remainder of the inpatient's stay (when patients usually anyway retained their body weights) and also during out-patient follow-up. In this respect both its pharmacological and its additional symbolic value in terms of the supportive effect of a 'transitional object' at a time when treatment is otherwise severely diluted in our own service, largely because of limited out-patient resources, should be borne in mind. However, we are aware that there would also have been problems with such a policy, because quite a number of patients complained of side-effects and some dropped out of the study prematurely on that account, and undoubtedly in a normal treatment setting there would have been some non-compliance.

Finally, this study invites further introspection concerning the mode of action of 'antidepressant' drugs. The first author has argued elsewhere (Crisp, 1980*b*; 1986) that the tricyclic drugs have an immediate effect on drive behaviour (e.g. foraging in association with the experience of appetite and hunger, sexual interest and activity and also generalised restlessness) and that it is this effect which enables some depressives to re-engage socially and thereby begin to interact and become less 'depressed' as the weeks go by. The present study, demonstrating the rapid impact of clomipramine on reported appetite and on actual caloric ingestion in anorectics, provides some support for this view.

References

CRISP, A. H. (1967) The possible significance of some behavioural correlates of weight and carbohydrate intake. *Journal of Psychosomatic Research*, **11**, 117–131.

—— (1978) Disturbances of neurotransmitter metabolism in anorexia nervosa. *Proceedings of the Nutritional Society*, **37**, 151–159.

—— (1980*a*) *Anorexia Nervosa: Let Me Be*. London: Academic Press.

—— (1980*b*) Sleep, activity, nutrition and mood. *British Journal of Psychiatry*, **137**, 1–7.

—— (1984) Treatment of anorexia nervosa: what can be the role of psychopharmacological agents? In *The Psychobiology of Anorexia Nervosa* (eds K. M. Pirke & D. Ploog). Heidelberg: Springer-Verlag.

—— (1986) 'Biological' depression; because sleep fails? *Postgraduate Medical Journal*, **62**, 179–185.

——, STONEHILL, E. & EVERSDEN, I. D. (1970) The design of a motility bed including its calibration for the subject's weight. *Medical and Biological Engineering*, **8**, 455–463.

HSU, L. K. G., CRISP, A. H. & HARDING, B. (1979) Outcome of anorexia nervosa. *Lancet*, **i**, 62–65.

LACEY, J. H. & CRISP, A. H. (1980) Hunger, food intake and weight; the impact of clomipramine on a refeeding anorexia nervosa population. *Postgraduate Medical Journal*, **56**, (suppl. 1), 79–85.

MORGAN, H. G. & RUSSELL, G. F. M. (1975) Value of family background and clinical features as predictors of longterm outcome in anorexia nervosa: four-year study of 41 patients. *Psychological Medicine*, **5**, 355–371.

PAYKEL, E. S. (1977) Depression, antidepressant drugs and appetite. In *Depression — the Biochemical and Physiological Role of Ludiomil* (ed. A. Jukes). Horsham: CIBA Laboratories.

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The Memory Clinic

A New Approach to the Detection of Dementia

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Memory impairment is a salient and early feature of developing dementia, but in practice is often not recognised until it has reached an advanced stage. The operation described is of a Memory Clinic opened on an experimental basis at the Geriatric Research Unit, University College London, in 1983, with the aim of identifying the causes of memory impairment in the elderly, with particular reference to the early detection of dementia. It proved possible to identify a group of people with early dementia who had previously been undiagnosed, and also to reveal deficiencies in the utilisation of existing services. Memory clinics would be a valuable addition to geriatric and psychogeriatric services.

Dementia is often not detected until the mental deterioration has reached an advanced stage. Family doctors may fail to recognise it and the general public tends to accept poor memory as a normal concomitant of ageing. Thus, in many patients the diagnosis of dementia is not made until some other condition precipitates admission to hospital.

Epidemiological studies in the elderly population have shown a remarkable consistency in the prevalence of dementia. The results have been summarised in the Report of the Royal College of Physicians on Organic Mental Impairment in the Elderly (1981); some reported on the incidence of early dementia. Although it can be assumed that patients exhibiting moderate to severe symptoms will have passed through a stage of mild dementia, the diagnosis of early deterioration in these cases has presented many problems which have not been resolved satisfactorily (Kay *et al*, 1968; Bergmann *et al*, 1971; Henderson & Huppert, 1984).

The detection of organic mental impairment in epidemiological studies has usually been based on mental status questionnaires (Roth & Hopkins, 1953; Hodkinson, 1974), which test for short- and long-term memory, orientation, awareness of events and ability to count forwards and backwards. Memory impairment is a salient and early feature of developing dementia, but simple tests such as these are in themselves of restricted value for diagnostic purposes (Hinton & Withers, 1971; Hare, 1978; Whitehead & Hunt, 1982).

We outline the operation of a Memory Clinic which we opened on an experimental basis in April 1983 with the aim of identifying the causes of memory impairment in the elderly, with particular reference to the early detection of dementia. The clinic was established to provide a service for patients, their carers and general practitioners. We describe a more comprehensive approach to diagnosis than is usual, based on team investigation, and report on the first 50 patients.

Operation of the clinic

General practitioners are encouraged to refer elderly people who complain about their memory or whose friends or relatives have noticed memory problems. The team in the clinic consists of a psychologist, a physician and a psychiatrist who see the patient independently, each using his own method of assessment.

The psychologist

The psychologist usually sees the patient first to ascertain whether there is a true memory deficit. A memory problem is defined as a condition experienced by the patient which may or may not be accompanied by an objectively observed memory deficit. By means of an in-depth interview, the psychologist elicits the evidence, in terms of actual incidents of behaviour, that has led the patient to suspect that he has a memory problem, and records the earliest failures which led to this suspicion being aroused. An informant is interviewed to obtain an independent and detailed behavioural history; this is especially necessary when the patient himself cannot given an accurate account. Several factors affect the readiness with which memory problems are reported.

Two simple tests are administered initially:

A: *The Cerebral Function Test (Silver, 1972)*

This is a brief screen of various mental abilities, including memory for recently learnt material, naming, vocabulary and spatial skill. It is a short test battery which was designed to be carried out by unskilled observers and to be easily understood. To this end, the items have face validity; i.e. their meaning is obvious and the implications of failure are readily interpretable. The predictive validity of the total test score has been established by Silver and again by Ross & Horne (1977) with respect to an old person's ability to live independently. In the Memory Clinic we were not concerned with this aspect of the test but used it as a brief neuropsychological screen for indications of language and spatial deficit, low educational level and inability to learn and retain new material. The memory component, learning a fictitious name and address and recalling it after 5 min has been described elsewhere (Priest & Woolfson 1986), whose list details the administration procedure with one exception. Prior to presentation of the target material, the patient is warned that it will be necessary to recall it after 5 min. If this warning is omitted, the nature of the task is altered and it becomes more difficult. This test is similar to that included in many screening tests familiar to geriatricians and psychogeriatricians. Many of these mini-batteries are of limited use, as they focus on memory and orientation alone, omitting screens of language and spatial ability. In the Silver test, spatial ability is tested in several ways, including that first described by Isaacs (1963) using toys. We had the test materials specially made in wood so that they looked less like toys and our patients found them perfectly acceptable.

B: *The Kendrick Battery for the Detection of Dementia (Gibson & Kendrick, 1979; Kendrick et al,* 1979)

This consists of two easily administered tests, for which there are normative data. The first subtest taps memory by immediate recall, i.e. immediately after exposure of the test material, which is a card displaying a number of pictures of common objects. There are four cards, the number of objects to be recalled increasing over the series. The second part tests perceptu-motor speed and patients are required to copy lines of digits as fast as possible. The instructions for administering, timing and scoring these procedures are precise. The battery has been tested for criterion validity on groups of normal and memory-impaired old people. It yields evidence of a person's definite memory deficit but does not indicate if he is below average for a person of his age and educational level. These tests can therefore offer evidence to confirm that a person's memory problem does indeed reflect an actual memory deficit, but a diagnosis of dementia should not be made on test scores alone, in the absence of other evidence and without excluding alternative causes for the memory deficit.

If the patient fails the memory component of test A and scores in the abnormal ranges of test B, more sophisticated memory assessment is redundant, but the procedures are repeated in 6 weeks to confirm the results of the first observations.

If the patient scores in the normal range on the screening tests, more demanding memory assessments are made with particular reference to dual performance tasks and delayed recall (Randt *et al*, 1980). At this stage, it is essential to establish a baseline of memory ability with which to compare future performance, as stability or decline in this skill is critical to establishing a diagnosis. To this end, the limits of the efficiency of the patient's memory are sought. Test instruments on which the patient easily achieves near-perfect scores risk being insensitive to any future change. In this respect, the usefulness of some computer-controlled tests that we have designed is also being assessed (Simpson & Linney, 1985).

The psychologist assigns each patient to one of three categories according to the amount of evidence that is found after several visits. This grouping parallels the process by which a clinical diagnosis of dementia is finally made, i.e. based on memory deficit in the absence of another explanation. Characteristics of the patients in the three categories are:

Category 1: Clear evidence of memory deficit

(a) Memory deficit on objective tests that have established cut-off points, e.g. Object Learning Test (OLT) of the Kendrick Battery.
(b) Behavioural evidence of deficit from the reports of an informant and/or as observed during visits to the clinic.
(c) Subjective complaints about memory problems may or may not be present according to the amount of insight retained.

Category 2: Equivocal evidence of memory deficit

(a) Normal scores on the OLT although other memory tests may yield scores lower than expected for the person's age-group and IQ.
(b) Behavioural evidence from an informant and/or from observations during visits to the clinic.
(c) Subjective feelings of memory problems.

Category 3: No objective evidence of memory deficit

(a) No clear evidence of memory deficit on testing; normal scores on OLT, and performance on other tests in the expected range for a person of this age and IQ.
(b) No clear behavioural evidence from any source.
(c) Subjective feelings of memory problems.

The physician

The physician attempts to identify any extra-cerebral causes of memory loss (Royal College of Physicians, 1981; Wood, 1984) and to distinguish between acute confusion, dementia and depression. He screens for focal cerebral lesions presenting as dementia, and tries to distinguish between dementia of Alzheimer's type and multi-infarct dementia, using the Hachinski score (Hachinski *et al*, 1975). This is an 'ischaemic score' based on the different clinical features of multi-infarct dementia and Alzheimer-type dementia. Patients with a score of 7 or more are rated as probable multi-infarct dementia; those below 4 as Alzheimer-type dementia. Exceptions to this demarcation are to be found in clinical practice, and the number and clinical extent of strokes, the amount of infarcted tissue seen on CT and the presence of pseudobulbar palsy should be taken into account, and perhaps weighted more heavily (Miller *et al*, 1984). In a study of elderly demented patients who came to autopsy, the score has been shown to identify accurately patients with multi-infarct dementia or with mixed pathology (Rosen *et al*, 1980).

A careful medical history is obtained from the patient and relatives wherever possible, a full medical examination is carried out, and in the neurological examination particular attention is paid to evidence of dysphasia and parietal lobe signs.

The laboratory investigations include a full blood count, serum glucose, urea and electrolytes, creatinine, rapid plasma reagin test and treponema pallidum haemagglutination assay (TPHA) test, and liver function and thyroid function tests. Serum vitamin B12 and folate concentrations are measured only when the mean cell volume is raised. It is recognised that the yield from these tests is low when applied as a routine in the investigation of dementia (Report of the Royal College of Physicians, 1981). Other diagnostic procedures, including CT scanning, are carried out when indicated.

Special attention is paid to the drugs that the patient is taking, since it is known that several groups of drugs can impair memory and cognitive performance in the elderly. These include hypnotics and other psychotropic drugs, anticholinergic agents and certain centrally acting antihypertensive drugs (Solomon *et al*, 1983).

The psychiatrist

The psychiatrist takes a history and carries out a mental state examination. The objects, are first, to diagnose any affective disorder that may be responsible for or contributing to the memory impairment; secondly, to diagnose dementia on clinical evidence—the diagnostic criteria are those of Hare (1978), based on the Kew Cognitive Map (McDonald, 1969). Thirdly, the psychiatrist assesses the overall severity of the dementia and the future provision that the patient will require; he will have the continuing clinical responsibility for the majority of the patients. In the absence of a simple rating scale of severity that takes account of all the cognitive impairments of dementia, the various degrees of severity are characterised as below. (The validity of these categories will be tested by the follow-up studies, and it is hoped that a simple and useful clinical rating scale can be provided.)

Absent	No evidence of cognitive impairment.
Equivocal	Slight memory impairment, definitely or possibly due to affective disorder.
Minimal	Definite memory impairment *or* dubious or minimal memory impairment, together with slight nominal dysphasia.
Mild	Definite memory impairment, together with definite nominal dysphasia.
Moderate	Definite impairment of both recent and remote memory, possibly with confabulation. Moderate dysphasia (including difficulty in naming simple objects) and some failure of comprehension. Possibly some parietal features (e.g. dressing apraxia). Possibly some personality deterioration (e.g. emotional lability, paranoia).
Severe	As above, but all worse in degree and with definite personality deterioration.
Profound	As above, but all worse in degree; totally dependent on others for self-care. Little coherent communication.

Main diagnostic categories

The team members meet regularly to discuss the patients they have investigated. A diagnosis is established or, if this is not possible, a hypothesis about the likely diagnosis is formulated. The diagnostic categories for the first 50 patients (32 female, 18 male; age-range 61–90 years; mean age 75.2 years) who have been fully assessed by all three members of the team are shown in Table I.

A firm clinical diagnosis was made in 66% of patients. Of the total series of patients, 50% were dementing to some degree, and all these patients had a memory deficit in category 1. A provisional diagnosis of dementia was made in 14% of patients, and these had memory deficits in categories 1 or 2. In 10%, a diagnosis of probable affective disorder was made, and these patients had memory deficits in categories 2 or 3; the other diagnoses in 8% of patients included potentially reversible organic disorders such as polypharmacy (two patients), hyperthyroidism (one patient) and Wernicke's encephalopathy (one patient). In 10% of patients, no clinical diagnosis could be established and neither could a memory deficit be demonstrated.

Of the 25 patients who had evidence of Alzheimer dementia, the psychogeriatrician rated eight as moderately demented, 11 as mildly demented and six as minimally demented. Amongst the 16 in this group who had been referred by their family doctor, only five had already been put in touch with the psychogeriatric and community support services prior to being referred to the Memory Clinic. Neither these five nor the other clearly demented patients in this group had thus far received the level of support their condition required. These patients, with a firm clinical diagnosis of dementia, were referred to their local consultant in psychogeriatrics who provided the continuation of care. Since the Memory Clinic was established for the early detection of dementia, only those patients whose diagnosis remained in doubt were followed up in the clinic.

The memory deficit was reversible in the two patients who suffered from the effects of polypharmacy. This is illustrated by the following case reports. Drugs which can impair memory are asterisked.

Case 1 (other diagnosis; memory deficit category 2)

Miss K.A., a 78-year-old woman, was referred to the Memory Clinic in December 1983. The referral had been initiated by her relatives, with whom she lived. In 1975 she had been firmly diagnosed elsewhere as suffering from cerebral atrophy and her relatives had been attending the relatives' support group of The Alzheimer Disease Society for several years. She had equivocal evidence of memory deficit (category 2). On the Kendrick Battery for the Detection of Dementia, she scored in the normal range but close to the cut-off point in both of the subtests (memory/visuospatial speed). She had some impairment of recent memory, and her scores on the Raven's Matrices indicated that her intelligence was below average for her age.

Past medical history: hypertension; hysterectomy.

She was taking oxprenolol hydrochloride*, hydralazine hydrochloride, Lasikal, allopurinol, flurazepam*, piroxicam and medroxyprogesterone. Her medication was gradually reduced until she took only medroxyprogesterone 100 mg *b.d.* and Navidrex-K, one three times per week. She was seen at regular intervals and, as the drugs were reduced, her memory tests improved. The psychogeriatrician did not consider her demented.

Diagnosis: memory deficit due to polypharmacy.

TABLE I
Clinical consensus diagnosis

	Memory deficit Category 1	Category 2	Category 3	
Firm diagnosis				
Alzheimer-type dementia	25 (9)			
Multi-infarct dementia	3 (1)		1	66%
Other diagnoses	2	1 (1)	1 (1)	
Provisional diagnosis				42%
Probable affective disorder		3 (1)	2	
Probable dementia	3	4 (1)		
No diagnosis			5 (2)	

Notes: The vertical axis describes the clinical diagnosis and the horizontal axis the degree of memory deficit. Figures in brackets are numbers of patients for whom referral was initiated by the patient or carer. Patients with other diagnoses, provisional diagnosis or no diagnosis are being followed up (42%).

Case 2 (other diagnosis; memory deficit category 1)

Mrs M.S., a 66-year-old woman, was referred by her family doctor in August 1984 because of increasing forgetfulness. She had been diagnosed elsewhere as suffering from dementia and her CT scan had been reported as showing small diffused areas of low density.

Past medical history: in 1952 she was overweight and treated with amphetamines which caused overexcitement and resulted in her being prescribed chlordiazepoxide which she had taken since. She had also been diagnosed as having Parkinson's disease and had been started on anti-parkinsonian drugs. In addition, she had a history of depression, hypertension and more recently of low back pain. Her medication consisted of Moduretic, carbamazepine*, Diphenal* (diphenihydantoin 100 mg plus phenobarbital 25 mg per tablet), benzhexol*, Buflomedil, Hypnocedon* (a sleeping tablet), chlorpromazine*, phenobarbitone*, diazepam* and biperiden hydrochloride*. Chlordiazepoxide* had been discontinued 2 days prior to her being seen in the Memory Clinic.

On examination she was fully orientated. She had scoliosis of the thoracic spine, but otherwise there were no significant physical abnormalities. She initially had clear evidence of memory deficit (category 1). Her performance was below normal on two memory tests and she became exceedingly upset and confused when required to learn a new task. She was diagnosed as having extensive osteoporosis of the thoracic and lumbar spine and was treated with physiotherapy and paracetamol. She was gradually weaned off all other drugs and improved both mentally and physically. The psychogeriatrician did not consider her to be demented.

Diagnosis: memory deficit due to polypharmacy.

Most of the other patients, 42% of the total series, are being followed up in order to confirm or refute the original diagnosis with particular reference to the diagnosis of early dementia.

Source of referral

In the total series, 26 patients had been referred directly by their family doctors and eight from other hospital departments. The other 16 patients had referred themselves, or their carers had asked the doctor to refer them. Of those who came directly or indirectly via their family doctors, some had not sought medical advice until the disease was far advanced; in other cases the doctor did not seem to be aware of the community support available.

As Table I indicates, there were larger numbers of self-referrals in those with memory deficit category 1 than in categories 2 and 3. Moreover, the 10 self-referred patients in this group had not received any support until they came to the clinic.

Discussion

Several studies have shown that a medical care system that depends upon the self-reporting of illness is severely handicapped in terms of the early detection of disease (Williamson *et al*, 1964; Gruer, 1975). This has particularly deleterious consequences in the elderly. In a study in three general practices in Edinburgh, Williamson *et al* (1964) found that 28% of the elderly patients were demented, but that the condition had not previously been recognised in over four-fifths of the cases.

In our study, it was disconcerting to find that many patients who were clearly demented had not been referred to the appropriate community support service; it was also instructive to learn how difficult it may be for a busy family doctor to recognise an early memory deficit. It is probably hard for a doctor to acknowledge such problems in a patient whom he has known for some years and who retains a good social facade, particularly if the patient is known to be of relatively high intelligence and to have held a responsible position in society. The results give support to Thompson's view (1985) that dementia is a latent condition, often detectable only by challenge.

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* indicates drugs which can impair memory.

The introduction of a new service (e.g. the opening of a geriatric day hospital) often brings to light deficiencies in existing services. We certainly found this to be the case after the Memory Clinic had been in operation for a few months. The Report of the Royal College of Physicians on Organic Mental Impairment in the Elderly (1981) emphasises that the present *ad hoc* crisis style of management of dementia is unsatisfactory; instead there should be a readiness to seek out problems and to respond promptly to patients' needs. Criteria which diseases should fulfil if screening in the general population is to be attempted have been stipulated by Acheson (1963) and by Wilson & Jungner (1968). Recently, Cooper & Bickel (1984) have argued that the criteria also apply to the early detection of dementing disorders in old age. It is generally recognised that for screening a single sign should carry with it a high probability that the disease is present, and eliciting the sign should be simple, economical and unobjectionable to the subject. Both these criteria are fulfilled by dementia. In the Memory Clinic, we have used memory deficit as a marker for dementia, and the assessment of this deficit is based on the history, observation and simple objective testing.

Ideally there should be reasonable prospects for cure of the disease: this does apply to the mental deterioration which can result from polypharmacy and to those with 'pseudodementia' due to affective disorder. However, in the practice of geriatric medicine, cure is not in general the sole aim of treatment, but rather early detection is a preliminary to intervention aimed at reducing disability and postponing the need for institutional care. Bergmann (1979) has drawn attention to the difficulties in making the diagnosis of early or mild dementia. He emphasises the importance of defective memory and early behavioural impairment in the development of dementing syndromes. He also stresses the need for follow-up, since in one epidemiological study (Kay *et al*, 1968) it was found that 30% of patients diagnosed as probable early dementia were not demented when reassessed 3–4 years later.

We have described a team of three different specialists each assessing the patient from a particular aspect. However, the focus of assessment should be on the different tasks involved, not on the different professionals represented in our clinic. These tasks are: the collection of evidence of memory deficit, the identification of detectable causes of this deficit, the detection of underlying depression, and the counselling and management of the patient and the carer(s). All these tasks could be carried out by one suitably qualified person, but in practice, at least two people should be involved to increase the reliability of the overall assessment.

The value of the Memory Clinic can be considered under three headings:

Practical value

It offers a facility to investigate people with memory problems and to attempt to arrive at a correct diagnosis, including the revision of the original diagnosis if found to be incorrect; to establish a diagnosis in patients suffering from the early stages of a dementing illness with a view to improving the patient's management; to treat curable causes of memory loss; and to control psychiatric symptoms.

Educational value

The general public and all health care professionals need reminding that memory failure is not a normal concomitant of old age. Older people in whom suspicion of mental impairment arises or where memory loss is a subjective or objective complaint, should be investigated. Clinics to which early cases can be referred, such as the Memory Clinic, would increase awareness that screening should be undertaken. Suitable screening tests are available and should be administered routinely by all professionals coming into contact with old people. A test which is particularly sensitive to severity of memory deficit is the ability to recall recently learnt new material after an interval (Graham-White *et al*, 1969; Erikson & Scott, 1977). The material should contain at least five items which are to be recalled after an interval of 5 min during which the patient is prevented from rehearsing by involvement in other tasks. The patient should be warned about the need for subsequent recall. This type of test, the recall after a brief interval of a fictitious name and address, is included in many geriatric assessments, e.g. the Mental Test Score (Roth & Hopkins, 1953) and the Cerebral Function Test (Silver, 1972).

Research value

Since the clinic offers follow-up over time to selected patients, it will be possible to describe more accurately the natural history of the various dementing illnesses. A group of patients with early dementia can be identified, from which suitable subjects for trials of potential therapeutic agents may be recruited. If such an agent should be developed, it is more likely to be effective in the early stages of dementia.

Conclusion

This pilot project has brought to light deficiencies in the utilisation of existing services and has confirmed the point made in the Report of the Royal College of Physicians (1981) that a readily accessible and integrated service is essential in order to arrive at a correct diagnosis in patients with early dementia and to facilitate their management. Although one of the aims of the Memory Clinic is to provide a service to family doctors, many people previously unknown to the health service referred themselves or were referred by their doctor only after prompting by others caring for the patient. We believe that memory clinics could be a valuable addition to the geriatric and psychogeriatric services within the NHS and so provide a better system of management for the vast number of elderly demented patients projected for the future.

References

Acheson, R. M. (1963) Thoughts on a service for the pre-symptomatic diagnosis of disease. *Public Health*, **77**, 261–273.

Bergmann, K. (1979) The problem of early diagnosis. In *Alzheimer's Disease: Early Recognition of Potentially Reversible Deficits* (eds A. I. M. Glen & L. J. Whalley). London: Churchill Livingstone.

——, Kay, D. W. K., Foster, E. M., McKechnie, A. A. & Roth, M. (1971) A follow-up study of randomly selected community residents to assess the effects of chronic brain syndrome and cerebrovascular disease. *Psychiatry (Part II) Medical International Congress Series No. 274*. Amsterdam: Excerpta Medica.

Cooper, B. & Bickel, H. (1984) Population screening and the early detection of dementing disorders in old age: a review. *Psychological Medicine*, **14**, 81–95.

Erikson, R. C. & Scott, M. L. (1977) Clinical memory testing: a review. *Psychological Bulletin*, **84**, 1130–1149.

Gibson, A. J. & Kendrick, D. C. (1979) *The Manual for the Kendrick Battery for the Detection of Dementia in the Elderly*. Windsor, UK: NFER Publishing Co.

Graham-White, J., Merrick, M. & Harbison, J. J. M. (1969) Williams scale for the measurement of memory: test reliability and validity in a psychiatric population. *British Journal of Social & Clinical Psychology*, **8**, 141–151.

Gruer, R. (1975) Needs of the elderly in the Scottish borders. *Scottish Health Service Studies No. 33*, 102–103.

Hachinski, V. C., Iliff, L. D. & Zilhka, E. (1975) Cerebral blood flow in dementia. *Archives of Neurology*, **32**, 632–637.

Hare, M. (1978) Clinical checklist for diagnosis of dementia. *British Medical Journal*, **ii**, 266–267.

Henderson, A. S. & Huppert, F. A. (1984) The problem of mild dementia. *Psychological Medicine*, **14**, 5–11.

Hinton, J. & Withers, E. (1971) The usefulness of the clinical tests of the sensorium. *British Journal of Psychiatry*, **119**, 9–18.

Hodkinson, H. M. (1974) Mental impairment in the elderly. *Journal of the Royal College of Physicians*, **7**, 305.

Isaacs, B. (1963) The diagnostic value of tests with toys in old people. *Gerontologica Clinica*, **5**, 8–22.

Kay, D. W. K., Bergmann, K., Foster, E. M. & Garside, R. F. (1968) A four-year follow-up of a random sample of old people originally seen in their own homes: a physical and psychiatric enquiry. *Proceedings, Fourth World Congress of Psychiatry, Madrid*, **3**, 1668–1670. Amsterdam: Excerpta Medica.

Kendrick, D. C., Gibson, A. J. & Moyes, I. C. A. (1979) The revised Kendrick Battery: clinical studies. *British Journal of Social & Clinical Psychology*, **18**, 329–340.

McDonald, C. (1969) Clinical heterogeneity in senile dementia. *British Journal of Psychiatry*, **115**, 267–271.

Miller, E., Pearce, J. M. S., Perry, E. K. & Perry, R. H. (1984) The dementing diseases. In *Dementia: A Clinical Approach* (ed. J. M. S. Pearce). London: Blackwell.

Priest, R. G. & Woolfson, G. W. (1986) In *Handbook of Psychiatry*. London: Heinemann.

Randt, C. T., Brown, E. R. & Osborne, D. P. (1980) A memory test for longitudinal measurement of mild to moderate deficits. *Clinical Neuropsychology*, **4**, 184–194.

Report Of The Royal College Of Physicians On Organic Mental Impairment In The Elderly (1981) *Journal of the Royal College of Physicians*, **15**, 4–29.

Rosen, W. G., Terry, R. D., Fuld, P. A., Katzman, R. & Peck, A. (1980) Pathological verification of ischemic score in differentiation of dementias. *Annals of Neurology*, **7**, 486–488.

Ross, N. & Horne, D. J. (1977) An evaluative study of Silver's cerebral function test. *Journal of the American Geriatrics Society*, **25**, 249–252.

Roth, M. & Hopkins, B. (1953) Psychological test performance in patients over sixty. I. Senile psychosis and the affective disorders of old age. *Journal of Mental Science*, **99**, 439.

Silver, C. P. (1972) Simple methods of testing ability in geriatric patients. *Gerontologica Clinica*, **14**, 110–122.

Simpson, J. M. & Linney, A. D. (1985) The use of computer automated psychological tests to assess mentally impaired old people. In *Interdisciplinary Topics in Gerontology* (ed. F. Clifford Rose); Vol. 20: *Modern Approaches to the Dementias*. Part II: *Clinical and Therapeutic Aspects*. Basel: Karger.

Solomon, S., Hotchkiss, S., Saravay, S. M., Bayer, C., Ramsey, P. & Blum, R. S. (1983) Impairment of memory function by antihypertensive medication. *Archives of General Psychiatry*, **40**, 1109–1112.

Thompson, M. K. (1985) Myths about the care of the elderly. *Lancet*, **i**, 523.

Whitehead, A. & Hunt, A. (1982) Elderly psychiatric patients: a five-year prospective study. *Psychological Medicine*, **12**, 149–157.

Williamson, J., Stokoe, I. H. & Gray, S. (1964) Old people at home: their unreported needs. *Lancet*, **i**, 1117–1120.

Wilson, J. M. G. & Jungner, G. (1968) Principles and practice of screening. *Public Health Paper No. 34*. Geneva: WHO

Wood, R. A. (1984) Clinical algorithms: memory loss. *British Medical Journal*, **288**, 1443–1447.

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Chronic Schizophrenic Disorder
I. Psychophysiological Responses, Laterality and Social Stress

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Bilateral palmar skin conductance and heart rate were measured throughout a series of psychological tests and during both sitting and ambulant social interactions in 14 right-handed men with chronic schizophrenic disorder and 12 healthy volunteers matched for age and handedness. Miniature radio telemetry equipment was used to collect the physiological data. The schizophrenic group was effectively unresponsive to 70-dB auditory stimuli, while all but one of the control group responded and habituated to a nil response by the tenth tone in sequence. The schizophrenic group showed some evidence of increased skin conductance activity at rest, and in socially demanding conditions skin conductance level and variability were increased in the right hand. The present group of electrodermal 'non-responders' was not in general autonomically under-active. Asymmetry of skin conductance activity during social interaction may be a characteristic of chronic schizophrenic disorder.

A number of theories concerning schizophrenic symptom formation have implicated arousal as an important source of primary dysfunction or as a factor involved in exacerbation of the disorder (Kasanin, 1932; Mednick, 1958; Fish, 1961; Lynn, 1963; Venables, 1968; Mirsky, 1969; Ax *et al*, 1970; Epstein & Coleman, 1970; Flekkoy, 1975; Lapidus & Schmolling, 1975; Farley, 1976). The evidence concerning a possible arousal dimension and schizophrenic disorder has remained contradictory with many findings to suggest either excessive or reduced physiological activity (Lynn, 1963; Depue & Fowles, 1973; Jordan, 1974; Venables, 1977; Bernstein *et al*, 1981; Gruzelier & Manchanda, 1982).

One possible explanation for the conflicting evidence, suggested by old and recent reports (Gruzelier & Venables, 1972, Venables, 1977; Gruzelier & Manchanda, 1982), is that in schizophrenic groups there is a bimodal distribution of physiological responsiveness, particularly to low intensity stimuli. Although some attempts to replicate this finding have been largely unsuccessful (Toone *et al*, 1981; Zahn *et al*, 1981), much evidence has supported it (Depue, 1976; Patterson, 1976; Deakin *et al*, 1979; Straube, 1979; Bernstein *et al*, 1981). Several studies suggest that in schizophrenic groups electrodermal responsiveness to tones reflects general physiological, and possibly emotional, responsivity (e.g. Gruzelier & Venables, 1972, 1974; Gruzelier, 1976; Straube, 1979).

Another possible reason for the lack of conclusive results may be that such procedures do not measure responses to meaningful events. The experimental methods used to explore putative changes in diffuse activation have, with few exceptions (e.g. Schooler & Zahn, 1968; Tarrier *et al*, 1979), employed responses to standardised sensory stimuli as indices of physiological activation.

The present investigation had two linked objectives. The first was to compare autonomic activity under circumstances which imposed relatively sustained demands on the subject with responses to standard laboratory procedures. The second objective was to test the hypothesis that social activity in people with schizophrenic disorder is associated with abnormal autonomic responses (Wing & Venables, 1962; Venables, 1968), reflecting either excessive activation or inhibition. In order to rate some social abilities and impose sufficient social stress over a short period, we asked subjects to mime in front of an audience of two people.

We were particularly interested in physiological responses to social interaction and stress, because social dysfunction is important as a component of both schizophrenic deficit states and premorbid rating scales. In addition, social incapacity has been cited as an index of poor outcome (Kokes *et al*, 1977; Johnstone *et al*, 1979). The evidence suggesting that important life events and critical expressed emotion in the families of schizophrenic subjects affect the course of the disorder has strengthened the impression that sustained social stress is an important determinant of relapse.

Asymmetry of autonomic patterning in schizophrenia has assumed prominence in recent years (Gruzelier & Venables, 1974; Gruzelier & Manchanda, 1982). In developing our equipment, the opportunity was taken to record skin conductance (SC) from both sides.

Method

Patients were identified initially by hospital diagnosis and the notes were reviewed. Selection was according to Feighner criteria for schizophrenia (Feighner *et al*, 1972), but all except one of the patients appeared to have had CATEGO features of core schizophrenic disorder (Wing *et al*, 1974) at some time in their clinical career and all had had positive schizophrenic features within the previous 6 months. Twelve were in-patients. Control subjects were selected from members of the public who responded to advertisements for participants and were matched for age. Those with a history of psychological disorder in the previous 10 years or cerebral trauma, or who were taking medication of any kind which might affect the results were excluded. All subjects were paid for participating.

Each subject attended one session. After being introduced to the experiments and being given an explanation of the procedures and equipment, subjects were asked if they agreed to take part. None refused at this stage, although some patients had refused when approached on the ward. The miniature telemetry equipment and electrodes were then attached, every effort being made to keep the subject relaxed. The subject then sat alone in a comfortable chair in a dimly lit, quiet room for the first part of the session which lasted about 60 min. Communication with the experimenter took place via a microphone and loudspeaker to the side of the chair. Another loudspeaker was placed behind and 90 cm above the subject's head for the presentation of tones. A 35-cm colour television was placed to the subject's left and used to display reminders of what the tasks involved. A new message was displayed for each task. All subjects were tested for visual and auditory acuity before taking part.

Bilateral palmar SC measurements were made throughout the session using miniature telemetric equipment with a a resolution of approximately 0.001 μS and a SC range between 50 and 0.04 μS (White & Charles, 1983). One radio transmitting unit was attached to each forearm. Silver–silver chloride electrodes, 9 mm in diameter, were attached to the thenar and hypothenar eminence of each hand using collodion to form a seal around the edge of the electrode. Beckman electrode gel was injected into the electrode chambers. Although this gel is not isotonic, we found that agar-agar gel dehydrated over a period of 2 to 3 h, was unevenly distributed and had other potential disadvantages (Venables & Christie, 1980). Careful comparison of results with Beckman gel or 0.05-M NaCl agar-agar using the present equipment (data available) indicated that the only significant effects of the hypertonic solution were to elevate SC level and to prevent a slow downward drift in SC level over time. Since the aim of the present investigation was to compare groups of subjects, the absolute SC levels are not of critical importance.

SC level was sampled by computer at a rate of 10 Hz. ECG signals from electrodes placed over the subject's sternum were also monitored telemetrically. Heart rate was measured using a QRS peak detector interfaced to the computer system. The physiological data were displayed on a high resolution graphics screen during collection and recorded continuously on floppy disk with time markers every 10 s. A system with two linked computers recorded the physiological data in order to provide continuous collection and storage. A third computer was employed to control psychological procedures whose timing was also stored. A sound recording was made of each session with a time marker recorded on one track of the stereo tape. This permitted the sound replay to be synchronised with the display of SC data during analysis. The microphone was sensitive enough to detect deeper breaths and larger movements of the limbs and trunk. Room temperature was maintained at a comfortable level above 20°C.

The relatively formal psychological procedures occupied approximately 50 min and consisted of: simple reaction time, 5 min sitting quietly, 8 min sitting with a sequence of 10 auditory stimuli, the determination of critical flicker frequency, two flash threshold and set index, a sustained attention test, and a second period of rest with 6 tones. Only physiological results will be reported here.

Before either tone sequence, the subject was told that he should sit quietly for about 10 min, and that he should relax as much as possible without falling asleep. He was also informed that during the later stages of this period there would be occasional tones over the loudspeaker but that they should be ignored as far as possible. A message on the television screen also instructed the subject to ignore any noises. Each 70-dB, 1000-Hz tone was of rapid onset and offset, with rise and fall time of <2 ms and a duration of 500 ms. Tones were spaced at pseudorandom intervals between 30 and 50 s with a mean inter-stimulus interval of 40 s. The sequence of intervals was the same for all subjects.

The later part of the session was designed to provide a series of increasingly stressful social interactions, although a friendly and uncritical atmosphere was maintained throughout. After the third tone of the second series of tones, an experimenter entered the room quietly without looking at the subject and sat in a chair. At the end of the sequence of tones several minutes later, the experimenter introduced himself and said that he would like to ask some questions about how the subject got on with the tests, but before that he had to go out for a moment. He returned after 1 min with a board and a pen and sat down again in the chair, and discussed how the subject had got on in the tests in a semi-structured interview. The subject then read aloud a children's standard comprehension task and answered four questions. After a slight pause, the experimenter said: "The last thing we would like you to do is to pretend to carry out some simple daily activities without using words. Before we do that I am going to fetch somebody else." He went out of the room, waited 30 s and returned with a second experimenter. The light was then turned on, the subject was asked to stand up and the television screen was turned towards him so that experimenters could not see the display.

The subject was then instructed that he would see the topics of his mimes on the television screen and that he should act them without speaking. When he understood what he had to do, there was a delay of about 1 min. The subject then carried out three mimes whose titles have been chosen as relatively easy to perform but susceptible to several interpretations. The first two titles were selected from a list of eight possible activities from daily life such as slicing bread or washing up. The third mime was one of four affects, either anger, fear, sadness or disgust. During the mime performances, the experimenters pretended not to identify the topics so that subjects performed the mime for up to 60 s.

In the analysis of the data, SC responses to the series of 70-dB tones were assessed visually by two experimenters and accepted if they had an onset between 1 and 3 from onset of the stimulus. Peak latency, amplitude and half decay time were calculated for each peak. Thirteen sections of the experimental session, each of 30 s duration, were selected for analysis with the start times in Table I. Before analysis, all physiological recordings were inspected by eye using a high resolution graphics display of both SC channels and heart rate. Within each of the 13 sections, the following SC parameters were calculated: mean SC level for left and right hands in μS and log μS, standard deviation for mean levels in left and right hands, the sum of the deflections in positive or negative direction during the section in each hand (trend), the mean difference in SC level between the two channels, positive and negative peak deflections of more than 2% deviation from baseline (peak count), mean peak amplitude on each side, and cross divergence between the two channels.

TABLE I

Timing of 30 second sections selected for analysis with times from start of first procedure in brackets

(1) 4 min after start of sitting quietly. (8 min)
(2) 2 min before end of tone sequence. (15 min)
(3) 35 s before experimenter's first entry. (60 min)
(4) 5 s after experimenter's entry.
(5) 30 s after start of discussing session.
(6) 10 s after start of reading aloud.
(7) 5 s after start of comprehension questions.
(8) Seated after mime procedure mentioned.
(9) Preparation for miming. After standing. (66 min)
(10) Explaining mime procedures. (30 s after section 9).
(11, 12, 13) 10 s after start of mime 1, 2, 3. (70 min)

The cross divergence measure was developed in order to estimate detailed differences in patterning of SC activity between the two sides. It was calculated by taking the difference between each successive pair of 10-Hz data points in each hand, dividing that difference by the SC level of the first value of the pair, and calculating the mean of the absolute difference between the derived left and right side values for the 299 samples. Lack of divergence is represented by zero: larger values represent increasing divergence.

TABLE II

Details of subjects in the schizophrenic group

Age	*Duration of history (years)*	*Medication (total daily dose for oral medication)*
21	5	Oral flupenthixol 9 mg, chlorpromazine 300 mg
23	3	Depot fluphenazine 25 mg weekly, haloperidol 60 mg
27	3	Depot fluphenazine 100 mg fortnightly, trifluoperazine 30 mg, orphenadrine 150 mg
28	4	Depot fluphenazine 25 mg weekly, haloperidol 120 mg, chlorpromazine 100 mg
31	14	Depot haloperidol 150 mg every 4 weeks, trifluoperazine 30 mg, chlorpromazine 100 mg, procyclidine 5 mg *t.d.s.*
32	6	Depot flupenthixol 100 mg fortnightly, haloperidol 40 mg
33	6	Depot fluphenazine 120 mg 3 weekly, haloperidol 90 mg
36	14	Depot fluphenazine 50 mg fortnightly, chlorpromazine 100 mg, procyclidine 15 mg
39	4	Pericyazine 30 mg, chlorpromazine 225 mg, orphenadrine 50 mg *t.d.s.*
42	13	Clopixol 400 mg, fortnightly, procyclidine 15 mg, temazepam 20 mg *nocte*
54	20	Depot fluphenazine 25 mg weekly, haloperidol 60 mg, chlorpromazine 60 mg
56	31	Depot fluphenazine 50 mg weekly, trifluoperazine 15 mg, chlorpromazine 300 mg
57	30	Trifluoperazine 30 mg, diazepam 15 mg
61	19	Depot fluphenazine 100 mg fortnightly, chlorpromazine 100 mg, trifluoperazine 30 mg

The heart rate data were in the form of inter-beat intervals in milliseconds and these required conversion to beats per minute (60 000/ms count). Mean beat frequency and standard deviation were calculated for each section. Occasional signal loss or highly deviant responses which might have been due to defects in signal transmission were eliminated after visual assessment on the high resolution graphics display.

Histograms of multiple samples from all the variables were assessed by eye for normality of distribution and, if there was doubt about the distribution, they were subjected to analysis for goodness of fit to normality using the χ^2 statistic.

The levels of statistical analysis for the electrodermal variables and heart rate included: differences between groups for mean values across the 13 samples and across the two basal and later social sequence (sections 3–13), analysis of trends across 13 samples, and sequential sample by sample analysis for differences under the varying experimental conditions. One- or two-way analysis of variance, with repeated measures analysis when appropriate, was employed.

The mean, s.e.m. and range of age in the two groups were similar (control group: 40.5 ± 3.93 years, range 25–61; schizophrenic group: 37.7 ± 3.57 years, range 21–61) and all subjects were right-handed. Eight members of the control group and all members of the schizophrenic group were male. Some clinical characteristics of the schizophrenic group subjects are given in Table II.

Results

Of the subjects in the schizophrenic group, electrodermal responses to the tones in the initial tone sequence occurred on one occasion in each of three subjects (after tones 1, 3 and 9 respectively) but otherwise no responses to tones were seen. In the control group, clear electrodermal responses were seen in 11 out of 12 subjects, one of whose responses failed to habituate by the tenth tone, and a steady decline in SC response amplitude was observed in responding subjects. Responses were also absent in all subjects in the schizophrenic group during the later tone sequence when only five of the control group had electrodermal responses to the tones.

Group mean results for electrodermal variables and heart rate across the 13 sections are shown in Table III. No major differences between the groups were found among the electrodermal variables, but trends towards increased coefficient of variability and cross divergence (higher values) were seen in the right hand of the schizophrenic group when compared with the control group.

TABLE IV

Two-way repeated measures analysis of variance (group × hand) of lateralised electrodermal variables across the basal two sections (d.f. = 1, 1) and the 11 sections in the social sequence (d.f. = 1, 10)

	Groups		*Hands*		*Interaction*	
	F	*P*	*F*	*P*	*F*	*P*
Basal sections						
SC level	6.73	0.24	75.78	0.07	29.75	0.01
SC CV	133.33	0.05	0.24	0.70	0.16	0.75
Peak frequency	39.70	0.10	967.86	0.02	8.99	0.21
Peak amplitude	2.91	0.34	0.20	0.73	2.37	0.37
Social sequence						
SC level	21.81	0.00	533.43	0.00	54.08	0.00
SC CV	54.76	0.00	10.09	0.01	17.8	0.00
Peak frequency	0.51	0.50	0.38	0.56	1.02	0.34
Peak amplitude	5.12	0.98	29.23	0.00	4.57	0.06

The results of two-way repeated measures analysis of variance for the lateralised electrodermal variables across the two basal sections and across the later sequence are shown in Table IV. In the basal sections there was

TABLE III

Grand mean results for electrodermal parameters and heart rate across 13 sections, analysed using one-way analysis of variance

	Control group n = 12		*Schizophrenic group n = 14*			
	mean	*s.e.m.*	*mean*	*s.e.m.*	*F*	*P*
SC[1] level left (μS)	3.95	0.36	3.95	0.35	0.00	0.97
SC level right (μS)	4.19	0.43	5.06	0.68	1.12	0.30
SC CV left	0.03	0.004	0.03	0.005	0.93	0.65
SC CV right	0.03	0.003	0.04	0.006	3.09	0.09
Difference between sides in SC level (R – L, μS)	0.25	0.34	1.11	0.52	0.00	0.98
Peak frequency left[2]	3.56	0.57	3.54	0.24	0.03	0.85
Peak frequency right	3.41	0.64	3.59	0.78	0.53	0.52
Peak amplitude left (μS)	0.18	0.03	0.15	0.02	0.53	0.52
Peak amplitude right (μS)	0.22	0.04	0.27	0.05	1.90	0.17
Cross divergence (nS)	0.68	0.09	1.00	0.16	2.90	0.10
Heart rate (beats/min)	74.77	2.56	92.05	3.37	14.5	0.00

1. SC = skin conductance; CV = coefficient of variation (s.d./mean).
2. Peak frequency in counts/30 s with a 2% deflection from baseline criterion.

(a) a significant interaction between groups and hands in skin conductance level, reflecting a higher mean right-hand value in both groups,
(b) a weakly significant effect for groups in coefficient of variation due to a greater variability in the schizophrenic group,
(c) a significant hands effect for peak amplitude due to increased peak amplitude in the right hand of both groups.

The difference between groups in peak frequency in the basal sections reached a 0.1 level of probability.

In the analysis of the later, social sequence (sections 3–13), strong effects for groups, hands and their interaction were found for skin conductance level and variability, reflecting the marked elevation of skin conductance level and variability in the right hand of the schizophrenic group. Peak frequency was not increased in either group or hand. An increase in peak amplitude on the right side in both groups was reflected in the significant effect for peak amplitude for hands, with a trend to a group × hands interaction.

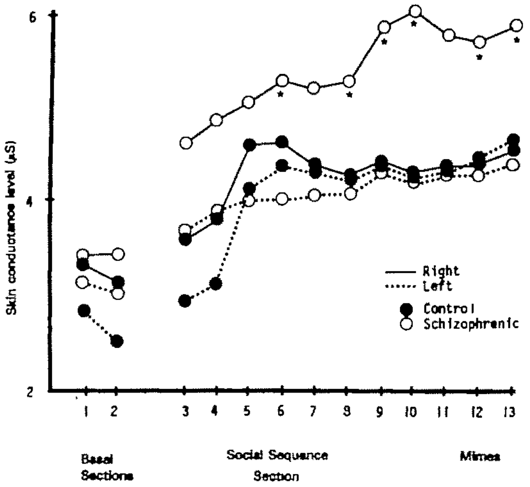

FIG. 1 Group mean skin conductance levels in 13 sections in control and schizophrenic groups. Significant difference between hands in schizophrenic group: $^*P<0.05$, using repeated measures analysis of variance.

Within groups, analysis of changes in the electrodermal variables across the 13 sections using one-way repeated measures analysis of variance yielded significant changes ($P<0.02$) in the control group for all variables except peak amplitude. The results were similar in the schizophrenic group except that no significant effect was seen for peak frequency or coefficient of electrodermal variability.

Evaluation of section by section changes in SC level in either hand using one-way repeated measures analysis of variance (Fig. 1, Table V) indicated that in the later sequence (sections 3–13) significant mean increases in both hands occurred in the control group after the experimenter came into the room (between sections 3 and 4) and when starting to discuss the session (between sections 4 and 5), matched by similar but weaker trends in the schizophrenic group.

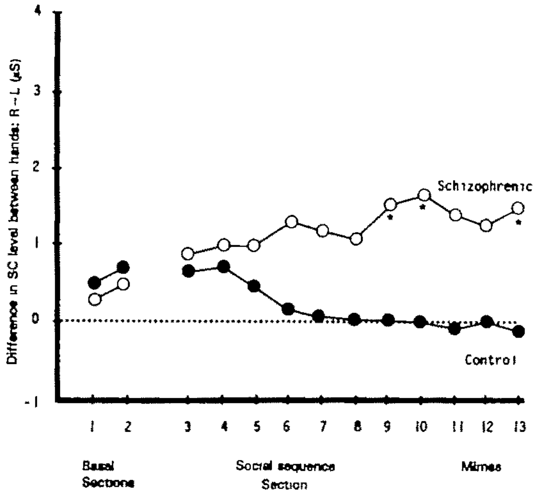

FIG. 2 Group mean differences in skin conductance level between hands in control and schizophrenic groups. Right hand minus left. Significant difference between groups using one-way analysis of variance: $^*P<0.05$.

A large increase in SC level in both hands was observed in the schizophrenic but not the control group when standing in anticipation of miming (between sections 9 and 10), while small but similar increases were seen in both groups between the second mime (of a daily activity) and the mime of an emotional state (between sections 12 and 13).

Section-by-section analysis of the change in difference in SC level between hands using repeated measures analysis of variance indicated no statistically significant effects in the control group but significant increases in the schizophrenic group in right-hand level over left when starting to read aloud (between sections 5 and 6: $F\,[1, 13] = 6.10$, $P=0.03$) and after standing up in anticipation of miming (between sections 8 and 9: $F\,[1, 13] = 17.56$, $P=0.001$). Figure 2 shows the tendency to equalisation of SC level in the two hands in the control group in later sections with a divergence between the hands in the schizophrenic group, reflected in a difference ($P<0.05$) in SC level between hands in the schizophrenic group in several of the later sections (Fig. 1). Between sections 3 (before the experimenter's entry) and 7 (comprehension questions), an electrodermal left shift was seen in nine members of the control group and a right shift was found in ten members

TABLE V

Significant ($P<0.05$) group mean changes in skin conductance level (μS) between sections

	Left hand				*Right hand*			
Sections	*Mean*	*s.e.m.*	*F*	*P*	*Mean*	*s.e.m.*	*F*	*P*
Control group								
Experimeter enters (4–3)	0.19	0.04	19.43	0.001	0.22	0.08	7.96	0.010
Discussion (7 – 6)	1.02	0.30	11.54	0.006	0.77	0.22	12.27	0.002
Mime 3 (13 – 12)	0.25	0.11	5.15	0.042	0.16	0.07	5.63	0.025
Schizophrenic group								
Experimenter enters (4 – 3)	0.17	0.12	2.00	0.179	0.27	0.13	4.34	0.055
Discussion (7 – 6)	0.12	0.07	3.31	0.089	0.18	0.08	5.02	0.041
Pre-mime (9 – 8)	0.25	0.04	38.09	0.000	0.56	0.08	46.07	0.000
Mime 3 (13 – 12)	0.11	0.05	5.38	0.036	0.22	0.10	4.99	0.042

of the schizophrenic group, the others showing small changes in the opposite direction.

Mean cross divergence was increased in the two basal sections in the schizophrenic group (control group 0.253 ± 0.049 nS per sample, schizophrenic group 0.651 ± 0.172 nS per sample: F [1, 24] = 4.36, $P=0.05$), but a trend in the same direction was weaker in the social sequence (control group 0.750 ± 0.098 nS per sample, schizophrenic group 1.076 ± 0.165 nS per sample: F [1, 24] = 2.75, $P=0.11$]. There was no significant difference in any of the electrodermal measures between the small group of women and the men in the control group.

The mean heart rate of the schizophrenic group was significantly higher ($P<0.01$) than that in the control group in all sections, but the amount of change in heart rate under varying circumstances did not differ between the two groups.

Discussion

Since we were breaking new ground by using radio telemetry for measurement of SC in a group of people, it was necessary to establish the reliability of the system. In general, reliable and sensitive measurement of SC and heart rate under ambulant conditions proved possible. Subjects quickly became used to wearing the small transmitter units.

Our aims in extending the range of techniques of psychophysiological measurement in this way were to compare the autonomic responses of a schizophrenic group in a social setting with responses to standardised low intensity tones, and to examine the possibility that people with chronic schizophrenic disorder might show excessive excitatory or inhibitory responses to social stress. The virtual absence of responses to tones in the schizophrenic group, other than in one subject who could be classified as a fast habituator (Patterson & Venables, 1978), was unexpected but provided the opportunity to assess other autonomic response characteristics of a 'non-responder' group.

There was no evidence to suggest that non-responsivity to low intensity tones in our sample was associated with reduced SC responsivity or variability in the schizophrenic group while sitting at rest or during the socially active sections approximately 1 h after the start of recording. Indeed, a number of findings indicated greater physiological activity than in the control group: a bilateral increase in SC variability associated with a trend to increased peak frequency when sitting early in the session, a trend towards bilaterally increased SC variability in some of the socially demanding sections even after compensation for differences in SC level, an overall increase in SC level and peak amplitudes on the right side in the later sections, and increased heart rate throughout.

Shorter-term autonomic responsivity in later sections in both groups varied with circumstances but was not generally increased or reduced in the schizophrenic group. The major exception was the electrodermal response which took place during the preparation for miming. This is difficult to evaluate because in the schizophrenic group electrodermal activity, particularly on the right side, changed when the subjects stood up. No comparable alteration was evident in the control group, and changes in heart rate in the groups were similar and at least partly attributable to alteration in posture. As SC level on the right in the schizophrenic group continued to rise in section 10 whilst miming was further explained, it seems probable that the changes from section 8 to 10 were mainly psychological in origin, but a greater separation of standing up and preparation for miming would have been preferable. Our pilot studies with healthy volunteers and hospital patients had led us not to expect effects on electrodermal activity due to standing up.

Contrary to our prediction, the sections when subjects mimed repeatedly in front of two experimenters were not accompanied by bilateral evidence of abnormal autonomic responses in the schizophrenic group when compared with controls. This might have reflected lack of social engagement, but participation and mime ability were not impaired in the schizophrenic group (White *et al*, 1987). Since the bulk of experimental evidence suggests that unmedicated subjects with schizophrenic disorder show raised SC levels (Lader, 1975; Gruzelier & Hammond, 1978; Bernstein *et al*, 1981), it is probable that SC level was reduced in our sample by neuroleptic treatment. Even so, the prediction of augmented responsiveness to social stress was partially correct since electrodermal activity was increased on the right side.

The most striking findings were the persistently increased heart rate and the electrodermal laterality effect. Elevated heart rate in schizophrenic groups is perhaps the most consistent finding in the literature (Depue & Fowles, 1973; Lader, 1975; Zahn *et al*, 1981) and may in part be ascribed to medication.

The electrodermal laterality effect was seen most clearly when the shifts in SC level between the start and end of the social section in the two groups were compared (Fig. 2). The effect began during questioning about the tests and reading aloud, while the subject was still seated, and was enhanced during the miming section.

The lateralised difference in SC level between the groups is consistent with previous reports (Gruzelier, 1973; Gruzelier & Manchanda, 1982), in which enhanced autonomic responsiveness to tones on the right in people with schizophrenic disorder has been the most frequent finding. Such right-sided enhancement of responses to tones was recently found by Gruzelier & Manchanda (1982) to be associated with 'negative' or Bleulerian symptoms. Bernstein *et al* (1981) reported that larger right-hand responses to tones were associated with increased EEG alpha blockade in the left hemisphere, indicating a physiological hemispheric activation effect, but that this link was not specific to a schizophrenic diagnosis. Gruzelier & Venables (1974) have also reported a sustained right-hand shift in electrodermal level during a psychological task with an increase in right-hand excess as SC levels increased. Other reports of sustained situational measures (e.g. Tarrier *et al*, 1979; Sturgeon *et al*, 1981) have recorded SC activity from one hand only, usually the left hand, and have not assessed lateral asymmetry.

The interpretation of the electrodermal laterality effects depends in part on evidence concerning anatomical lateralisation of central nervous control of electrodermal activity. Suggestions that limbic structures in the temporal lobe could mediate lateralised autonomic effects remain unsupported by relevant evidence in animals or man (see review by Toone *et al*, 1981), but the recent demonstration of left-sided elevation of dopamine concentrations in the amygdala of post mortem brains from schizophrenic subjects (Reynolds, 1983) might reflect relevant limbic changes.

Lateralisation of influences from other sites must also be considered. Manipulation of the cerebral cortex in cats, either in the 'limbic' area on the medial surface of a cerebral hemisphere (Isamat, 1961) or in the premotor area 6 (Schwarz, 1937), can produce preferential alterations in electrodermal activity on the contralateral side. In man, Gruzelier & Manchanda (1982) cited neurosurgical evidence in favour of contralateral electrodermal modulation from frontotemporal sites. Furthermore, early reports clearly suggested contralateral influences on sweating from the frontal, premotor cortex (Kennard *et al*, 1934; Kennard, 1935; Netsky & Starr, 1948). Despite the general lack of consistency in findings of asymmetrical SC activity (Freixa i Baque *et al*, 1984), it would appear inappropriate to exclude an anatomical basis for contralateral SC control at a cerebral level.

If contralateral control of electrodermal does occur, then our data would support suggestions of abnormal cerebral activity in the dominant hemisphere in people with schizophrenic disorder.

In interpreting the non-responsiveness to tones in our schizophrenic group, several factors require consideration. The bulk of reports indicate that non-responsivity to low intensity tones correlates with generally reduced autonomic activity (Gruzelier & Venables, 1972; Gruzelier, 1976; Straube, 1979). Since our data are inconsistent with those reports and since we had no responding schizophrenic group for comparison, conclusions from our results can only be tentative. We have no reason to suspect that methodological factors affected responding to tones. In other work, non-responsivity to low intensity tones has not been accompanied by reduced responsivity under other standardised stimulus conditions (Gruzelier *et al*, 1981), or by altered EEG changes after tone stimuli (Bernstein *et al*, 1981). Gruzelier & Manchanda (1982) recently commented that responsivity to tones reflects factors which are relatively specific to the tone response procedure, while sustained changes in SC level are more likely to reflect arousal.

Another pertinent issue is that of the physiological need for increased pseudomotor activity on hands under some circumstances. Some of the determinants of SC responses appear to be emotional, and others more cognitive or anticipatory of motor function (Germana, 1969; Bernstein, 1979; Maltzman, 1979; O'Gorman, 1979; Lang *et al*, 1980). There does appear to be agreement in the literature that electrodermal responses to intermittent abrupt events, such as tone stimuli, depend on sensory input analysers which are largely unconscious and, at least at times, cognitively sophisticated. Therefore, such responses may be sensitive to cognitive disruption as well as to putative general changes in activation, and so theoretically unsuitable as a measure of arousal in people susceptible to schizophrehnic disorganisation. The reports by Straube (1979) and Bernstein *et al* (1981), suggesting that non-responding to tones was associated with increased cognitive disorganisation in a schizophrenic group, are compatible with this hypothesis.

With regard to the effect of medication, although it remains possible that a bimodal distribution of responding is somehow revealed by medication, sustained treatment with neuroleptics appears not to modify transient responses to low intensity auditory stimuli and previous results have in general not supported a pharmacological interpretation of electrodermal non-responding (Venables, 1977; Straube, 1979; Bernstein *et al*, 1981).

The incongruity between normal or increased levels of electrodermal activity and increased heart rate and non-responsiveness to tones in our schizophrenic group may be explained by inadequacy of the tone response procedure as an index of arousal in schizophrenia. Sustained differences between the groups under socially active conditions, as used in the present investigation, appear to be appropriate and practicable indices of a possible underlying dimension of arousal and may indicate the time course of excessive psychophysiological responses, despite the relatively poor standardisation of experimental conditions when compared with tone presentation procedures.

The hypothesis that social dysfunction is a result of excessive activation is supported at least partially by our results. If SC activity is controlled from the contralateral side of the brain, the data may support the hypothesis of dysfunction in the dominant hemisphere in at least some patients with persistent schizophrenic disorder, but further information is required to substantiate such a suggestion.

References

Ax, A. F., Bamford, J. L., Beckett, P. G. S., Fretz, N. F. & Gottlieb, J. S. (1970) Autonomic conditioning in chronic schizophrenia. *Journal of Abnormal Psychology*, **76**, 140–154.

Bernstein, A. S. (1979) The orienting response as novelty and significance detector: reply to O'Gorman. *Psychophysiology*, **16**, 263–273.

——, Taylor, K. W., Starkey, P., Juni, S., Lubowsky, J. & Paley, H. (1981) Bilateral skin conductance, finger pulse volume, and EEG orienting response to tones of differing intensities in chronic schizophrenics and controls. *Journal of Nervous and Mental Disease*, **169**, 513–528.

Deakin, J. F. W., Baker, H. F., Frith, C. D., Joseph, M. H. & Johnstone, E. C. (1979) Arousal related to excretion of noradrenaline metabolites and clinical aspects of unmedicated chronic schizophrenic patients. *Journal of Psychiatric Research*, **15**, 57–65.

Depue, R. A. (1976) An activity-withdrawal distinction in schizophrenia: behavioural, clinical, brain damage and neurophysiological correlates. *Journal of Abnormal Psychology*, **85**, 174–185.

—— & Fowles, D. C. (1973) Electrodermal activity as an index of arousal in schizophrenics. *Psychological Bulletin*, **79**, 233–238.

Epstein, S. & Coleman, M. (1970) Drive theories of schizophrenia. *Psychosomatic Medicine*, **32**, 113–140.

Farley, J. D. (1976) Phylogenetic adaptations and the genetics of psychosis. *Acta Psychiatrica Scandinavica*, **53**, 173–192.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

Fish, F. J. (1961) A neurophysiological theory of schizophrenia. *Journal of Mental Science*, **107**, 828–839.

Flekkoy, K. (1975) Psychophysiological and neurophysiological aspects of schizophrenia. *Acta Psychiatrica Scandinavica*, **51**, 234–248.

Freixa i Baque, E., Catteau, M., Miossec, Y. & Roy, J. (1984) Asymmetry of electrodermal activity: a review. *Biological Psychology*, **18**, 219–239.

Germana, J. (1969) Central efferent processes and autonomic–behavioural integration. *Psychophysiology*, **6**, 78–90.

Gruzelier, J. H. (1973) Bilateral asymmetry of skin conductance orienting activity and levels in schizophrenics. *Biological Psychology*, **1**, 21–42.

—— (1976) Clinical attributes of schizophrenic skin conductance responders and non-responders. *Psychological Medicine*, **6**, 245–249.

—— & Venables, P. H. (1972) Skin conductance orienting activity in a heterogeneous sample of schizophrenics: possible evidence of limbic dysfunction. *Journal of Nervous and Mental Disease*, **155**, 277–287.

—— & —— (1974) Bimodality and lateral asymmetry of skin conductance orienting activity in schizophrenics: Replication and evidence of lateral asymmetry in patients with depression and disorders of personality. *Biological Psychiatry*, **8**, 55–73.

—— & Hammond, N. V. (1978) The effect of chlorpromazine upon psychophysiological, endocrine and information processing measures in schizophrenia. *Journal of Psychiatric Research*, **14**, 167–182.

—— & Manchanda, R. (1982) The syndrome of schizophrenia: relations between electrodermal response, lateral asymmetries and clinical ratings. *British Journal of Psychiatry*, 141, 488–495.

——, Eves, F. F., Connolly, J. F. & Hirsch, S. R. (1981) Orienting, habituation, sensitisation and dishabituation in the electrodermal system of consecutive, drug-free, admissions for schizophrenia. *Biological Psychology*, **12**, 187–209.

Isamat, F. (1961) Galvanic skin responses from stimulation of limbic cortex. *Journal of Neurophysiology*, **24**, 176–181.

Johnstone, E. C., Frith, C. D., Gold, A., & Stevens, A. (1979) The outcome of severe acute schizophrenic illness after one year. *British Journal of Psychiatry*, **134**, 28–33.

Jordan, L. S. (1974) Electrodermal activity in schizophrenics: further considerations. *Psychological Bulletin*, **81**, 85–91.

Kasanin, J. (1932) Pavlov's theory of schizophrenia. *Archives of Neurology*, **23**, 210–218.

Kennard, M. A. (1935) Vasomotor disturbances resulting from cortical lesions. *Archives of Neurology and Psychiatry*, **33**, 537–545.

——, Viet, H. R., & Fulton, J. F. (1934) The syndrome of the premotor cortex in man. *Brain*, **57**, 69–84.

Kokes, R. F., Strauss, J. S. & Klorman, R. (1977) Premorbid adjustment in schizophrenia. *Schizophrenia Bulletin*, **3**, 99–106.

Lader, M. H. (1975) *The Psychophysiology of Mental Illness*. London: Routledge & Kegan Paul.

Lang, P. J., Kozak, M. J., Miller, G. A., Levin, D. N. & McLean, A. (1980) Emotional imagery: conceptual structure and pattern of somato-visceral response. *Psychophysiology*, **17**, 179–192.

Lapidus, L. B. & Schmolling, P. (1975) Anxiety, arousal and schizophrenia: A theoretical integration. *Psychological Bulletin*, **82**, 689–710.

Lynn, R. (1963) Russian theory and research on schizophrenia. *Psychological Bulletin*, **60**, 456–498.

Maltzman, I. (1979) Orienting reflexes and significance: a reply to O'Gorman. *Psychophysiology*, **16**, 274–282.

Mednick, S. A. (1958) A learning theory approach to schizophrenia. *Psychological Bulletin*, **55**, 316–327.

Mirsky, A. F. (1969) Neurophysiological basis of schizophrenia. *Annual Review of Psychology*, 321–348.

Netsky, M. G. & Starr, H. (1948) Autonomic functions of the frontal lobes: studies on patients with head trauma. In *The Frontal Lobes* (eds J. F. Fulton, C. D. Aring & S. B. Wortis). Baltimore: Williams & Wilkins.

O'Gorman, J. G. (1979) The orienting reflex: novelty or significance detector. *Psychophysiology*, **16**, 253–262.

Patterson, T. (1976) Skin conductance responding/nonresponding and pupillometrics in chronic schizophrenia: a confirmation of Gruzelier & Venables. *Journal of Nervous & Mental Disease*, **163**, 200–209.

—— & Venables, P. H. (1978) Bilateral skin conductance and skin potential in schizophrenic and normal subjects: the identification of the fast habituator group of schizophrenics. *Psychophysiology*, **15**, 556–560.

Reynolds, G. P. (1983) Increased concentrations and lateral asymmetry of amygdala dopamine in schizophrenia. *Nature*, **305**, 577–578.

Schooler, C. & Zahn, T. P. (1968) The effect of closeness of social interaction on task performance and arousal in chronic schizophrenia. *Journal of Nervous and Mental Disease*, **147**, 394–401.

Schwarz, H. G. (1937) Effect of experimental lesions of the cortex on the psychogalvanic reflex of the cat. *Archives of Neurology and Psychiatry*, **38**, 308–320.

Straube, E. R. (1979) On the meaning of electrodermal nonresponding in schizophrenia. *Journal of Nervous and Mental Disease*, **167**, 601–611.

Sturgeon, D., Kuipers, L., Berkowitz, R., Turpin, G. & Leff, J. (1981) Psychophysiological responses of schizophrenic patients to high and low expressed emotion relatives. *British Journal of Psychiatry*, **138**, 40–45.

Tarrier, N., Vaughn, C., Lader, M. H. & Leff, J. P. (1979) Bodily reactions to people and events in schizophrenics. *Archives of General Psychiatry*, 36, 311–315.

Toone, B. K., Cooke, E. & Lader, M. H. (1981) Electrodermal activity in the affective disorders and schizophrenia. *Psychological Medicine*, **11**, 497–508.

Venables, P. H. (1968) Experimental psychological studies of chronic schizophrenia. In *Studies in Psychiatry* (eds M. Shepherd & D. L. Davies). Oxford: Oxford University Press.

—— (1977) The electrodermal psychophysiology of schizophrenics and children at risk for schizophrenia: controversies and developments. *Schizophrenia Bulletin*, **3**, 28–48.

—— & Christie, M. J. (1980) Electrodermal activity. In *Techniques in Psychophysiology* (eds I. Martin & P. H. Venables). London: John Wiley.

White, C. de B. & Charles, I. P. (1983) Telemetric skin conductance with computer interface. *Psychophysiology*, **20**, 597–599.

——, Farley, J. & Charles, I. P. (1987) Chronic schizophrenic disorder II. Reaction time, social performance and arousal. *British Journal of Psychiatry*, **150**, 374–380.

Wing, J. K. & Venables, P. H. (1962) Level of arousal and the subclassification of schizophrenics. *Archives of General Psychiatry*, **7**, 114–119.

——, Cooper, J. E. & Sartorius, N. (1974) *Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

Zahn, T. P., Carpenter, W. T. & McGlashan, T. H. (1981) Autonomic nervous system activity in acute schizophrenia, I & II. *Archives of General Psychiatry*, **38**, 251–266.

British Journal of Psychiatry (1987), **150**, 374–379

Chronic Schizophrenic Disorder

II. Reaction Time, Social Performance and Arousal

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Reaction time, set index, critical flicker frequency, two-flash threshold, a sustained performance task and psychophysiological parameters from people with chronic schizophrenic disorder ($n = 14$) and a control group ($n = 12$) were analysed to assess the association between arousal, attentional dysfunction and social dysfunction in schizophrenia. Shorter reaction time, rated ability to mime and, in the schizophrenic group, scores on Venable's ward activity scale correlated with each other. In the schizophrenic group, prolonged reaction time latency correlated positively with skin conductance level in the right hand and negatively with skin conductance variability in the left hand, the latter being in the opposite direction to that for the control group. The results may provide support for the hypothesis that lateralised cerebral dysfunction is associated with performance deficits in people with chronic schizophrenic symptoms.

Several investigations have indicated that, despite the achievement of reliability in the measurement of 'positive' symptoms of schizophrenic disorder, such features have only weak predictive value for clinical and social outcome (Hawk *et al*, 1975; Vaillant, 1978; Johnstone *et al*, 1979; Kendell *et al*, 1979). Although outcome has limitations as an index of psychopathology (Strauss & Carpenter, 1977; Stephens, 1978), chronicity remains of clinical importance and may reflect severity of disorder. The best predictor of poor outcome other than previous chronicity appears to be poor social adjustment (Kokes *et al*, 1977; Strauss & Carpenter, 1977; Johnstone *et al*, 1979; Ciompi, 1980). Therefore, elucidation of the factors underlying social difficulty, and possibly negative symptoms, may be of value in understanding the nature of schizophrenic psychopathology.

The objectives of the present investigation were first to assess social performance in people with chronic schizophrenic disorder using two procedures: Venables's (1957) ward activity scale and the subject's ability to mime; and secondly to evaluate the relationship of scores on those measures to peripheral and cerebral measures of arousal, reaction time latency and attention. Other than miming, the procedures were selected from those in the literature which have shown reliable performance deficits in people with schizophrenic symptoms (Shakow, 1962; Kornetsky, 1972; Nuechterlein, 1977).

Method

Each subject attended one session which lasted about 90 min. The subject sat alone in a comfortable chair in a dimly lit, quiet room during the relatively formal procedures. Communication with the experimenter took place via a microphone and loudspeaker to the side of the chair. A 7-cm square array of 16 high brightness light-emitting diodes (LEDs) (total luminance 400 mcd) was placed on a table in front and slightly to the right of the subject, approximately 60 cm from his eyes, with a small dimly lit LED in the centre of the array for visual fixation. A 35-cm colour television was placed to the subject's left and used to display reminders of what the tasks involved. A new message was displayed for each task.

On either arm of the chair there was a bounce-free, Hall effect switch. These were used during all the active procedures for reaction timing and choice indication. The subjects were instructed to rest their index fingers lightly on the switches when performing a task and to tap them quickly when making a response. During the reaction time tasks the dominant hand was employed. All subjects were tested for visual and auditory acuity before participation.

Bilateral skin conductance and heart rate were recorded telemetrically throughout the session as described elsewhere (White *et al*, 1987).

The early part of the session occupied by formal psychological procedures lasted approximately 60 min and consisted of the following sequence:

1. simple reaction time,
2. 5 min sitting at rest,
3. 12 min sitting with ten intermittent tone stimuli,
4. critical flicker frequency,
5. two-flash threshold,
6. set index,
7. a sustained performance task,
8. 5 min sitting with six tone stimuli.

A short reaction time procedure was placed at the start of the sequence in order to familiarise the subjects with a simple task. The instructions were to watch the lights and to press the response button as quickly as possible when they lit up. Several practice attempts were given. The stimulus sequence consisted of 16 illuminations of the light array (rise time $<20\ \mu s$) with an inter-stimulus interval (ISI) of either 2, 4 or 7.5 s in a pseudorandom order.

Both critical flicker frequency (CFF) and two-flash threshold (TFT) were measured using two procedures: (a) a staircase procedure in which the sequence started at clear separation of flashes and progressed through threshold to a fused stimulus and back to the initial inter-flash interval. (b) A successive approximation procedure (Taylor & Creelman, 1967), in which changes in flicker rate were presented in a stepped sequence determined by the computer, following rules which allowed the subject repeatedly to cross his threshold from either a flicker or fused direction until he reached a criterion step size which terminated the sequence.

During the CFF procedure, subjects were instructed to keep their gaze fixed on the small central LED and to respond with the right-hand button if the lights were flickering when they were lit and with the left-hand button if not. They were instructed to make a choice on every occasion, but the timing interval and display were terminated if no response had occurred within 5 s of stimulus onset. Mark to space ratio in the flicker display was unity. The visual fixation LED flickered with the rest of the display but was left on during the ISI.

After several practice attempts, the first sample of responses was collected using the staircase procedure. An approximate threshold was selected from the staircase procedure as the flicker interval perceived as just flickering in both the ascending and descending directions, and used as the starting interval for a successive approximation procedure which followed. In this latter section the subject completed four runs of which the first and third started at a flicker interval 20 ms above threshold and the second and fourth 20 ms below. If the starting interval in a run was less than 20 ms, the minimum increment was changed to 500 μs and the starting points were 10 ms above or below threshold. The terminating criterion for each run was a step size of 1 ms. Four estimates of threshold were obtained in this fashion and the mean of these taken for further analysis. If the subject became stuck in a series of widely swinging oscillations, the experimenter had the facility to change the terminating criterion. In practice, the need for this did not arise with CFF.

The procedures used to determine TFT were similar to those for CFF with an initial staircase sequence followed by a forced choice successive approximation procedure. The subject was instructed that the task was similar to that for flicker but that only two flashes would be presented and he had to watch carefully for the warning signal when the central LED went out and for the flash stimuli 1 s later. If two flashes were visible, he was to press the right-hand button, and if only one flash was seen, the left. The mean duration of each flash was approximately 50 ms, but the stimulus sequence (on–off–on) was adjusted by the computer so that total duration was constant at 140 ms, and light intensity was adjusted on a trial-by-trial basis by the computer to compensate for flash duration so that perceived light intensity was constant. The formula for this adjustment was determined by the experimenter during the development of the programme.

The CFF and TFT data were recorded as critical flicker and two flash intervals (cycle length) rather than frequency (interval = 1000/Hz). In the set index procedure (Rodnick & Shakow, 1940; Nuechterlein, 1977), reaction time measurements were made using the same apparatus as in the early reaction time section, but the test was divided into two halves. In the first half, 40 light stimuli were presented with a balanced but pseudorandom order of ISIs with ten presentations at each ISI. In the second half, the same number of stimuli were presented with the ISIs in blocks of ten of 2, 4, 7.5 and 15 s. Each subject received the same sequence of ISIs. The subject was told that in the first half the intervals between the onset of lights would be unpredictable, but that during the second half the intervals between the lights would be the same for ten stimuli at a time and that he could use this to anticipate when the next stimulus would occur. A reminder of this was given at the start of the second half of the task. Failures to respond were counted and excluded from analysis. Mean response times for each ISI in each half were calculated and the set index for each subject calculated according to the formula derived by Rodnick & Shakow (1940).

In the sustained performance test, subjects were presented with either the word 'LEFT' or the word 'RIGHT' on the television screen, with a display period of 100 ms and an ISI of 1100 ms, for 60 trials. They were instructed to react only to the word right (target stimulus) which occurred on 20 occasions. Response and error rates and mean reaction times were calculated. This procedure was designed to be similar to the continuous performance test (Kornetsky, 1972), but simplified to allow for the relative lack of practice time. Subjects practised until they achieved correct responses on several trials.

The sequencing of the social sections has been described elsewhere (White *et al*, 1987). In the later stages of the sequence, subjects performed three mimes whose titles were selected so as to be relatively easy to perform but susceptible to multiple interpretations. Two experimenters attempted to guess the mime titles, but pretended not to recognise the mimes so that the subject had to continue to act for up to 60 s. The two experimeters scored each of three mimes for each subject for mime ability (how recognisable was the mime? Score 1–3) and amount of movement (1–4), and overall for expressed embarrassment and difficulty (1–5) and expressed affect (1–5). Mime ability and movement scores were the total of the ratings for each of three mimes.

Clinical features were assessed using the Present State Examination (Wing *et al*, 1974) and the Krawiecka scale (Krawiecka *et al*, 1977). Venables's (1957) ward scale for activity and sociability was completed by members of the nursing staff for 11 of the 12 in-patients.

All the grouped data were assessed for goodness of fit to normality and between-group comparisons for individual measures were carried out using analysis of variance. Mean values are given as mean ± s.e.m. Correlations were performed on data from both groups combined and each group separately using Pearson's product moment correlation coefficient. For the mime ability ratings, the mean between-raters concordance score was calculated according to the method suggested by Cicchetti (1976): agreement = 1, disagreement by one = 0.5, by two = 0.

Results

Details of the subjects and the psychophysiological data are given in White *et al* (1987). Both group mean simple reaction time at the start of the session and the set index value were greater in the schizophrenic group than in the control group (Table I). The correlation between mean initial simple reaction time and set index was 0.82 ($n=12$, d.f. $=10$, $P<0.01$) in the control group and 0.73 ($n=14$, d.f. $=12$, $P<0.01$) in the schizophrenic group, suggesting considerable consistency in individual levels of performance within groups.

TABLE I

Mean reaction time latency and set index scores in control and schizophrenic groups

	Control group mean	Control group s.e.m.	Schizophrenic group mean	Schizophrenic group s.e.m.	F	P
Reaction time:						
ms	237.9	17.0	446.6	93.9	4.43	0.04
Sex index	482.4	45.6	1082.5	282.7	5.22	0.03

Many participants found the TFT procedure more difficult to perform than CFF estimation, and this difficulty was reflected in the fact that six subjects in the schizophrenic group were unable to complete the two-flash testing, reducing the schizophrenic group size to eight. The group mean critical flicker and two-flash threshold intervals (CFI, TFTI, 1000/Hz) are shown in Table II. The between-group mean CFF levels were not significantly different for the staircase procedure, but for the successive approximation procedure the CFI in the control group was significantly shorter, reflecting improved discrimination. None of the group mean levels in TFTI was significantly different from others (Table II).

Within-group correlation coefficients for results of the staircase and successive approximation procedures for CFI and TFTI were 0.94 and 0.89 respectively in the control group ($n=12$) and 0.60 ($n=14$) and 0.74 ($n=8$ for TFTI) for the schizophrenic group. Within-group correlations between mean CFI and TFTI scores derived from the two staircase and successive approximation procedures were 0.27 in the control group ($n=12$) and 0.54 in the schizophrenic group ($n=8$).

Mean error rates in the control and schizophrenic groups on the sustained performance test were 0.5 ± 0.28 ($n=12$) and 8.1 ± 2.23 ($n=14$), but the data in the schizophrenic group were bimodally distributed. The schizophrenic group was divided on the basis of error scores (less or more than five errors respectively) on the sustained performance task. The mean reaction time of the two groups was 462.1 ± 195.1 ($n=8$) for the group with few errors and 433.6 ± 73.9 ($n=6$) for the group with many errors. Respective mime scores were 12.1 ± 2.0 and 12.5 ± 1.15.

For the mime ratings, the mean concordance score was 0.81 ± 0.04. The mean mime ability scores in the two groups were similar (Table III), but the amount of motor activity during miming in the schizophrenic group was less than that of controls.

The mean Present State Examination (PSE) score for psychotic and neurotic symptoms in the schizophrenic group was 10.1 ± 4.1, with a mean psychotic symptom score of 4.9 ± 1.9. The mean symptom score on the Krawiecka scale was 3.57 ± 0.9. The scores on Venables's ward activity scale for the ten questions were totalled for each subject for further use (positive score represents more activity and sociability).

In order to test the hypothesis that mime score and reaction time reflected a similar aspect of psychological function, their correlation coefficient was calculated in both the combined group and each group separately using mean reaction time during the set index procedure (80 trials). In the combined group data, a correlation coefficient significantly different from zero was found for reaction time during the set index procedure with mime score (-0.43, d.f. $=24$, $P<0.05$), while in the control and schizophrenic groups the respective figures (Table IV) were -0.53 (d.f. $=10$, $P<0.1$) and -0.44 (NS). Assessment of scatter plots of the combined data and of values for each group and recalculation after elimination of outliers showed clearly that the interaction was consistent across and within the groups.

TABLE II

Mean critical flicker interval and two-flash threshold interval in control and schizophrenic groups by staircase and successive approximation procedures

	Control group			Schizophrenic group			
	n	mean	s.e.m.	n	mean	s.e.m.	
CFI: ms							
Staircase	12	32.0	2.13	14	31.4	2.35	NS
Successive approx.	12	26.7[1]	1.44	14	30.7	1.97	NS
TFTI: ms							
Staircase	12	39.5	3.5	8[2]	44.1	7.9	NS
Successive approx.	12	39.2	5.6	8	31.3	13.0	NS

1. Mean CFI in control group using the successive approximation procedure significantly shorter than that using the staircase procedure in the control group: F (1, 11) $=20.11$, $P=0.001$.
2. Six members of schizophrenic group failed to complete the TFT procedure.

TABLE III
Group mean scores on rated aspects of miming

	Control group *n = 12*		*Schizophrenic group* *n = 14*			
	mean	*s.e.m.*	*mean*	*s.e.m.*	*F*	*P*
Mime ability	14.0	0.67	12.3	0.99	1.91	0.17
Difficulty	1.29	0.30	1.07	0.27	0.50	0.82
Movement	18.42	1.22	11.29	1.10	18.97	0.00
Affect	4.58	0.61	4.43	0.51	0.39	0.84

Further evaluation of possible associations with this reaction time/mime interaction was possible in the schizophrenic group using the ward activity scale scores which correlated 0.53 (d.f. = 9, $P<0.1$) with mime ability, −0.66 (d.f. = 9, $P<0.05$) with initial reaction time and −0.50 (d.f. = 9, NS) with mean reaction time during the set index procedure.

Since the major difference between the groups in the physiological data (White *et al*, 1987) was of increased skin conductance (SC) activity in the right hand under socially active conditions, we hypothesised that high right-hand SC level would interact negatively with performance measures. The correlation between grand mean SC level in the right hand and reaction time in the combined group data of 0.37 (d.f. = 24) was significant at the 0.05 level. This was reflected in a similar although non-significant correlation in the schizophrenic group (Table IV). Appraisal of the scatter plot for electrodermal right shift between sections 3 and 13 with reaction time in the schizophrenic group suggested that the correlation coefficient (0.40, NS) underestimated the strength of interaction. Electrodermal right shift between the start and end of the social sequence (White *et al*, 1987) correlated more strongly with the reaction time result at the start of the session ($r = 0.58$, d.f. = 12, $P<0.05$). The mean right-hand SC level in subjects in the schizophrenic group with reaction time latencies during the set index procedure above or below 450 ms ($n = 7$ in each group) were 6.23 ± 1.07 and 3.94 ± 0.64 respectively ($F[1, 12] = 3.31$, $P<0.1$, with only one subject overlapping from each group).

An unpredicted difference between the groups was that SC variability on the left in the schizophrenic group correlated positively with mime ability and negatively with reaction time while the reverse relationship was present in the control group (Table IV).

The relationships between CFF and performance measures were contrary to predictions, since longer CFI (lower frequency) correlated with better mime and reaction time scores in the control group and better mime scores in the schizophrenic group (Table IV). In the control group, heart rate correlated positively with mime ability and showed a trend to negative correlation with reaction time. No such interaction was seen in the schizophrenic group.

TABLE IV
Correlation matrices for control and schizophrenic groups

	Age	*RT*	*mime*	*CFI*	*SCL*	*SCR*	*CVL*	*CVR*	*LS*	*HR*
Control group, n = 12										
Age	1.00	0.32	−0.12	0.12	0.55*	0.35	0.06	0.06	0.10	−0.14
RT		1.00	−0.53*	−0.38	0.27	0.07	0.45†	0.50	0.02	−0.46
Mime			1.00	0.58**	−0.10	−0.27	−0.02	0.17	−0.17	0.65**
CFI				1.00	0.18	0.01	−0.03	0.07	−0.44	0.50*
SCL					1.00	0.65**	−0.28	−0.18	−0.42	0.04
SCR						1.00	−0.26	−0.46	−0.44	−0.31
CVL							1.00	0.82**	0.05	−0.37
CVR								1.00	0.06	−0.05
LS									1.00	−0.44
HR										1.00
Schizophrenic group, n = 14										
Age	1.00	−0.07	−0.12	−0.08	0.56**	0.50*	−0.54**	−0.26	0.22	−0.21
RT		1.00	−0.44	−0.13	0.14	0.35	−0.48*	−0.25	0.40	−0.29
Mime			1.00	0.48*	−0.26	−0.29	0.65**	0.25	0.12	0.08
CFI				1.00	−0.12	−0.10	0.31	0.13	−0.11	0.00
SCL					1.00	0.65**	−0.42	−0.18	0.26	−0.52*
SCR						1.00	−0.41	−0.28	0.68**	−0.15
CVL							1.00	0.69**	−0.03	0.07
CVR								1.00	0.15	−0.24
LS									1.00	−0.04
HR										1.00

Significantly different from zero: *$P<0.1$, **$P<0.05$. Significant difference between groups, $^{\dagger}P<0.05$. For each group, d.f. = n-2. Abbreviations: Age: age in years, RT: mean reaction time during set index procedure, Mime: mime ability score, CFI: critical flicker interval, SCL: skin conductance level on left, SCR: skin conductance level on right, CVL: coefficient of variation of skin conductance on left, CVR: coefficient of variation of skin conductance on right, LS: change in mean difference in skin conductance level between sides from start to end of social sequence, HR: heart rate. The physiological variables other than LS are the grand means for each individual across 13 sections.

Discussion

Reaction time and set index scores differentiated reliably between the groups, and the stability of reaction time scores through the session suggested that they were reliable characteristics of the individuals. These results are therefore consistent with previous work where reaction time delay has proved one of the very few reliable associates of schizophrenic disorder (Yates, 1973; Nuechterlein, 1977; Steffy, 1977). Prolongation of reaction time has been found to correlate with schizophrenic disorganisation (Rosenthal *et al*, 1960). It should be noted that the initial reaction time procedure was very brief (16 trials), while the set index procedure required sustained attention over 10-min periods (80 trials) and effectively acted as a sustained performance task.

As the present results for TFT appeared to be less reliable than those for CFF, the CFF results were employed in further analyses. The low correlation of CFF and TFT in the control group suggested either that they are not similar measures or that factors other than flash discrimination sensitivity *per se* played a role in the determination of results. The most obvious reason for impaired performance in the TFT procedure is that, even when a warning signal is given, the subject has to attend very carefully to see the two flashes, whereas during CFF estimations the subject is able to contemplate the stimulus at length before making a decision. This difficulty may have been enhanced by the present procedure since flash durations were considerably shorter than those used elsewhere (e.g. Gruzelier & Venables, 1974).

A high proportion of the schizophrenic group commited many errors on the sustained performance task. We hypothesised that since the ISI was 1.1 s, this was primarily due to slow reaction time. However, mean set index reaction time was not slowed in the group making numerous errors compared with that making few errors. It is possible that performance was affected by factors other than reaction time or sustained attention, particularly since relatively little time was given to training.

Mime assessment was employed because we hypothesised both that social abilities *per se* may be deficient in people with schizophrenic disorder (Farley, 1976) and that abnormal arousal function was a determinant of social dysfunction. It was necessary to assess directly abilities that might form part of social behaviour while subjects were under stress. We know of no other reported means of doing this. The present mime assessment procedure with relatively small numbers of subjects appeared to yield good levels of inter-rater agreement with little formal training. The lack of group mean difference in mime scores was unexpected, since we had predicted that people with chronic schizophrenic disorder would find this section difficult.

In assessing the significance of correlations between variables, the relatively small numbers of subjects compared to the number of variables prevents conclusions from being more than tentative. However, the data do suggest hypotheses which merit further evaluation.

In contrast to the apparent absence of correlation between the sustained performance task and reaction time measures, mime scores correlated with fast reaction time in both groups. Furthermore, Venables's ward activity scale scores correlated positively with mime scores and negatively with reaction time in the schizophrenic group. Correlation between reaction time latency and ability to mime in the human population appears not to have been reported previously and raises questions concerning the importance of the attributes of the nervous system which underlie reaction time performance in social behaviour. The positive interaction with the ward scale scores for the schizophrenic subjects suggests that a unitary dimension of behavioural arousal may be involved.

With regard to interactions between performance and physiological dysfunction, the elevated SC level in the right hand reported in White *et al* (1987) correlated with longer reaction time latency in the schizophrenic group, supporting the hypothesis suggested by the results of Gruzelier & Manchanda (1982) that elevated right-hand electrodermal activity is associated with slowing.

An unanticipated observation was that the interaction between SC variability on the left side and performance was significantly different between the groups. In the schizophrenic group, better performance was associated with increased SC variability on the left side. However, increased SC variability on the left and high SC level on the right did not generally occur in the same individuals in the schizophrenic group, as indicated by the lack of correlation between lateral shift or right-hand SC level and left-hand SC variability, suggesting that separate effects, possibly lateralised, may be present for SC level and variability.

In the control group, mime score and reaction time performance appeared to correlate with fast heart rate and low CFF which may imply that better performance is associated with high peripheral autonomic activity and low cerebral arousal. A similar effect for heart rate was not seen in the schizophrenic group, but a trend to correlation of mime score with low CFF was evident.

A simple interpretation of the data indicating impaired reaction time in schizophrenic subjects with high SC level in the right hand is that of underlying dysfunction in the left cerebral hemisphere in at least a subgroup of people with schizophrenic syndromes (Gruzelier & Flor-Henry, 1979). However, further information is required before interpretations of lateralised indices of arousal and schizophrenic disorder can be made with any confidence, in view of both the lack of detailed information concerning the localisation of control of electrodermal level or variability (White *et al*, 1987) and the indications here and elsewhere (e.g. Crider & Lunn, 1971; Depue & Fowles, 1973) that at least two aspects of electrodermal activity (level and variability) may differ in their significance for psychophysiological function.

Our results suggest that a social stress procedure such as the present one may provide a useful means of evaluating determinants of social dysfunction when suitable physiological and performance measures can also be assessed within a short time. Lateralised SC variables may relate to altered psychological performance in people with schizophrenic disorder in ways which are relatively complex but still measurable.

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References

Cicchetti, D. V. (1976) Assessing inter-rater reliability for rating scales: resolving some basic issues. *British Journal of Psychiatry*, **129**, 452–456.

Ciompi, L. (1980) The natural history of schizophrenia in the long term. *British Journal of Psychiatry*, **136**, 413–420.

Crider, A. & Lunn, R. (1971) Electrodermal lability as a personality dimension. *Journal of Experimental Research in Personality*, **5**, 145–150.

Depue, R. A. & Fowles, D. C. (1973) Electrodermal activity as an index of arousal in schizophrenics. *Psychological Bulletin*, **79**, 233–238.

Farley, J. D. (1976) Phylogenetic adaptations and the genetics of psychosis. *Acta Psychiatrica Scandinavica*, **53**, 173–192.

Gruzelier, J. H. & Venables, P. H. (1974) Two-flash threshold, sensitivity and beta in normal subjects and schizophrenics. *Quarterly Journal of Experimental Psychology*, **26**, 594–684.

—— & Flor-Henry, P. (1979) *Hemisphere Asymmetries of Function in Psychopathology*. Amsterdam: Elsevier.

—— & Manchanda, R. (1982) The syndrome of schizophrenia: relations between electrodermal response, lateral asymmetries and clinical ratings. *British Journal of Psychiatry*, **141**, 488–495.

Hawk, A. P., Carpenter, W. T. & Strauss, J. S. (1975) Diagnostic criteria and 5-year outcome in schizophrenia. *Archives of General Psychiatry*, **32**, 343–347.

Johnstone, E. C., Frith, C. D., Gold, A. & Stevens, A. (1979) The outcome of severe acute schizophrenic illness after one year. *British Journal of Psychiatry*, **134**, 28–33.

Kendell, R. E., Brockington, I. F. & Leff, J. P. (1979) Prognostic implications of six alternative definitions of schizophrenia. *Archives of General Psychiatry*, **36**, 25–31.

Kokes, R. F., Strauss, J. S. & Klorman, R. (1977) Premorbid adjustment in schizophrenia. *Schizophrenia Bulletin*, **3**, 99–106.

Kornetsky, C. (1972) The use of a simple test of attention as a measure of drug effects in schizophrenic patients. *Psychopharmacologia*, **24**, 99–106.

Krawiecka, M., Goldberg, D. & Vaughan, M. (1977) A standardised psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

Nuechterlein, K. H. (1977) Reaction time and attention in schizophrenia: A critical evaluation of the data and theories. *Schizophrenia Bulletin*, **3**, 373–428.

Rodnick, E. & Shakow, D. (1940) Set in the schizophrenic as measured by a composite reaction time index. *American Journal of Psychiatry*, **97**, 214–225.

Rosenthal, D., Lawlor, W. G., Zahn, T. P. & Shakow, D. (1960) The relationship of some aspects of mental set to degree of schizophrenic disorganisation. *Journal of Personality*, **28**, 26–34.

Shakow, D. (1962) Segmental set: a theory of the formal psychological deficit in schizophrenia. *Archives of General Psychiatry*, **6**, 1–17.

Steffy, R. A. (1977) Issues in the study of schizophrenic reaction time: a review of the K. H. Nuechterlein paper. *Schizophrenia Bulletin*, **3**, 445–451.

Stephens, J. H. (1978) Long-term prognosis and follow-up in schizophrenia. *Schizophrenia Bulletin*, **4**, 25–47.

Strauss, J. S. & Carpenter, W. T. (1977) Prediction of outcome in schizophrenia. *Archives of General Psychiatry*, **34**, 159–163.

Taylor, M. M. & Creelman, C. D. (1967) PEST: efficient estimates of probability functions. *Journal of the Acoustical Society of America*, **41**, 782–787.

Vaillant, G. E. (1978) A 10-year follow-up of remitting schizophrenics. *Schizophrenia Bulletin*, **4**, 78–85.

Venables, P. H. (1957) A short rating scale for "activity-withdrawal" in schizophrenics. *Journal of Mental Science*, **103**, 197–199.

White, C. de B., Farley, J. & Charles, I. P. (1987) Chronic schizophrenic disorder. I. Psychophysiological responses, laterality and social stress. *British Journal of Psychiatry*, **150**, 365–374.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

Yates, A. J. (1973) Abnormalities of psychomotor functions. In *Handbook of Abnormal Psychology* (ed. H. J. Eysenck) London: Pitman.

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Influence of Husbands on the Outcome of their Agoraphobic Wives' Therapy

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Of 30 agoraphobic married women undertaking home-based behaviour therapy, 15 each had a female friend as co-therapist and 15 had their own husband as co-therapist. At 12 months follow-up, women spent a mean of approximately 40 minutes a day more outside the home alone than at baseline. Women assisted by female friends made somewhat more behavioural gains than those assisted by husbands. Women in both groups whose husbands had been more depressed 2 months after the beginning of therapy did less well at 6 months. Husbands who become more depressed may retard their wives' progress in therapy, but the effect was small, and was outweighed by other considerations.

Hafner (1977*a*,*b*) claimed that many husbands of agoraphobic women become depressed when their wives' phobic problems improve in therapy. He argued too that a husband who becomes symptomatic can thereby induce his wife to relinquish therapeutic gains, and to relapse back into her agoraphobic condition.

Family therapists (e.g. Minuchin & Fishman, 1981) have proposed that within a family a circular homeostatic pattern may occur in which a set of roles helps to maintain symptoms in one family member, while the symptoms tend to maintain family members in their roles. Applying this to families with agoraphobia, the dependent role of a more fearful partner may be complemented by a role of helpfulness in the more mobile partner, and this complementarity (cf. Bateson, 1935) may help to maintain agoraphobia. Simultaneously, symptoms help maintain a dependency in which both partners have an interest.

According to this argument, attempts to displace the family from its equilibrium by therapy are likely to be resisted by forces which tend to restore homeostasis in the couple.

The therapy used by Hafner (1977*b*) in his study of 30 marriages in which wives were agoraphobic was based on 12 hours of group exposure to feared situations within a 4-day training period, with questionnaire measures taken at baseline and at intervals during the following year. He divided patients into a more hostile and a less hostile set. The more hostile patients did less well. Husbands of this set became more hostile during the follow-up period, and their mean psychiatric symptoms increased, but then decreased again when their wives partially relapsed. In some husbands, depression and jealousy reached alarming levels. Milton & Hafner (1979) studied 18 patients in a similar design. Patients less satisfied with their marriages did less well, and their marital satisfaction decreased.

These findings suggest that families may resist therapeutic change, but the increase in husbands' psychiatric symptoms and the relapses of the patients were small and not statistically significant. Moreover, Hafner's conclusions have been questioned: Cobb *et al* (1984) found no decrease in marital satisfaction during agoraphobic wives' therapy, and were sceptical that spouses influence recovery from agoraphobia.

The home-based therapy of Mathews *et al* (1977, 1981*b*) for agoraphobia enabled a further test to be made of the homeostatic hypothesis of marriages in which the condition occurred. In this procedure the client undertakes outings of graded difficulty, and is in charge of her own therapy. Her husband is recruited as co-therapist, and the professional therapist acts mainly in a consultative role, making a small number of visits.

The present study was an independent trial of the treatment programme of Mathews *et al*, with measures taken at baseline and 2, 6 and 12 months. Either the subject's husband or a woman friend was randomly chosen to act as co-therapist. We tested Hafner's hypothesis of resistance by spouses, and included behavioural measures. We aimed to ascertain also whether the therapy of Mathews *et al* is effective where patients recruit friends rather than family members to assist them.

Hypothesis 1. The principal hypothesis was that there would be a difference in outcome between women with husbands as co-therapists and women with female friends as co-therapists. We could not predict which condition would do better, because Hafner's hypothesis allows two different directions of effect.

In one direction, acting as co-therapist might provide a new role for a husband to replace the one he was losing as his wife's caretaker. This might counter depression associated with loss of his previous role, diminishing any tendency to retard his wife's progress. Hence, women whose husbands were acting as co-therapists might do better.

In the other direction a husband who wished to keep his wife at home would be more able to retard therapy in a co-therapist role. Co-therapists outside the client's marital relationship might enable the client to do better.

Hypothesis 2. In direct replication of Hafner's 1977*b* report, as wives become more independent, husbands might experience change in their marital equilibrium and suffer symptoms. As Hafner implied and as would be expected from a major role loss (Oatley & Bolton, 1985), these symptoms would be mainly depressive. Hence husbands' depression scores were predicted to rise at 2 months, and perhaps fall again later. Moreover, if acting as co-therapist did provide husbands with a new role, then those who were co-therapists would become less depressed than those who were not co-therapists.

Hypothesis 3. Clients whose husbands became depressed at 2 months would be expected to go out less by 6 months. There would be a negative correlation between husbands' depression scores at 2 months and their wives' behavioural improvement at 6 months.

Hypothesis 4. According to the homeostatic theory, marriages in which wives felt controlled by their husbands might be more resistant to change. Hence, there would be a larger negative correlation between husbands' early depression and wives later behavioural scores than in marriages in which wives felt less controlled.

Hypothesis 5. We derived an index of hostility to try and replicate Hafner's 1977*b* finding that more hostile clients made poorer progress than those who were less hostile.

Method

Subjects

Subjects were drawn from 59 women in Brighton, Eastbourne and North London who volunteered for the study: 24 were contacted via self-help organisations for phobics, 31 got in touch with us after articles in the local press, and four were referred by GPs.

Criteria for acceptance were that the subjects were aged 18–70 years; had been married or in stable heterosexual cohabitation for at least 3 years; had been agoraphobic for at least 3 years; and had both a husband (or male partner) and a woman friend prepared to help. They had to be literate (in order to fill in forms and read the therapy booklets), be free of physical disability, and not be receiving treatment for agoraphobia other than tranquillisers or anti-depressants prescribed by their GP.

Agoraphobia was defined using the criteria of DSM-III (American Psychiatric Association, 1980) of (a) fear and avoidance of being alone or in public places, or of places where escape is difficult because of sudden incapacitation; (b) severely constricted daily activities because of inability to leave home alone, or to enter public places; and (c) condition not due to an obvious primary major depressive episode, obsessional–compulsive disorder, paranoid disorder or schizophrenia. In addition we administered the Leeds Self Assessment of Anxiety Specific Scale, range 0–18 (Snaith *et al*, 1976). This has a case threshold between 6 and 7. All but one of the 30 subjects who undertook the trial were above case threshold on this scale, with 26 of them having scores of 12 or above.

We explained the programme to the 39 women who met the criteria. We said we would decide randomly whether their husband or their woman friend would be co-therapist for them, but that to take part they had to make sure that both were willing to help. Six further women decided against going ahead after learning more about the programme or because their husbands refused to be available as co-therapists. This left 33 cases. They were assigned randomly, two to each condition in blocks of four, either to a condition in which their female friend would be their co-therapist (FF group), or to one in which their husband would be their co-therapist (HB group).

TABLE I
Patient characteristics in each treatment group

	FF group (*n* = *15*)	*HB group* (*n* = *15*)
Mean age (years)	45	42
Mean duration of agoraphobia (years)	18	14
Proportion on tranquillisers (%)	60	80
Proportion on anti-depressants (%)	7	7
Proportion on both types of drug (%)	20	7
Proportion who had previously received psychological help to overcome agoraphobia (%)	33	60
Social class (Registrar General's classification)		
I & II (%)	53	46
III & IV (%)	46	46
V (%)	0	7

In the FF group, two subjects dropped out early in therapy because going out made them too anxious. Another subject was excluded from the HB group because we discovered that she had become partially blind as a result of diabetes, and had been getting her husband to fill in the assessment forms for her. This left 15 subjects in each group. We collected complete data up to 6 months. We followed up all 30 couples for 12 months, though at 12 months one FF and one HB couple would complete only the Leeds scales (see below) but no other instruments.

Table I shows the characteristics of the subjects. The severity and duration of symptoms were similar to those of clinical samples (cf. Buglass *et al*, 1977; Mathews *et al*, 1981*a*).

Assessment methods

Three main measures were taken at baseline, at 2 months, at 6 months (i.e. end of therapy), and at 12 months (i.e. 6-month follow-up). A Response to Treatment Questionnaire was taken at 12 months.

Behavioural diaries

Behavioural diaries were modified from those of Mathews *et al* (1977, 1981*a*). After detailed discussion, a patient listed activities which for her were 'safe' (i.e. did not cause anxiety even on a bad day). The patient was then asked to record in the diary everything else she did. She kept the diary every day for 2 months, and then again for 2 weeks at the 6-month and 12-month assessment points.

An Items score was derived from a generalised hierarchy of 15 items which provoke anxiety and avoidance by agoraphobics. At the lower end of this hierarchy, items 1 and 2 are, respectively, the patient spending up to 30 and 60 minutes outside a particular safe room in her house. These were only applicable to a few people. Items 3, 4, 5 and 6 are leaving home alone for at least 5, 15, 30 and 60 minutes, respectively. The next items, also to be accomplished alone, are (7) local shops, (8) main shops, and (9) trips on public transport. Next are items of outings up to 4 hours, which may be accompanied, and in order of increasing social constraint: (10) accompanied outings (e.g. car trips or walks with a spouse), (11) visits (e.g. to friends, or visits of others to the patient's home), (12) visits to pubs, cafes etc., (13) attending public assemblies such as doctor's, parents' meetings at school, cinema etc. Finally there are two items about longer outings; (14) more than 4 hours, and (15) more than 8 hours—away from home on visits of various kinds during which the patient may be accompanied. Details of content analysis and scoring developed for these diaries are given by Oatley and Hodgson (in prep.). Item scores range from 0 to 15.

Diaries at each assessment point (baseline and 2, 6 and 12 months) were analysed to determine how many items on this hierarchy had been accomplished. To score one item the patient had to record its performance twice during a 5-day assessment period that included a weekend.

A subsidiary diary measure was the total amount of time away from home during these 5-day periods. Diary entries are made each day after performance. Hence, these scores may be more valid than retrospective questionnaire measures of phobic symptoms.

Leeds Scales for the Self Assessment of Anxiety and Depression

The Leeds self-report questionnaire (Snaith *et al*, 1976) was completed by all patients and husbands at each assessment point, with no consultation between each other. It has 15 statements, each describing a symptom of anxiety or depression to be rated on a four-point scale and scored 0–3. We used two principal measures as described by Snaith *et al*; (a) for patients the Specific scale of diagnosed anxiety, which we will call Clients' Leeds Anxiety Scores, and (b) for husbands, the General scale of non-diagnosed depression, which we will call Husbands' Depression Scores. Each scale of six items has a range of 0–18.

Dyad repertory grids of relationship

To try and predict in which marriages therapy might go more or less well, we administered a dyad repertory grid (Ryle & Lunghi, 1970), to patients and husbands. Supplied constructs were 'Feels guilty about, Looks after, Exerts control over, Gives in to, Blames, Is irritated by, Depends on', to be rated on seven-point scales for the marital relationship and relationships with parents. Patients and husbands were asked to complete grids without consultation, though we said they could discuss them afterwards if they wished.

Two criteria derived from baseline grids were used to predict who would be most resistant to therapy. One was of patients who felt controlled by their husbands. The criterion of high control by husband was a score of 4 or more at baseline on the construct 'Exerts control over' as applied to the husband. Over all 30 subjects, irrespective of co-therapist, this criterion gave a split of 16 wives who felt control was high and 14 who felt control was low.

The other criterion, to parallel Hafner's index of hostility, selected patients scoring 5 or more at baseline on either or both of the constructs 'Blames' and 'Is irritated by' applied to their husbands. This gave 14 high hostility patients and 16 low hostility patients, irrespective of co-therapist.

Principal components analysis of the whole grids will be reported elsewhere.

Response to Treatment Questionnaires

Response to Treatment Questionnaires were distributed at the 12-month follow-up meeting. Patients were asked to complete them not while the therapist was present but later, and return them by post. Ratings on five-point scales were made on the following questions: overall satisfaction with the programme, change in being able to go out alone, change in being able to go out accompanied, change in depression, change in anxiety, change in optimism about being able to overcome agoraphobia. The five-point scales were of the form (1) very dissatisfied, very much less able to go out, etc., (2) dissatisfied etc., (3) no change, (4) satisfied, etc., (5) very satisfied, etc. Clients were also asked for details of any other therapy or self-help they had undertaken during the 12 months.

To reduce bias we did not check any Leeds or grid measure until after the 12 month interviews.

Therapy programme

Therapy was based on the home-based treatment of Mathews *et al* (1977, 1981*a*). Instructions to client and co-therapist were provided in booklets (Mathews *et al*, 1981*b*), supplemented by our written introduction and by the relaxation audiocassette of Sharpe (1976).

The authors were the therapists. After working under supervision with four pilot cases using this therapy, one of us (KO), with 8 years' experience of conducting groups and 3 years' of giving individual interpretive and cognitive therapy, saw eight clients, and the other (DH), with 6 years' experience working behaviourally with self-help groups, saw 22.

Before the first meeting we had decided by telephone or letter if acceptance criteria were met. The random choice of husband or woman friend as co-therapist was then made.

At the first visit, lasting 1.25 hours, the therapist saw the patient with her husband, irrespective of whether she had been assigned to the FF or HB group. We told the couple whether husband or woman friend would be acting as co-therapist. We described the programme and the role of the co-therapist, and explained that as well as giving the therapy we were doing research to see how husbands or friends might help in therapy and to see what feelings husbands and wives had in the course of therapy. A history and demographic information were collected.

Husband and wife were asked to complete the Leeds scales and the repertory grid forms. The patient was helped to prepare a safe list for her diary which was left so that she could make daily entries for a baseline week. We left the treatment booklets and the relaxation tape. We also left two copies of a contract recording agreement to the therapy, signed by the therapist.

We discussed the implications of therapy for the couple's relationship, and how it might mean substantial changes for both of them. We said that they should only go ahead after they had both read both booklets and discussed the programme together. If they decided to go ahead then the wife and the co-therapist should sign the contract form.

The client was asked to practice relaxation twice a day using the tape, but to make no other changes in her life until the next meeting, a week later. To acquire a baseline measure of activity, she was asked to record in her diary her performance of all activities not on her safe list.

Subsequent visits lasted an hour or less, and were at 1 week and 1, 2, 6 and 12 months, with phone calls at 2 weeks and in order to make appointments for visits.

Up to 6 months the co-therapists were asked specifically to attend meetings, which were largely concerned with describing the therapy as specified by Mathews *et al*, discussing progress, and advising patient and co-therapist. Some patients who had a woman friend as co-therapist had their husband also present. At 2, 6 and 12 months we also collected the measures, which were typically sent by post before visits.

At 6 months we discussed the long-term plans of the patient, and enquired about marital difficulties if this had not been raised spontaneously. We discussed further help where such difficulties had occurred.

At the 12-month follow-up visit we saw the patients and their husbands for further discussion and closure.

Two patients, one each in the FF and HB group, started further therapy near the 6-month point. At 12 months we referred two other couples, one in each of the FF and HB groups, for marital therapy.

Results

As may be seen from Figs 1 and 2, both FF and HB groups of patients improved on mean behavioural Items and Leeds anxiety scores by 6 months, and the improvement was sustained at 12 months. Separate analyses of variance were performed on the scores for each measure. In the Items analysis the within-subjects improvement over time was significant ($F=15.85$; d.f. 3, 78; $P<0.001$), although neither the difference between the FF and HB groups, nor the group × time interaction were significant. (P-values of 5% or less will be regarded as significant in this paper.) Improvement in Leeds anxiety scores over time was significant ($F=13.75$; d.f. 3, 84; $P<0.01$), although again neither the between-group effect nor the group × time interaction were significant.

On both measures most of the improvement was accomplished in the first 2 months, and these gains were sustained thereafter. This was confirmed by significant *a posteriori* *t*-test between baseline scores and subsequent

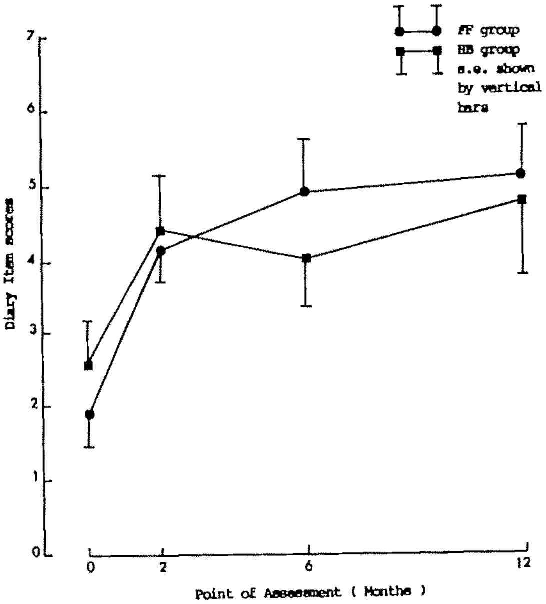

FIG. 1. Means and standard errors of Items scored on a generalised hierarchy, as assessed from behavioural diaries, by 15 patients with female friend (FF) co-therapists, and 15 with husband (HB) co-therapists, at baseline (0), after 2 and 6 months of therapy, and at a 12-month follow-up.

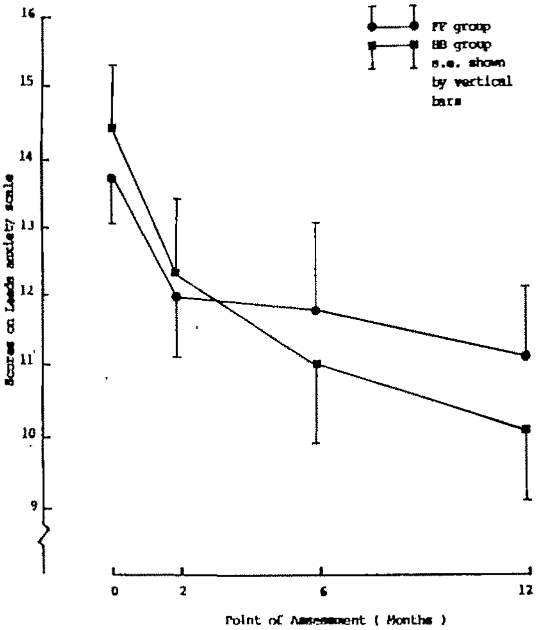

FIG. 2. Means and standard errors of Leeds anxiety scores by 15 patients with female friend (FF) co-therapists, and 15 with husband (HB) co-therapists, at baseline (0), after 2 and 6 months of therapy, and at a 12-month follow-up.

assessments, but no significant differences between assessments at 2, 6 and 12 months.

To derive scores of items gained since baseline, the items at baseline were subtracted from items at 2, 6 and 12 months to indicate new items attained. At 6 months the FF group had gained three items as against 1.6 items in the HB group ($t = 2.1$, d.f. 28, $P < 0.05$, two-tailed). By 12 months, the difference (3.2 items gained by the FF group compared with 2.3 items gained by the HB group) was no longer significant.

The prediction of Hypothesis 2 was that husbands would be more symptomatic at 2 months. Leeds depression and anxiety scores of husbands stayed low and approximately constant in both FF and HB groups, with mean levels staying between 2 and 5 on both scales in both groups. Analysis of variance on these scores showed no significant effects of time, of FF versus HB condition, or of the interaction, in either depression or anxiety scores. Nor were there any significant *a posteriori* differences between specific means.

According to Hypothesis 3, husbands' depression early in therapy should correlate negatively with wives' gains at 6 months. First, taking husbands' depression at 2 months and wives' Items Gained at 6 months, the product-moment correlation over all subjects was $r = -0.36$ (d.f. 28, $P < 0.05$, one-tailed). In the FF and HB groups, r was -0.52 and -0.32 respectively. Adding husbands' depression and anxiety scores to produce total symptom scores made no substantial difference: the overall correlation rose to $r = -0.39$. Correlations between husbands' depression at 2 months and wives' raw Item scores at 6 months were slightly lower, $r = -0.31$ ($P < 0.05$). At baseline the association between husbands' depression and wives' Item scores was $r = 0.09$ over all subjects. Husbands' baseline depression correlated with wives' 6-month Items Gained at $r = -0.14$ (NS). These subsidiary correlations indicate that it was mainly the change in some husbands' depression in the first 2 months, rather than any association at baseline, that predicted their wives' progress.

According to Hypothesis 4, patients who saw their husbands as more controlling would be more affected by their husbands' depression. For the high perceived control set ($n = 16$), correlations between husbands' depression at 2 months and wives' Items Gained at 6 months was $r = -0.42$ (d.f. 14, P approx. 0.05). For the low perceived control set the correlation was $r = -0.20$ (d.f. 12, NS).

Hypothesis 5 predicted replication of Hafner's 1977*b* finding that the more hostile clients would do worse in therapy. The mean of the Items Gained by the more hostile patients ($n = 14$) was 1.75 by 6 months, and by less hostile patients 2.67. Thus, there was a difference but it was not significant ($t = 1.24$, d.f. 28, P approx. 0.1). The mean of the anxiety scores in the more hostile patients at 6 months was 11.36, and for the less hostile patients 11.50 ($t = 0.9$, d.f. 28, NS).

Patients meeting the criterion of perceiving their husbands to be more controlling, and those meeting the criterion of being more hostile were scattered evenly between FF and HB groups.

As may be seen from answers to the Response to Treatment Questionnaire, Table II, most clients were satisfied with the programme, and most rated themselves as improved on the factors asked about.

TABLE II

Positive responses to questions on the post-treatment questionnaire

	Number of positive responses FF group (n = 14)	*HB group (n = 14)*	*Overall percentage of positive responses (n = 28)*
Satisfied and very satisfied with programme	12	12	86
More and much more easy to go out alone	10	11	75
More and much more easy to go out accompanied	11	9	71
Less and much less depressed	6	8	50
Less and much less anxious	10	9	68
More and much more optimistic	11	12	82

Discussion

Overall neither husbands nor women friends were significantly better in assisting the agoraphobic women to go out more. Nor was either kind of co-therapist clearly superior in helping the remission of anxiety symptoms. In this respect our results resemble those of Cobb *et al* (1984) who found that, of women undertaking the treatment of Mathews *et al* (1981*a*,*b*), those whose husbands acted as co-therapists did no better than those without co-therapists. In terms of the amount of time spent out alone, our results are also comparable with those of Mathews *et al* (1977), who state that the mean time that subjects spent out alone approached, but did not reach, an hour a day. For our subjects the mean time spent out alone rose from 13 minutes a day at baseline to 53 minutes a day at 12 months.

There was a non-significant trend towards the FF group doing better on the behavioural diary measure. The FF group was significantly better ($P<0.05$) in Items Gained at 6 months, but by 12 months the difference was no longer significant. This may have been because the HB group had continued to improve while the FF group had not. Taking all the results up to 12 months it seems safest to conclude that the partial relapse of the HB group at 6 months was due to chance in a syndrome which has wide temporal fluctuations (Buglass *et al*, 1977).

On Leeds anxiety scores neither group was superior (Fig. 2), though the HB group improved slightly more.

Overall the somewhat larger behavioural gains of the FF group might imply that husbands had less interest in increasing their wives' mobility than did women friends. We had the impression that some of the women friends were better as co-therapists than most of the husbands. Certainly there was more novelty for women who had a friend helping them. They gained extra support and perhaps more understanding from another woman.

The claim of Hafner (1977*b*) that husbands would become symptomatic was not replicated. On average, husbands did not become significantly more depressed or anxious at any time.

We were careful to avoid provoking husbands' jealousy or feelings of exclusion. We warned each couple beforehand of possible readjustments to their marriage. We did not accept in the study couples in which the husband was not willing to act as co-therapist. Two couples were excluded because of husbands' hostility and refusal at this point. We encouraged couples to discuss changes that occurred between them. Though important therapeutically, these steps probably diminished the effect we were studying, so the study provided a severe test of Hafner's hypothesis.

In designing the study, we planned to test Hypothesis 3 only by exploring correlations between husbands' depressive symptoms at 2 months and wives' Items gained at 6 months, to avoid too many significance tests on limited data. These correlations showed that those husbands who were more depressed at 2 months had wives who had gained fewer new behavioural items by 6 months. Over all 30 couples the correlation ($r=-0.36$) accounts for only 13% of the variance of Items Gained, and even the larger correlation ($r=-0.52$) in the FF group accounts for only 27% of the variance.

Perceived control and hostility which were predicted to increase the effect (Hypotheses 4 and 5) had small influences.

Therefore, we concluded that there was an effect of the kind that Hafner described, but that its size was modest. In this study, it principally took the form of husbands being slightly less effective as co-therapists than women from outside the family; and of wives whose husbands became more depressed in the first two months of therapy going out less than those whose husbands became more cheerful. No husbands became suicidal and no marital separations occurred during the year of the study.

Becoming depressed is only one possible type of influence. Several other kinds of pressure apart from onsets of symptoms did occur. For instance, one woman started a part-time job, which she liked, but her husband made her give it up. Another husband, perhaps hoping to enhance her enjoyment of being at home, had an elaborate new kitchen built for his wife early in her therapy. In another couple, the husband decided that therapy was making his wife worse despite her going out more and sent her to another clinician who gave her a course of flooding, following which she relapsed below her baseline scores. Clinical intuition about family homeostatic processes may, therefore, often be correct. The difficulty is in casting it into an empirical form. Though this trial specifically estimates the extent of resistance associated with depressive symptoms in the non-treated partner, a composite index is needed. Measures of hostility, of husbands' symptoms, or of any other single parameter may each be relevant in only a proportion of marriages.

We found nothing to negate the principle of including spouses in therapy for agoraphobia. Indirectly our results imply that including the spouse is important, as stressed by Webster (1953). For many couples change does require readjustment in the relationship. But the small negative effects for husbands are outweighed by gains in mobility for the patients.

The results also indicate something of the nature of agoraphobia as a chronic anxiety state. Most subjects went out more. Some began to use public transport. Many went on long visits, and seven (23%) started paid jobs. Most were pleased with their progress (Table II) and anxiety levels decreased. But despite this they remained anxious people (Fig. 2). At 12 months, 27 were above the case level of Snaith *et al* (1976) on the Leeds anxiety scale (between 6 and 7) as compared with 29 at baseline.

The principal effect of this behavioural therapy was that more of the environment was made relatively safe. This kind of therapy leaves largely unsolved the problems of allowing comfortable levels of anxiety to be attained.

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References

Ackerman, N. W. (1958) *The Psychodynamics of Family Life*. New York: Macmillan.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* 3rd edn. Washington, DC: American Psychiatric Association.

Bateson, G. (1935) Culture, contact and schismogenesis. *Man*, **35**, 178–183.

Buglass, D., Clarke, J., Henderson, A. S., Kreitman, N. & Presley, A. S. (1977) A study of agoraphobic housewives. *Psychological Medicine*, **7**, 73–86.

Cobb, J. P., Mathews, A. M., Childs-Clarke, A. & Blowers, C. M. (1984) The spouse as co-therapist in the treatment of agoraphobia. *British Journal of Psychiatry*, **144**, 282–287.

Hafner, R. J. (1977*a*) The husbands of agoraphobic women: assortative mating or pathogenic interaction? *British Journal of Psychiatry*, **130**, 233–239.

—— (1977*b*) The husbands of agoraphobic women and their influence on treatment outcome. *British Journal of Psychiatry*, **131**, 289–294.

Hodgson, D. L. (1985) *Self-help in the Treatment of Agoraphobia*. DPhil. thesis, University of Sussex.

Mathews. A. M., Teasdale, J., Munby, M., Johnson, D. & Shaw, P. (1977) A home-based treatment program for agoraphobia. *Behavior Therapy*, **8**, 915–924.

——, Gelder, M. G. & Johnson, D. W. (1981*a*) *Agoraphobia: Nature and Treatment*. London: Tavistock.

——, —— & —— (1981*b*) *Programmed Practice for Agoraphobia: Vol. 1 — Clients' Manual; Vol. 2 — Partners' Manual*. London: Tavistock.

Milton, F. & Hafner, R. J. (1979) The outcome of behavior therapy for agoraphobia in relation to marital adjustment. *Archives of General Psychiatry*, **36**, 807–811.

Minuchin, S. & Fishman, H. C. (1981) *Family Therapy Techniques*. Cambridge, Mass: Harvard University Press.

Oatley, K. & Bolton, W. (1985) A social-cognitive theory of depression in reaction to life events. *Psychological Review*, **92**, 372–388.

Oatley & Hodgson, D. L. Analysis and scoring of behavioural diaries kept by agoraphobics. (In prep.)

Ryle, A. & Lunghi, M. (1970) The dyad grid: a modification of repertory grid technique. *British Journal of Psychiatry*, **117**, 323–327.

Sharpe, R. (1976) *A Complete Course in Relaxation*. Audiocassette. London: Stresswatch.

Snaith R. P., Bridge, G. W. K. & Hamilton, M. (1976) The Leeds scales for the self-assessment of anxiety and depression. *British Journal of Psychiatry*, **128**, 156–165.

Webster, B. (1953) The development of phobias in married women. *Psychological Monographs*, **67**, (367), 1–18.

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Point of View

The Concepts of Mild Depression

R. P. SNAITH

The term 'depression' is now used to cover a wider range of emotional disturbances than was the case when psychiatrists confined their attention to patients referred to psychiatric hospitals. There is considerable confusion about the variety of meanings and the loose terminology leads to claims and counter-claims about aetiology and treatment. The current classificatory systems and the variety of rating scales do not clarify issues concerning appropriate management. The guidelines for a clearer definition of a biogenic form of the concept of depression, which is likely to respond to antidepressant drug therapy, would be of considerable value to psychiatrists, non-psychiatric physicians and others who try to treat or help people in states of disordered emotion. Such guidelines are proposed.

Depression is one of the great themes of psychiatry; in addition to psychiatrists, a considerable number of people attempt to treat, counsel or otherwise help other people who are said to be suffering from depression; yet the concept of the state is obscure. There is little confusion about the recognition of a severe state, sometimes called a psychosis, but *milder* degrees of the condition lack definition. Sometimes the term depressive neurosis is used but Klerman *et al* (1979) have pointed out that a variety of concepts are associated with this term: stress precipitation, maladaptive personality patterns, unconscious conflicts, 'non-psychotic' symptoms; hence the term is so vague as to be useless if not frankly misleading. Kendell (1969, 1976) has argued, from a number of sources of evidence that the only reliable distinguishing feature between depressive psychosis and depressive neurosis is that of the severity of the disorder.

Some historical considerations

The term 'depression' was occasionally used by the early students of mental disorder, and by the end of the nineteenth century attempts were made by some psychiatrists to define states of depression; for instance one early British teacher (Bevan Lewis, 1899) wrote: "In simple pathological depression . . . the patient exhibits a growing indifference to his former pursuits and pleasures: the ordinary duties of life and business become irksome and devoid of interest: especially do all forms of mental exertion cause ennui and disaster . . . and the environment fails to call up pleasurable associations—a dreariness and gloom pervade the outside world, since it is interpreted in terms of the predominant feeling . . .". However, so far as institutional psychiatry was concerned the concept of 'Melancholia' was dominant and the history of this has been recorded by Lewis (1934) who recognised the term as: "Suffering many mutations, at one time the tenacious guardian of outworn schemes or errant theories; presently misused, cavilled at, disposessed, it has endured into our own times, a part of medical terminology no less than of common speech". The word 'depression' was not widely used until the second decade of this century and Alexander Walk (personal communication) wrote: "The word 'depression' only appears once in the Indexes of our Journal before 1909; this is a review of Clouston's Annual Report from Morningside for 1890, where he wrote that states of depression are becoming as common as exaltation as causes of admission to the asylum". ['our Journal' is *The Journal of Mental Science*, forerunner of *The British Journal of Psychiatry.*] It was Adolf Meyer (1905) who started the widespread adoption of the term and, curiously, this was in order to try to eliminate the confusion surrounding the use of the word melancholia: "If, instead of melancholia, we apply the term depression to the whole class, it would designate in an unassuming way exactly what was meant by the common use of the word melancholia and nobody would doubt that, for medical purposes, the term would have to be amplified so as to denote the kind of depression. In the large group of depressions we would naturally distinguish our cases according to etiology, the symptom-complex the course of the disease and the results". Alas, Meyer's hopes have remained unfulfilled in the subsequent eight decades. In fact the confusion worsened and Ascher (1952) commented: "There appears to be a good deal of confusion in the literature, as well as in practice, about depressive illness which is a left-over from the era of descriptive psychiatry. Concepts of psychogenesis, endogeny, reactivity, neurosis, psychosis and others have been introduced to clarify the issue . . . without producing the desired result".

If we now leave the domain of institutional psychiatry and consider the manifold states of human misery which are all sometimes called 'depression', we are in an even more confusing area. Here are the states of grief at loss, frustration of failed aspiration, the gloom of despair, the accidie of disillusion, the demoralisation of long suffering and the cynical outlook of the pessimist. Recently, in the wake of a new psychotherapeutic endeavour, cognitive therapy, there has been an interpretation of 'depression' as a lifelong habit of self-depreciation. Also in this confused field, is to be found the mild state of the biogenic disorder, also called 'depression' but so lacking in definition that it is widely overlooked. This is the state for which Kräupl Taylor (1966) regretted the absence of a term, a 'hypomelancholia' by analogy with hypomania: "Unfortunately this term is not in common use; instead such vague and theoretically misleading expressions as 'mild depressive reaction' or 'neurotic depression' are employed".

Definitions of terms

In the Present State Examination manual Wing and his colleagues (1974) have provided the most succinct definitions of psychopathological terms and, in order to clarify the issues of terminology the definitions of those terms relating to depressive states are set out here.

Depressed Mood
Depressed mood may be expressed in a number of ways—sadness, misery, low spirits, inability to enjoy anything, dejection. . . . The extent to which the subject has cried during the past month is one criterion of severity although it is not sufficient in itself (the deepest depression may be frozen misery which is beyond tears). Another criterion of severity is lack of variability; moderate forms of depression tend to come and go more than severe forms.

The authors state that depressive apathy and other forms of unpleasant affect e.g. nervous tension or anxiety should not be included in the concept of Depressed Mood.

Recent Loss of Interest
There is a definite recent diminution in the subject's interests, either some interests have been dropped, or the intensity of the interest has decreased. Everyone has interests of some sort but the extent of the diminution must be measured in the context of the range and depth of the subject's usual activities. Take into account everyday taking an interest in clothes, food and appearance, keeping up to date with news, etc. . . .

Self-Depreciation
The subject feels inferior to others, even—in the most intensive form of the symptom—worthless. . . . Ideas of self-depreciation are an exaggerated form of self-knowledge about part of the subject's own character, which gives a false picture because it is not balanced by the recognition that most human beings have similar faults. Self-depreciation implies a raising of the standard applied to the subject without an equivalent raising of the standard applied to others, or taking into account balancing merits . . .

Hopelessness
The subject's view of the future is bleak and without comfort. Two degrees of intensity are distinguished by whether the subject has given up attempting to cope, because he can see no point in trying because the future is hopeless. . . .

Observed Depression
Rate a sad, mournful look with tears in the eyes and gloomy monotonous voice. . . .

In addition to these items there are definitions for *Neglect Due to Brooding*, *Subjective Anergia*, *Slowness and Underactivity*, *Inefficient Thinking*, *Poor Concentration*, *Suicidal Plans or Acts*, *Morning Depression*, *Social Withdrawal*, *Guilt* and other related symptoms. The manual must be consulted for full definitions. It is evident that authors of the PSE have provided a valuable service by these definitions but even so it may be noticed that the definition of *Depressed Mood* is less precise than the other symptoms and covers, in fact, a range of concepts.

The nosological systems

Two widely used systems are the International Classification of Diseases, at present in its ninth revision (ICD-9: World Health Organization, 1978) and the third revision of the Diagnostic And Statistical Manual (DSM-III: American Psychiatric Assocation, 1980).

The ICD-9

Preview of the proposals for revision leading to ICD-10 indicate that the term 'depressive neurosis' will be abolished and depressive states will no longer be defined partly in terms of their supposed aetiology. However no substantial revision of the following comments is required). The glossary to the Mental Disorders Section of this system provides a convenient guide (p. 58) to the categories in which depression is a major or significant feature. There are 10 three-digit and 19 four-digit categories. In order to understand the guidance provided for classification the significant parts of the definitions are listed:

295.7 *Schizoaffective Type*
A psychosis in which pronounced manic or depressive features are intermingled with schizophrenic features . . .

296.1 *Manic-Depressive Psychosis—Depressed Type*
An affective psychosis in which there is widespread mood of gloom and wretchedness with some degree of anxiety . . .

298.0 *Other Non-Organic Psychosis—Depressed Type*
A depressive psychosis which can be similar to 296.1 but is apparently provoked by saddening stress such as bereavement or severe disappointment or frustration . . .

300.4 *Neurotic Depression*
A neurotic disorder characterized by disproportionate depression which has usually recognisably ensued on a distressing experience; it does not include among its features delusions or hallucinations and there is *often* preoccupation with psychic trauma which preceded the illness e.g. loss of a cherished person or possession. Anxiety is also *frequently* present; mixed states of anxiety and depression should be included here . . . [My italics]

301.1 *Affective Personality Disorder*
. . . is characterized by lifelong predominance of pronounced mood which may be persistently depressive . . .

308.0 *Acute Reaction to Stress Predominant Disturbance of Emotions*
Panic states, excitability, fear, depression and anxiety . . .

309.0 *Adjustment Reaction: Brief Depressive Reaction*
States of depression, not specifiable as manic-depressive, psychotic or neurotic, generally transient, in which depressive symptoms are usually closely related in time and content to some stressful event.

309.1 *Adjustment Reaction: Prolonged Depressive Reaction*
States of depression, not specifiable as manic-depressive . . . generally long lasting; usually developing in association with prolonged exposure to a stressful situation.

311 *Depressive Disorder Not Elsewhere Classified*
States of depression, usually of moderate but occasionally of marked intensity, which have no specifically manic-depressive or other psychotic depressive features and which do not appear to be associated with stressful events or other features specified under neurotic depression".

313.1 *Disturbance of Emotions Specific to Childhood And Adolescence; With Misery And Unhappiness*
. . . the main symptoms include misery and unhappiness . . .

Surveying these definitions it is clear that the word 'depression' is introduced into most of them without further qualification or definition; the two exceptions are the manic-depressive psychosis where the mood is described as one of "widespread gloom and wretchedness" and the emotional disorder of childhood characterised by "misery and unhappiness". For the rest it seems that the user is free to include any concept of the term depression that he wishes. The definition of Neurotic Depression is particularly vague and there are so many generalisations that the only certain statement is that symptoms of delusions or hallucinations preclude the diagnosis.

The DSM-III

(This paper was written before the proposals for revision were seen by the author. Inspection of the draft for DSM-III-R indicates that major revision of the following comments is not necessary). In this system the terms neurosis and psychosis have been abolished and diagnostic categories are independent of supposed aetiology. Depressive disorders are defined in the terms of the prevailing symptoms, their intensity and their duration. These are classified under the major category of Affective Disorders into Major Affective Disorders, Other Specific Affective Disorders and Atypical Affective Disorders. (The authors are apologetic about the use of the German word *Affekt* instead of the English word Mood but state that tradition has made this inevitable). Apart from the Affective Disorders there is only one other category in which the word 'depression' is used and that is *309.00 Adjustment Disorder With Depressed Mood.*

296.2 *Major Depressive Episode*
For this category to be correct the subject must experience one of two alternative symptoms and experience four other symptoms from a list of eight. The first two symptoms are: 'dysphoric mood' and 'loss of interest in all or almost all usual activities and pastimes (anhedonia). Dysphoric mood is defined by a choice of epithets: depressed, sad, blue, hopeless, low, down in the dumps and (surprisingly) irritable. The list of eight symptoms, of which four must have been present nearly every day for at least 2 weeks (in children under 6, at least three of the first four) are: a) poor appetite or significant weight loss or increased appetite and significant weight gain; b) insomnia or hypersomnia: c) psychomotor agitation or retardation; d) loss of interest or pleasure in usual activities or decrease in sexual drive; e) loss of energy, fatigue; f) feelings of worthlessness, self-reproach or excessive inappropriate guilt; g) diminished ability to think or concentrate; h) recurrent thoughts of death, suicidal ideation or attempt.

The category can be further classified as 'With Psychotic Features' when either mood congruent or mood incongruent psychotic features are present or as 'Melancholia' if some of the symptoms are of more severe degree and there is evidence of diurnal variation of mood. It should be noticed that anhedonia appears in both sections of the definition so making it unlikely that the category could be correctly applied in the absence of that symptom.

Two other categories of affective disorder depend upon symptoms of lesser severity than Major Depressive Episode and chronicity. These are:

301.13 *Cyclothymic Disorder.*
During the past two years numerous periods during which some symptoms characteristic of both the depressive and the manic syndromes were present but not of sufficient severity and duration to meet the criteria for a Major Depressive or Manic Episode. . . .

The disorder must not be simply a partial remission of bipolar disorder.

300.40 *Dysthymic Disorder*
It is recognised that this may be termed depressive neurosis in other classificatory systems. The disorder must have been present for two years; it usually commences at a young age; chronic physical illness or psychosocial stresses, personality disorder or an affective disorder which does not fully remit are predisposing factors; the symptomatic criteria are the same two mandatory alternatives as in Major Depressive Disorder and then three of a list of 13 accessory symptoms must be present.

296.82 *Atypical Depression*
This is a residual category for individuals with depressive symptoms who cannot be diagnosed as Major or Other Specific (Cyclothymic or Dysthymic) or Adjustment Disorder.

The DSM-III provides fairly well defined criteria for categorisation but unfortunately a heterogeneous group of individuals may be included under each of the categories.

A further problem with the category Major Depressive Episode is that many physically sick individuals could be included simply on account of their physical illness and without the necessity to postulate a presence of a mental disorder; thus depressed mood, loss of appetite or weight, insomnia, loss of energy, loss of sexual drive and recurrent thoughts of death may all occur in serious physical illness. This failing, widely overlooked, is examined by Kathol and Petty (1981). The inconsistencies arising from different definitions of depression have been studied by Sashidharan (1985).

Individual Diagnostic Systems

Research findings and the possibility of extrapolation of meaning to other contexts is seriously hampered by the variety and imprecision of diagnostic systems and also by the adoption system individual to a particular research group. Space allows only one example of such an individual system which is, however, selected on account of the fundamental questions and conclusions about the nature of depression arrived at by the leader of the research team, Professor George Brown and his associates. The criteria for 'case' and 'borderline case' of depression are based on the Present State Examination symptoms (Finlay Jones *et al*, 1980). For a case Depressed Mood is mandatory and then any four of a list of ten symptoms: Hopelessness, Suicidal Ideas or Actions, Weight Loss, Early Waking, Delayed Sleep, Poor Concentration, Neglect Due to Brooding, Loss of Interest, Self-Depreciation, and Anergia. Symptoms must have been present for 2 weeks but there are no clear criteria of severity. Given the difficulties, already noted, in the PSE definition of Depressed Mood it is therefore clear that these criteria constitute a very wide trawl into which large numbers of sick, poor or unhappy people may be caught up. It is perhaps not surprising that Brown and his colleagues find this state they define as 'depression' to be related to social and personal disadvantage and also to have a high prevalence.

Rating scales

Many research studies have based the definition of a 'case' of depression on a certain score having been attained on one or other of the large number of depression rating scales. The different construction of these scales, and their individual characteristics of item array, cause serious difficulties in comparing one study using one scale with another using a different scale. The procedure of arriving at a definition of a case by adding up numbers derived from severity grade score of a number of symptoms has a certain attraction until it is realised that one case may be very different from another. As an instance of this, Cooper & Fairburn (1986) found that a group of bulimic and a group of depressed patients had similar scores on the Montgomery-Åsberg Depression Rating Scale but different symptoms, or scale items, contributed strongly to the overall score in the two groups.

The widely used Hamilton Depression Scale (Hamilton, 1967) consists of 17 items: depressed mood, feelings of guilt, suicidal ideation, work and activities, insight, retardation, agitation, insomnia (early, middle and late), psychic anxiety, somatic anxiety, gastrointestinal symptoms, general somatic symptoms, genital symptoms (loss of libido or menstrual disturbances), hypochondriases and loss of weight. The last six of these items are symptoms of somatic disturbance and account for 31% of the possible total score; if the insomnia items, which so often rate highly in physical illness are included, then 42% of the total is accounted for. The Depression Inventory (Beck *et al*, 1961) is another widely used 'depression' scale and again contains a high proportion of symptoms relating to somatic disturbance. Stewart *et al* (1965) pointed out that such symptoms are common in medically ill patients and advised that such assessment scales for depression should be avoided but their advice has largely been unheeded. An improvement on previous scales is the Montgomery-Åsberg Depression Rating Scale (Montgomery & Åsberg, 1979): this scale is oriented toward psychic symptoms and are: apparent sadness, observed sadness, inability to feel, difficulty in concentration, inner tension, pessimistic thoughts, suicidal thoughts, lassitude, reduced sleep, and reduced appetite. However even here 30% of the total score is accounted for by the last three items and all three commonly occur in all illnesses and states of distress, not just depression.

The range of self-assessment scales are popular as screening instruments for the presence of mood disorder. They are open to the same fallacies. For instance Kutner *et al* (1985) studying depression and anxiety in dialysis patients found that scales containing disease related items yielded exaggerated scores. The depression scale they used was the Self-Rating Depression Scale (Zung, 1965); this contains 20 statements, five of which are: 'I have trouble sleeping at night', 'I eat as much as I used to', 'I notice I am losing weight', 'I have trouble with constipation', 'My heart beats faster than usual'. Other items which may well be rated highly by people who suffer from physical sickness or are under some other adversity are: 'I feel that others would be better off if I were dead', 'My life is pretty full', 'I feel I am useful and needed', 'I feel hopeful about the future', 'I enjoy looking at, talking to and being with attractive women/men'. Altogether over half of the items of this widely used depression scale are composed of feelings or symptoms which do not necessarily indicate the presence of a mental illness.

The majority of depression rating scales contain a motley collection of symptoms, attitudes and feelings and will produce high prevalence rates and a large proportion of false positives if case detection relies upon them.

Discussion

In 1984 Mayou pointed again to the well-known fact that lack of agreement on definitions of disorders causes great difficulty in psychiatry. In no area is this more true than in the concept of depression and Andreason (1982) wrote: "Although affective disorders have been recognised for thousands of years, clinicians and researchers have still not reached agreement on the basic concepts of these disorders and the best methods of classifying them". Richmond & Barry (1985) surveyed the confused areas between mental illness and 'normal' states of distress, the large numbers of professional and other workers engaged in these grey areas and the potential enormous waste of resources and misapplication of treatments; they concluded: "Better definitions of clinical disorders must be developed *e.g. differentiating demoralisation from depression.* Psychiatry must clarify the areas where its expertise overlaps with other mental health disciplines (Lowy) and take the initiative in reallocating resources so that the paradox of increasing resources creating additional need can be overcome". [My italics]

Psychiatry is still overconcerned with providing classificatory systems but these too often end as mere pigeon holes into which patients can be placed with their appropriate diagnosis but frequently the diagnosis fails to lead to correct treatment or else treatments are applied blanket-fashion no matter what the diagnosis. In a leading work on depression (Weissman & Paykel, 1971), the authors wrote: "Attempts have been made to isolate within clinical depression two or more types, classified by such labels as neurotic, psychotic, reactive, endogenous. These are of considerable research interest. . . . However their practical application in guiding treatment is still limited".

At the time when psychiatrists confined their attention to patients admitted to mental hospitals there was rarely much difficulty about the diagnosis of the depressive phase of manic-depressive psychosis. This is a severe illness with fairly stereotyped symptomatology; the addition of information from the course of the disorder, the history of mania and familial history leads to fairly certain conclusions about the correct management and treatment. But the situation has now changed in three respects. a) Attention is now paid to the milder mood disorders occurring in the community and non-psychiatric hospital departments; b) workers other than psychiatrists are involved in the detection and treatment or care of people suffering from these mild mood disorders; c) finally, effective treatments, both psychological and pharmacological are now available, or rather such treatments can be effective when applied in the correct circumstances.

At the present time there is a vogue for community studies in the field of depression. Commonly, rating scales or diagnostic systems of one type or another are applied and a high prevalence of 'depression' is revealed. Researchers wonder at the fact that general practitioners, cancer physicians, neurologists and so on should be so inept at diagnosing this treatable condition but all that has happened is that a larger number of people have been declared to be in the company of the mentally ill or in need of some type of psychiatric or psychological treatment which is frequently not specified.

In future, research in this area must rest upon the need to provide more certain guidance on whether or not treatment is required and if so, what form that treatment should take. Now that effective antidepressant drugs are available the next essential task of psychiatry is to provide clear working guidelines of who might reasonably be expected to respond to such prescription, cutting short much misery and confusion and the frustration of other physicians and psychological therapists to help them.

How then may 'depression in this mild form be defined and assessed? One solution would be to stop using the term altogether and to describe states of mind in simple terms: grief at loss, poor self-esteem, demoralisation due to adversity and so on. The American DSM has made a start by reverting to the old term 'melancholia to describe the severe biogenic depressive illness; but what of the milder biogenic disorder, the state which has a high chance of improvement with antidepressant drug treatment, i.e. Kräupl Taylor's 'hypomelancholia'? In this paper I have put forward the reasons why current diagnostic systems are unsatisfactory and are frequently misleading. The wide range of current depression rating scales are also mostly misleading since they are composed of a motley of items on which any sick or unhappy person may score highly. Reliable laboratory tests are not available, possibly because attempts have been made to validate them on poorly defined clinical samples. In any case a laboratory test would have to be very easily conducted to be of real value to the large numbers of people who are involved in the management of 'depression'.

A solution to the problem of defining mild biogenic depression must rest upon the recognition of well defined clinical features which may relatively easily be established as being present or absent. For all the foregoing reasons these clinical symptoms must not include somatic symptoms which may occur in a wide variety of bodily illnesses, nor must a non-specific state such as insomnia and lack of energy be included. Symptoms indicative of severe mood disorder, such as suicidal preoccupation are unsuitable for they would lead to a high false negative rate. Referring again to the list of symptoms in the DSM definition of Major Depressive Disorder, what remains? There remain: depressed mood (dysphoria) loss of pleasure response (anhedonia), psychomotor retardation or agitation, feelings of worthlessness, self-reproach, inappropriate guilt, and diminished ability to think or concentrate. Two further symptoms from the list of 'depressive' symptoms in the PSE may be considered: hopelessness and neglect due to brooding, although both have implications of a severe disorder.

Kräupl Taylor described the essential hypomelancholic symptoms: "There is usually a decrease in mental and physical activity, in self-confidence and in the enjoyment of pleasure. The patient lacks energy and initiative in general". Another student of mild biogenic depressive disorders is Klein (1974); he recognised that stress sometimes was a factor in the precipitation of depressive states which were nonetheless to be considered biogenic in terms of having a high likelihood of response to antidepressant drugs, and so he coined the term 'endogenomorphic depression' and described the pathognomonic feature of this state: "A sharp, unreactive, pervasive impairment of the capacity to experience pleasure or to respond affectively to the anticipation of pleasure".

Which features then may be taken to be pathognomonic of the mild biogenic depressive state? Depressed mood is too diffuse a concept, too ill-defined as witness the number of epithets which may be provided to substitute for it. Feelings of worthlessness and guilt are too liable to have a basis in reality i.e. to be psychogenic; hopelessness may also be based upon the undeniable situation of the person and therefore unreliable as an indicant of biogenic depression. Psychomotor retardation is a reasonable proposition, a feeling of being slowed in thought and action; but in many patients prominent anxiety is present and agitation not retardation is the experience; moreover retardation is too liable to be a symptom of physical diseases, such as parkinsonism or arthritis, to be a reliable guide to the presence of biogenic depression in the setting of physical illness.

Of all the states anhedonia emerges as the central and reliable symptom of hypomelancholia. Anhedonia, the loss of ability to experience pleasure may also be fairly reliably ascertained by a few key questions: does the sight of flowers or a beautiful sunset lift the heart or the sound of one's favourite music make one want to sing with it? Can one anticipate with pleasure, look forward to a usually enjoyed event such as a weekend outing or a visit from a friend? Does one still pursue old hobbies or pastimes with real pleasure or does one simply do them for the sake of doing something or to put on appearances? And can one still enjoy a good joke with real mirth?

Some time ago we were asked by a physician to help him sort out, in his busy medical practice, which of his patients were just unhappy, demoralised or miserable for some other reason and which may be helped by the prescription of an antidepressant drug. As a result of this exercise we devised the Hospital Anxiety And Depression Scale (Zigmond & Snaith, 1983); this is fully described elsewhere but the essential feature is a subscale called 'depression' which comprises seven items: one is a 'dysphoria' item—I feel cheerful—and another a retardation item, but the essential basis is anhedonia and the other five items describe this experience in one form or another. The scale therefore measures anhedonic depression which we take to be the best indicant of hypomelancholia and we advised our colleagues to consider prescribing antidepressants if a high score appeared in this scale. Kendell (1982) advised that two or more criteria should be used when selecting subjects for biological research in mental illness and I also advocated the combined use of both a researcher administered and a self-assessment scale (Snaith, 1981).

The definition of the ubiquitous states that masquerade under the term 'depression' is of central importance and no real progress will be made in research and the development of clear guidelines to effective intervention until this is done. Considering the vastness of the sea of human unhappiness and the huge numbers of people attempting to fish souls out of it, this definition is perhaps the most urgent problem demanding solution by psychiatrists. In this essay I have developed the view, based upon examination of the work of others and on my own clinical experience, that anhedonia, if it is persistent and not adequately accounted for by the person's circumstances is at present the nearest guide to the biogenic depressive disorder. This suggestion needs to be put to the test of critical research.

References

American Psychiatric Association (1980) Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. Washington DC American Psychiatric Association.

Andreason, N. C. (1982) Concepts and classification of depression. In: *Handbook Of Affective Disorders* (ed. E. S. Paykel) Edinburgh and London: Churchill Livingstone.

Ascher, E. (1952) A criticism of the concept of neurotic depression. *American Journal of Psychiatry*, **108**, 901–908.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–571.

Bevan-Lewis, W. (1899) *Textbook of Mental Diseases*. London: Charles Griffin and Co.

Cooper, P. J. & Fairburn, C. G. (1986) The depressive symptoms of bulimia nervosa. *British Journal of Psychiatry*, **148**, 268–274.

Finlay-Jones, R., Brown, G. W., Duncan-Jones, P., Harris, T., Murphy, E. & Prudo, R. (1980) Depression and anxiety in the community: replicating the diagnosis of a case. *Psychological Medicine*, **10**, 445–454.

Hamilton, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–296.

Kathol, R. G. & Petty, F. (1981) Relationship of depression to medical illness. *Journal of Affective Disorders*, **3**, 111–121.

Kendell, R. E. (1969) The continuum model of depressive illness. *Proceedings of The Royal Society of Medicine*, **62**, 335–339.

—— (1976) The classification of depression. *British Journal of Psychiatry*, **129**, 15–28.

—— (1982) The choice of diagnostic criteria for biological research. *Archives of General Psychiatry*, **39**, 1334–1339.

Klein, D. F. (1974) Endogenomorphic depression. *Archives of General Psychiatry*, **31**, 447–454.

Klerman, G. L., Endicott, Jean, Spitzer, R. & Hirschfeld, R. M. A. (1979) Neurotic depression: A systematic analysis of multiple criteria and meanings. *American Journal of Psychiatry*, **136**, 57–61.

Kräupl Taylor, F. (1966) *Psychopathology: its Causes and Symptoms*. London: Butterworth.

Kutner, N. G., Fair, P. L. & Kutner, M. H. (1985) Assessing depression and anxiety in chronic dialysis patients. *Journal of Psychosomatic Research*, **29**, 23–31.

Lewis, A. J. (1934) Melancholia: a historical review. *The Journal of Mental Science*, **80**, 1–42.

Mayou, R. (1984) Sick role, illness behaviour and coping. *British Journal of Psychiatry*, **144**, 320–322.

Meyer, A. (1905) Discussant in: The classification of melancholias. *Journal of Nervous & Mental Diseases*, **32**, 112–117.

Montgomery, S. A. & Åsberg, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, **134**, 382–389.

Richmond, A. & Barry, A. (1985) More and more is less and less: the myth of massive psychiatric need. *British Journal of Psychiatry*, **146**, 164–168.

Sashidharan, S. P. (1985) Definitions of psychiatric syndromes—comparison in hospital patients and general population. *British Journal of Psychiatry*, **147**, 547–551.

Snaith, R. P. (1981) Rating Scales. *British Journal of Psychiatry*, **138**, 512–514.

Stewart, M. A., Drake, F., Winokur, G. (1965) Depression among medically ill patients. *Diseases of The Nervous System*, **26**, 479–485.

Weissman, M. M. & Paykel, E. S. (1971) *The Depressed Woman: A Study In Social Relationships*. Chicago and London: University of Chicago Press.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification In Accordance With The Ninth Revision Of The International Classification Of Diseases*. Geneva: WHO.

Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

Zung, W. W. K. (1965) A self-rating depression scale. *Archives of General Psychiatry*, **12**, 63–70.

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Brief Reports

Case Report: Severe Depressive Illness in the Context of Hypervitaminosis D

Hypercalcaemia, whether due to undiagnosed hyperparathyroidism or some other cause, can lead to clinical depression. The patient described here developed a severe depressive illness in the setting of hypervitaminosis D after 15 years of inappropriate therapy with calcium gluconate and strong calciferol. The importance of monitoring serum calcium levels in anyone on vitamin D therapy is stressed.

It has been known for some time that hyperparathyroidism may present as a psychiatric disorder varying in type from a minor depression to frank psychosis and even catatonic stupor (Paterson, 1974). Karpati & Frame (1964) studied 14 cases of primary hyperparathyroidism and noted that 42% of the patients had subjective or objective neuropsychiatric manifestations. These were of such a severe nature in four of the cases that a neurological or psychiatric consultation was obtained. Reinfrank (1961) described a case of hyperparathyroidism that presented with depression of such severity that a course of ECT was contemplated. In Petersen's (1968) series of patients with hypercalcaemia, mostly arising from hyperparathyroidism, 36% showed mental changes such as depression, listlessness and lack of initiative and, sometimes, irritability and explosive outbursts. Flanagan *et al* (1970) described a large family suffering from familial hyperparathyroidism, where psychiatric features were prominent. The severity of psychiatric signs correlated with the degree of hypercalcaemia in each case.

The reason for emotional disturbances in hyperparathyroidism is not yet clear. Gatewood *et al* (1975), however, feel that the hypercalcaemia is basically responsible because of the general dampening effect that high blood levels of calcium have on neurological function. According to Lishman (1978) the cause of any psychiatric disorder lies chiefly with the elevation of the serum calcium.

Hypercalcaemia from any cause can give rise to disabling symptoms such as fatigue, malaise, thirst and polyuria (Fourman & Leeson, 1959). Other common symptoms include anorexia, nausea and vomiting and constipation.

One well recognised cause of hypercalcaemia is excessive therapy with vitamin D or one of its analogues. Karpati & Frame (1964) describe 13 cases of hypercalcaemia arising from causes other than hyperparathyroidism. Three of these resulted from vitamin D intoxication. Paterson (1980) in a recent survey describes 21 patients who suffered from vitamin D poisoning. Nine of these cases presented with fatigue or confusion or both (Paterson, personal communication). Paterson emphasised that vitamin D is a cumulative drug so that the plasma calcium level at any particular time represents the combined effect of therapy given over a period of several months. Vitamin D is stored for long periods in both adipose tissue and muscle.

Carman & Wyatt (1979), in a double-blind study, observed that patients with a history of periodic psychoses experienced relapses 2 weeks after being started on dihydrotachysterol (DHT), a synthetic analogue of vitamin D, which causes rises in both serum calcium and serum phosphate.

Case report

The patient, a 58-year-old woman, was referred for an urgent psychiatric opinion as she had stated that she felt she could no longer carry on and she wished to die. At interview the patient sat in a languid, drooping fashion in an armchair. She presented as a pale, pathetic and thin figure. Her speech was retarded and delivered in a monotone. Much of the patient's thought content was morbid. Shortly before admission she had retired to bed each night hoping she would never wake up.

For many years she had been troubled with constipation and lower abdominal discomfort. Various laxatives had been tried without effective relief. She stated her general misery as follows: 'I know I am dying, because I have been constipated so long and I can't take any more pills for the trouble.' Insomnia, affecting all parts of the night, had been a further feature.

During the previous 2 months, the patient had lost 7 kg in weight. She had been aware of loss of appetite, and occasionally nausea, for a considerable time before this. There had been accompanying difficulty in swallowing foods and her diet comprised fluids or semi-solids. In view of the patient's state of deep depression, and her frail physical state, immediate admission to the local psychiatric hospital was arranged.

From the psychiatric point of view, the patient was extremely depressed and her mood was noted to be unaffected by external events. Because of her excessive dependence on her husband the patient had felt extremely guilty about the effect that her condition was having on him. She was also extremely anxious that some calamity might befall her husband and in that event she would be unable to cope. A loss of self-respect had been present for some time and the patient said she was ashamed that she was no longer able to keep up her usual tidy appearance or do her hair properly. There was a considerable degree of psychomotor retardation. The patient frankly said that life was not worth living and she would be better away from it all. There was no evidence of thought disorder, delusional thinking or hallucinations. With respect to her memory, the patient was able to give a reasonably consistent and clear history and there was no evidence of memory defect on formal testing. She did admit, however, that she had recently had a tendency to mislay things. She herself put this down to periodic failure of concentration. There was some degree of diminished concentration. Orientation was correct in all respects. The patient's level of consciousness was diminished, resembling that seen in pre-stuporose states.

Normally an avid reader of novels, she had been, of late, unable to read either books or newspapers. Routine tests for intellectual functions were administered but showed no evidence of organic brain damage. There was an awareness of recent events and she appeared to be of average intelligence.

Physical examination revealed recent evidence of weight loss. The patient weighed 44.5 kg (height 166 cm). There was marked facial pallor and also signs of anaemia in the tongue and mucous membranes. The physical examination was otherwise unremarkable apart from the presence of a laparotomy scar.

The patient, an only child, had a middle income background. There was no history of neurotic traits during the patient's development years. Her schooling was marked by achievements, both academic and social. After leaving School, she worked as a shorthand typist before her marriage. She had no children. The patient and her husband had marked interpersonal difficulties. The husband worked in a job which he loathed and possibly, partly as a result of this, he had been abusing alcohol for some time. Interviewed at the time of his wife's admission to hospital, the husband appeared to be unaware of how unwell his wife had been.

For 10 years the patient had suffered from periodic agoraphobic symptoms for which she had received both behaviour therapy and a course of clomipramine.

The patient's first serious physical illness had been 29 years earlier when she developed thyrotoxicosis. Her principal symptoms were exhaustion, emotional lability and a choking sensation on lying down. Partial thyroidectomy was done. There was an episode of post-operative tetany.

The patient experienced alopecia areata 6 years later. This was thought to have been of emotional origin. Five years later a D & C was carried out for menopausal menorrhagia. A further 3 years later, the patient was referred to a physician because of complaints of tiredness and feelings of panic when out shopping. A physical examination and extensive tests were all negative apart from a marginally reduced serum calcium (2.10 mmol/litre). It was suggested that this hypocalcaemia had occurred as a result of the removal of parathyroids at the time of the thyroid surgery. She was put on calcium gluconate tablets 600 mg *t.d.s.* and strong calciferol tablets 1.25 mg each day. She remained on this medication till the occasion of her in-patient psychiatric treatment 15 years later.

At the time of her admission to hospital the most striking abnormality was that of an elevated serum calcium 3.12 mmol/litre (normal range 2.15–2.65 mmol/litre) and a lowered serum phosphate 0.48 mmol/litre (normal range 0.8–1.55 mmol/litre). Serum electrolytes, blood urea, blood glucose, serum albumin, liver function tests and T4 were within normal limits. Haematological values were as follows: haemoglobin 9.4 g/dl, PCV 0.30, MCHC 31.5%, MV 97 fl, WBC 10.7×10^{12}/l, ESR 15 mm/h (Westergren). Blood film revealed normocytic to slightly macrocytic RBCs which were normochromic. Some elliptocytes were seen. There was marked thrombocytopenia. Leucocytes were in normal proportion. Serum B12 and folate were within normal limits. Faecal occult blood was positive on two out of three occasions. The chest X-ray was reported on as follows: "The heart is not enlarged. There are numerous calcified foci in the left lung field. The lung fields appear otherwise clear." A barium swallow and follow through, as well as a barium enema, were normal.

It was considered that the patient's depressive illness, which was essentially of the endogenous variety, had occurred in response to the abnormally high levels of blood calcium. Both the calcium gluconate tablets and the strong calciferol tablets were discontinued after the patient's admission to hospital. The blood calcium and phosphate levels were checked regularly. The former reached normal levels after 9 weeks, the latter after 6 weeks. She remained depressed and lethargic until 3 weeks following admission. At that stage the serum calcium had dropped from 3.12 mmol/litre to 2.94 mmol/litre. Improvement, both physical and emotional, started about this point and continued throughout the rest of her 2-month stay in hospital.

Medication comprised oral iron therapy, a hypnotic periodically and laxatives. By the end of her hospital stay the haemoglobin level had raised slightly to 9.9 g/dl. She was discharged on no medication.

Follow up for 1 year from both the psychiatric and medical point of view revealed no serious problems. Her mood remained stable and the serum calcium levels remained within normal limits. Sadly, 6 months after that follow up she had a further episode of depression and drowned herself. Despite this it was felt that the short-term response to correction of the plasma calcium was worthwhile.

Discussion

Symptoms of vitamin D poisoning can occur as early as 12 days or as late as 6 years after the commencement of therapy (Paterson, 1974). In the present case the acute toxic effects, in the form of suicidal depression, occurred 15 years after treatment with a daily dose of 1.25 mg of calciferol and 1.8 g of calcium gluconate. A failure to regularly monitor the patient's serum calcium, and adjust the calciferol dosage accordingly, had led to the hypercalcaemia remaining undetected.

The case under discussion had other features seen commonly in hypercalcaemia, i.e. abdominal pains and calcification in a lung. A less prominent aspect was the normocytic normochromic anaemia seen in the patient (Gabriel *et al*, 1970). It is postulated that this type of anaemia results from damage to the kidneys or the marrow by the hypercalcaemia.

This patient was falsely assumed to have had postoperative hypoparathyroidism. As a consequence of this incorrect diagnosis she received inappropriate medication with calciferol and calcium gluconate for the next 15 years. By that stage she had developed a severe depressive illness, which necessitated inpatient treatment.

As the patient committed suicide by drowning 18 months after discharge from hospital, at a time when the hypercalcaemia had been corrected, the aetiological relevance of the chronic hypercalcaemia to the patient's presenting illness must be in some doubt.

The Scottish legal verdict of 'not proven' is perhaps the most appropriate comment on the significance of the association but this case report may prompt others to seek or publish details of more convincing cases.

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References

CARMAN, J. C. & WYATT, R. J. (1979) Calcium: pacesetting the periodic psychoses. *American Journal of Psychiatry*, **136**, 1035–1039.

FLANAGAN, T. A., GOODWIN, D. W. & ALDERSON, P. (1970) Psychiatric illness in a large family with familial hyperparathyroidism. *British Journal of Psychiatry*, 117, 693–698.

FOURMAN, P. & LEESON, P. M. (1959) Thirst and polyuria with a note on the effects of potassium deficiency and calcium excess. *Lancet*, I, 268–271.

GABRIEL, R., JOEKES, A. M. & ORTON, E. (1970) Hypervitaminosis D anaemia and renal failure. *Postgraduate Medical Journal*, 46, 455–457.

GATEWOOD, K. W., ORGAN, C. H. & MEAD, B. T. (1975) Mental Changes associated with hyperparathyroidism. *The American Journal of Psychiatry*, 132, 131.

KARPATI, G. & FRAME, B. (1964) Neuropsychiatric disorders in primary hyperparathyroidism. *Archives of Neurology*, **10**, 394.

LISHMAN, W. A. (1978) *Organic Psychiatry*. Oxford: Blackwell Scientific Publications.

PATERSON, C. R. (1974) *Metabolic Disorders of Bone*. Oxford: Blackwell Scientific Publications.

—— (1980) Vitamin D Poisoning: Survey of Causes in 21 patients with hypercalcaemia. *Lancet*, 1,1164–1165.

PETERSON, P. (1968) Psychiatric disorders in primary hyperparathyroidism. *Journal of Clinical Endocrinology and Metabolism*, 28, 1491–1495.

REINFRANK, R. F. (1961) Primary hyperparathyroidism with depression. *Archives of Internal Medicine*, **108**, 606–610.

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Tuberous Sclerosis and the Autistic Syndrome

A case of tuberous sclerosis presenting with autistic behaviour is described. Although tuberous sclerosis is a rare cause of the autistic syndrome, it should be more strongly suspected when autistic behaviour is associated with seizures in a young child.

Tuberous sclerosis, inherited in an autosomal dominant fashion, is a rare disease with a multiplicity of clinical presentations including mental retardation, seizures, adenoma sebaceum, nodular sclerosis of the cerebral cortex and tumours of the kidneys, eyes and other organs. It was Bourneville in the late nineteenth century who first recognised this condition as a pathological entity and he named it 'tuberose sclerosis' because of the potato-like or tuberous appearance of the brain lesions on gross examination.

The possible neuropsychiatric manifestations of tuberous sclerosis have been described (Critchley & Earl, 1932): early mental deterioration, mutism, absent speech development, bizarre attitudes and stereotyped behaviours especially in the hands and fingers, and primitive movements such as rocking and swaying. The parallels with Kanner's descriptions of early infantile autism are obvious. Below we describe a case of tuberous sclerosis which was initially diagnosed as infantile autism and discuss the early diagnosis of tuberous sclerosis with this atypical picture.

Case report

S.E., a 7-year-old black male was referred to a children's inpatient mental unit by school psychology services for contingency management of behavioural problems. School assessment had revealed that he could not be taught or managed in a classroom situation. His main problems were hyperactivity, poor language function consisting of single words only, and autistic behaviours such as repetitive rocking and chewing of non-edible objects. He interacted minimally with peers and exhibited poor eye contact. Intellectual assessment prior to admission put him at an unspecified level of retardation.

He was the product of a normal pregnancy, weighing 7 lb 2 oz at birth. Soon after birth, he developed neonatal hyperbilirubinaemia which was treated with phototherapy. Development was normal until 8 months of age, when he developed a seizure disorder. Infantile spasms and possible tuberous sclerosis were considered at that time, but EEG and biopsy of hypopigmented skin lesions were non-conclusive.

The patient was treated with anticonvulsants for 2 years and remained free of seizures. However, speech delay, mental retardation and abnormal behaviour were obvious from the age of 3 years.

Physical examination at the time of admission to the unit revealed adenoma sebaceum of the face. Skull X-ray and head CT scan showed numerous intracranial subependymal calcifications. Eye examination was positive for a retinal phakoma.

Discussion

Although there is some overlap between the neuropsychiatric manifestations of tuberous sclerosis (Critchley & Earl, 1932) and infantile autism, tuberous sclerosis presenting with autistic behaviour is a rarely reported phenomenon (Creak, 1963; Mansheim, 1979). In our review of the reported cases, including the above report, we found that all patients had a history of onset of a seizure disorder before the age of 2 years. While the early onset of seizures is a predominant and fairly constant clinical feature of tuberous sclerosis (Pampligione & Moynahan, 1976), this is in contrast with the age pattern of seizures seen in autistic children (Rutter, 1970; Deykin & Macmahon, 1979) where the peak incidence of seizures tends to be at puberty.

We conclude that the clinical suspicion of tuberous sclerosis must be raised when the behavioural syndrome of autism is associated with seizures in a young child. While early diagnosis in tuberous sclerosis may not affect the ultimate prognosis, it will certainly be important for parental screening and genetic counselling.

References

Creak, E. M. (1963) Childhood psychosis: a review of 100 cases. *British Journal of Psychiatry*, **109**, 86–89.

Critchley, M. & Earl, C. J. C. (1932) Tuberous sclerosis and allied conditions. *Brain*, **55**, 311–346.

Deykin, E. & Macmahon, B. (1979) The incidence of seizures among children with autistic symptoms. *American Journal of Psychiatry*, **10**, 1310–1312.

Mansheim, P. (1979) Tuberous sclerosis and autistic behavior. *Journal of Clinical Psychiatry*, **40**, 97–98.

Pampligione, G. & Moynahan, E. J. (1976) The tuberous sclerosis syndrome: clinical and EEG studies in 100 children. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 666–673.

Rutter, M. (1970) Autistic children: infancy to adulthood. *Seminars in Psychiatry*, **2**, 435–450.

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Asperger's Syndrome and Aminoaciduria:

A Case Example

An 18-year-old male, originally diagnosed as suffering from infantile autism but with a developmental history and clinical picture in keeping with Asperger's syndrome, and showing current signs of impairment of higher cerebral functioning, is presented. The subject and several males of his family present behavioural disturbances of an enduring type. An aminoaciduria was discovered in the subject and his father. A possible relationship between the metabolic disturbance and the neurological and behavioural disturbance is raised. Mechanisms for this relationship are discussed, including sporadic hyperlysinaemia.

The relationship between organic and functional disorders has bedevilled theorists and practitioners for decades, and will continue to do so. Establishing a causal link between a metabolic lesion and problem behaviour is a difficult task. The following case presentation suggests such a connection. Unfortunately not all the pieces of the puzzle were available to make a complete picture, but sufficient is known to suggest an interesting possibility. The case also illustrates the usefulness of keeping an open mind about clinical data, with a willingness to rethink a problem, even in chronic cases.

Case presentation

'A' is an 18-year-old, unemployed male, born in England of Dutch-Indonesian parents. He was referred to the Adult Psychiatric Ward of a general hospital, because of a self-inflicted wrist laceration, 3–4 weeks of deteriorating behaviour and work performance at a Work Rehabilitation Centre, and despair by the Centre staff that his unemployability seemed due to a 'schizoid-autistic type personality'.

'A' was the eighth and ultimate offspring, following an uneventful pregnancy and delivery. Mother reports no problems in early infancy and initial milestones were adequately gained. He was noted to be different from other children as he became mobile and independent. Restlessness, a tendency to isolate himself and severely impaired speech development were the main features. Language skills were not acquired by the age of 5. He was a difficult child to make contact with and never related with others.

Following clinical assessment and a diagnosis of autism, he attended a school for autistic children. The parents were told that he had an above average IQ. Mother felt he was different to other children at the school in that he seemed more in contact with his surroundings. In 6 months he rapidly developed abilities, could speak coherently as well as read and write, and moved to an ordinary school.

The family emigrated to New Zealand and 'A' continued schooling in ordinary classes. Academic progress was reasonable with particular emphasis on mechanical and memory abilities. He continued to be a loner who exhibited an extreme need for perfection. A tendency to focus on microscopic details of rather idiosyncratic subjects was evident as was extreme emotional reaction to sudden environmental changes.

From 4 through 11 'A' would frequently wake in the night in a very agitated state talking of a vision which was described as a large black mask he could see in his room. There were no other descriptions of potential perceptual or delusional phenomena.

The isolation, minimal social involvement and preoccupation with singular pursuits continued during secondary education. At the time of impending secondary school examinations 'A' took an overdose of mother's nortriptyline. A convulsion and admission to a neurological ward resulted. Atypical involuntary movements, unsteadiness of gait, mild dysarthria and a mild right ptosis were noted. An EEG showed non-specific disturbance, most marked anteriorly. This featured low amplitude, fast activity and some low amplitude theta waves. No epileptiform activity was seen however. A CT scan performed was normal: 'A' was discharged with a diagnosis of 'hereditary neurological disorder not otherwise specified'.

After moderate success in the Sixth Form examinations he left school and was unable to find employment. The Labour Department referred him to a Work Rehabilitation Centre from where he was referred to the psychiatric service.

Family history

There is a very strong history of psychiatric disorder and personality disorder in the family. The paternal grandfather was an alcoholic with an explosive temper and frequent physical violence. The paternal grandmother had several admissions to mental hospitals and died in one, the exact nature of the disorder is not known—she was said to be violent at times. 'A's' father has had frequent business problems of 'unsound' deals. His wife sees him as a man with no insight into his own actions, lacking feelings for others and accordingly having bad social relationships. Interestingly, 'A's' father reports he is the best of a bad bunch when referring to his own siblings.

The maternal grandfather was very obsessional, frequently depressed and committed suicide. Mother has had prescriptions of antidepressant medication on a number of occasions.

Of the five male sibs four are seen as disordered. This tends to follow the same pattern with social inability, difficulties accruing from lack of concern for others, evasiveness, isolation, and physical slowness and clumsiness. Of the three female sibs only one shows disorder, which is quite different from the brothers, with extreme mood swings, confusion and illogicality. Her disorder only became evident at the age of 11, whereas the parents consider the boys have a lifetime of disordered patterns.

Examination findings

'A' was a tall, ungainly youth who was difficult to make contact with. He at times exhibited a number of small brief jerky, purposeless, seemingly involuntary movements of the hands, arms and trunk. When talking his breathing was poorly co-ordinated with the speech patterns. Little affect was displayed apart from considerable frustration when he was not able to perform cognitive testing.

He talked volubly without evidence of associative disorder. There were occasional grammatical errors and an intense preoccupation with particular subjects such as drugs, bee-keeping, and an intention to buy a racing cycle. There was no evidence of delusions or perceptual disorder.

He was fully orientated with good short- and long-term memory; concentration was adequate as was calculating ability. Slight dysarthria was evident. Rhythm disturbance in speech was typified by a questioning type raising of intonation at the end of a non-question statement. An echolalic tendency was evident, particularly of questions about social interaction or feeling state. Comprehension of simple and complex commands of either verbal or written type was good and there were no difficulties in reading or written expression.

Basic visuospatial organisation was adequate but on more complex copying tasks there was a lack of constructional ability. Performance of complex motor tasks and rapid alternating hand movements were impaired. An unusual gait was noted, especially lack of arm movement when walking. When asked to perform motor tasks, secondary and unwanted motor activity, such as the assumption of an unusual posture of an arm or hand was evident.

Psychometric data on the Weischler Adult intelligent scale (WAIS) (Weischler, 1958) showed a full scale IQ of 108, with a verbal IQ of 120, a performance IQ of 92, with a verbal/performance difference of 28. There was a subtest scatter with markedly low scores on Block Design, Object Assembly and Digit Symbol. The Graham & Kendall Memory for Design Test (Graham & Kendall, 1960) showed him to have borderline function.

Case summary

The developmental unfolding and old case records originally diagnosed as infantile autism, but on review consistent with Asperger's syndrome, showing the atypical speech patterns, problems with social interactions, repetitive activities, problems of motor co-ordination and pre-occupations with limited specialised topics (Wing, 1981) is presented briefly. There is evidence both on clinical examination and psychometric evaluation of deficits in higher cerebral functioning. These findings, coupled with the strong family history of behavioural disturbance in many of the male members, raised the possibility of some inherited and metabolic neurological disorder.

Investigations

Basic biochemical and haematological screening showed no abnormality. Chromosome pattern was normal male. An EEG showed signs similar to the previously described, namely anterior low-amplitude fast and theta activity.

Serum amino acid screen was normal. A urinary amino acid screen showed gross abnormality with elevation of arginine, ornithine, histadine, threonine, and serine. This picture is similar to that described as hyperdibasicaminoaciduria (Wasler, 1983). Further testing confirmed a transport mechanism deficit in that there was a less than normal elevation of serum lysine in response to a lysine load (Rajantie *et al*, 1980).

The strong family history of disturbed interpersonal behaviour with a striking male predominance encouraged us to screen the family for aminoaciduria. It was unfortunate that samples could not be obtained from the sibs. We were able to obtain samples from both parents. This showed the father to possess a similar aminoaciduria but to a lesser degree, whilst mother's urinary amino acid pattern was normal.

Discussion

We present a case with a history consistent with Asperger's syndrome, showing a current behavioural problem and signs of higher cortical dysfunction. Also demonstrated is an aminoaciduria of similar type to the described hyperdibasic aminoacidurias (Whelan & Scriver, 1968; Kihara *et al*, 1973; Rajantie *et al*, 1980; Wasler, 1983). Association of the two findings in a causal sense must remain speculative. The possibility that the psychiatric syndrome relates to aminoaciduria is heightened by the presence of the aminoaciduria in the subject's father and by the strong trend for male members of the family to display an unusual but uniform social deficit coupled with a motor skill deficit, suggesting a genetically sex linked mechanism.

The particular defect of the amino acid metabolism seen in this case is at variance with other reported disorders of basic amino acids (Whelan & Scriver, 1968; Kihara *et al*, 1973; Scriver & Rosenberg, 1973; Rajantie *et al*, 1983; Wasler, 1983). Typically elevations of urinary arginine, ornithine and lysine, are seen. Our case has, in addition, elevated serine, threonine and histadine. There is, however, added similarity to other described hyperdibasicaminoacidurias in the deficient handling of a lysine load, suggesting transport defect (Rajantie *et al*, 1980). The rarity of this defect has limited study. Reports (Wasler, 1983) would suggest at least two different syndromes; one is declared in early life and includes gross mental handicap, and the other can appear later with less profound but progressive mental impairment.

Reports of possible association of psychiatric symptoms and aminoacidurias involving the dibasic group are limited. Reviewing hyperdibasic aminoaciduria, Kihara *et al* (1973) consider that the amino acid metabolic abnormality is a result of a genetically determined alteration in the transcellular transport mechanism for the dibasic aminoacids. The finding of inability to handle lysine load is further supporting evidence of this. Whelan & Scriver (1968) discuss hyperdibasicaminoaciduria and its relationship to mental retardation. The possibility that the pathological agent is serum lysine is raised by both authors. They cite work that had been carried out, primarily with rats, showing sporadic hyperaminoacidemia can seriously impede the process of cerebral production of proteins. The suggestion ensuing from these observations is that sporadic episodes of hyperlysinaemia either experienced *in utero* or early infancy might seriously disrupt the development of particular aspects of nervous system integration.

Though obviously this case report raises far more questions than it is able to answer we feel it points to a potential area of worthwhile ongoing research. The human nervous system development is quite incomplete at birth and continues rapidly in early infancy. The development is especially evident in the neocortical areas and it is perhaps no accident this case displays deficits consistent with function ascribed to those areas. The importance of further studies to substantiate our speculation can be seen when considering the disorder phenylketonuria (Scriver & Rosenberg, 1973). When untreated this presents a severely debilitating neurological disorder. Comparatively recent development of simple screening procedures in the new born has allowed detection of cases before irreversible neurological damage has been caused and strict attention to diet removes the unpleasant sequelae.

If the hypothesised link between the hyperdibasicaminoaciduria and neurological system impairment is substantiated then we may both explain and even prevent some of the more unusual psychiatric syndromes such as Asperger's Syndrome that currently bedevil us.

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References

Graham, F. K. & Kendall, B. S. (1960) Memory-for-Design test: Revised general manual. *Perceptual Motor Skills*, **11**, 147–188.

Kihara, H., Valente, M., Porter, M. T. & Fluharty, A. L. (1973) Hyperdibasicaminoaciduria in a mentally retarded homozygote with a peculiar response to phenothiazines. *Pediatrics*, **51**, 223–229.

Rajantie, J., Simell, O. & Perheentupa, J. (1980) Intestinal absorption in lysinuric protein intolerance: Impaired for diamino acids, for normal citrulline. *Gut*, **21**, 519–524.

Scriver, C. R. & Rosenberg, L. E. (1973) *Amino-acid Metabolism and its Disorders*. Philadelphia: W. B. Saunders.

Wasler, M. (1983) Urea cycle disorders and other hereditary hyperammonemic syndromes. In *The metabolic basis of inherited disease* (eds J. B. Stanbury *et al*), 5th edn. McGraw Hill.

Wechsler, D. (1958) *The Measurement and Appraisal of Adult Intelligence* 4th edn, Baltimore: Williams & Williams.

Whelan, D. T. & Scriver, C. R. (1968) Hyperdibasicaminoaciduria: An inherited disorder of aminoacid transport. *Pediatric Research*, **2**, 525–534.

Wing, L. (1981) Asperger's Syndrome: A Clinical Account. *Psychological Medicine*, **11**, 115–129.

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Expressed Emotion in the Families of the Mentally Handicapped: A Pilot Study

The birth of a mentally handicapped child to a couple is a traumatic event. It has even been compared with a bereavement. In the crisis of family handicap, however, there is also anxiety, fright, guilt and shame. Some of these reactions have been denied by families but it has to be borne in mind that mothers of abnormal babies, although they are not aware of it consciously, may be protective and dominant (Prechtl, 1963). The reactions of parents of mentally handicapped may swing from extremes of overprotection to complete rejection. On the other hand, there is stress both emotional and physical in looking after the handicapped day in, day out. The amount of the emotional stress has not been quantified, but of the many forms of interactions between parent and offspring, over-involvement, criticism, hostility, and warmth can be evaluated using the Expressed Emotional scales.

Method

Expressed Emotion was assessed on the basis of the scales of (i) Hostility, (ii) Critical Comments, (iii) Positive remarks, (iv) Warmth—as has been done with the schizophrenic patients (Vaughn & Leff, 1976). The parents or next of kin of mentally handicapped individuals were interviewed according to a modified version of the Camberwell Family Interview Schedule. The reliability of such an interview schedule as a research tool has been established by Brown & Rutter (1966). The interview lasted 45–60 minutes. The biological father was alive in only four of the cases. The interview was tape recorded and the tapes were rated according to the training received. Out of the ten tapes, four were taken at random and checked by other research workers trained in the use of the scale (Table II).

The parents or carers looked after mentally handicapped individuals of both sexes between the ages of 16 and 50. The lower limit of 16 years was fixed, because this is the youngest age at which patients are admitted to Ely hospital. The upper limit of 50 years was arbitrarily chosen, because of the possibility that in the case of patients older than this, the parents may be dead or less able to respond in the interview by virtue of their age. The parents or carers of ten mentally handicapped individuals within the described age range were interviewed. The patients were chosen at random from a list of those who live at home but come to Ely hospital for short term care for regular family relief. It was explained to the interviewees that all the information they gave would be strictly confidential and used for research purposes only.

Discussion

The Expressed Emotion (EE) scale is being used with various populations. Apart from the pioneering work on schizophrenic patients, the Camberwell Family Interview Schedule is being modified and used with a population of intractable epileptic and autistic individuals. This pilot study attempted to use the instrument to evaluate EE in families looking after a mentally handicapped member. A striking finding was the total absence of hostility which can be explained by the fact that fathers (who tend to show more hostility than the mothers) were too few in

TABLE I
Author's results

Families		*Critical comments*	*Hostility*	*Positive remarks*	*Warmth*	*Emotional over-involvement*
[1]Mother and father of Jeanette	I	1	0	3	3	3 (Borderline)
Mother of Derek	II	0	0	0	2	2 (Low)
[1]Mother and father of Joseph	III	0	0	1	2	2–3 (Borderline)
Mother of Victoria	IV	1	0	2	2	2 (Low)
Adopted mother of Mary	V	0	0	1	2	0 (Low)
Sister of Audrey	VI	1	0	0	2	1 (Low)
Mother of Yvonne	VII	1	0	0	2	2 (Low)
[1]Mother and father of Maria	VIII	0	0	3	3	3–4 (High)
Sister of Rita	IX	0	0	0	1	1 (Low)
Sister-in-law of Avril	X	0	0	1	2	1 (Low)

1. Ratings pertain to mother who was the key informant

TABLE II
Co-rater's results

Families		*Critical comments*	*Hostility*	*Positive remarks*	*Warmth*	*Emotional over-involvement*
[1]Mother and father of Joseph	III	1	0	2	3	2 (Low)
Mother of Victoria	IV	0	0	1	2	1 (Low)
Mother of Yvonne	VII	2	0	0	1	2–3 (Low)
[1]Mother and father of Maria	VIII	1	0	2	3–4	4 (High)

1. Ratings pertain to mother who was the key informant

number and not interviewed separately. Critical comments which correlate with hostility reached a score of one in four of the families. Warmth was rated at 2–3 almost uniformly, whereas positive remarks were found in three of the families. Emotional over-involvement (EOI) also reached the accepted threshold in three of the families. In another two families, EOI was rated 2.

The study was started on the premise that parents of mentally handicapped members, have suppressed feelings which should be explored. The point in using a semi-structured interview based on the CFI was that like the parents of schizophrenics, parents of mentally handicapped usually know very little about the condition of their offspring and spend much of their day-to-day life with them. The 'face to face' contact was more than 40 hours a week, this has to be considered in the context of both the physical and the mental limitations of the individuals. Indeed some of the families said that the inability to have a conversation made the time spent together more intolerable.

The population chosen was in the 16–50 year age group. The life pattern resembled that of chronic schizophrenics who attend either a day centre or an industrial therapy unit. However, because of the age of the handicapped individuals, the parents had had a long time to adjust and therefore could appear to be more accepting of the situation. Moreover, in the case of older individuals it was often difficult for parents or carers to recall and discuss emotions that were aroused nearly two decades ago. However, the older parents or carers in contradistinction with younger parents or carers, had specific problems relating to their age group. One was anxiety about what would happen when they died or became physically unable to look after their handicapped relative. In those cases where there was physical handicap, parents said they could no longer cope with lifting and other physical effort involved in bathing and dressing. A more appropriate age group may therefore be the 5–10 year group.

Parents of mentally handicapped people also differ from parents of schizophrenics in many ways. Mental handicap is invariably detected during the first few years of life. This presents the parents with the option of either rejection or total dedication. This could also account for the low score of hostility which was observed. On the other hand there was no remarkable increase in the rating for warmth or emotional over-involvement which would reflect self-sacrifice through utter devotion.

In schizophrenia, even though the parents and some professionals may dispute the diagnosis, there are periods of remission which may give parents hope that the condition is not irredeemable. On the other hand, in mental handicap, once the diagnosis is made, some parents will go to great lengths to deny its reality.

As far as the questionnaire is concerned, the modified version is even shorter than the revised version of Vaughn & Leff (1976). Its brevity suggests that there are areas which need expanding namely regarding irritability and quarrelling. Observations made during the interviews indicate that there is a sense of dissatisfaction which should be taken into consideration in any further work. Despite these reservations, the overall rating of EOI is still very relevant in connection with mental handicap. It will enable the identification of those families who could be helped to cope with their feelings, thus allowing them to give their handicapped dependant the maximum scope for the development of potential.

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References

Brown, G. W. L. & Rutter, M. (1966) The measurement of family activities and relationships: a methodological study. *Human relations*, **19**, 241–263.

Pretchl, H. F. R. (1963) *Determinants of Infant Behaviour*, Vol II (ed. B. Foss). London: Methuen.

Vaughan, C. & Leff, J. (1976) *The British Journal of Social and Clinical Psychology*, **15**, 157–165.

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Delusional Infestation and Dementia: A Case Report

The case of an 82-year-old woman with delusional infestation and senile dementia of the Alzheimer type is reported. Her symptoms of infestation ceased dramatically with pimozide given for 5 weeks, and did not recur during follow-up over 9 months. The importance of organic factors in patients with this disorder is emphasised.

The syndrome of delusional infestation is a clinical state in which the patient has the unshakeable false belief that her skin is infested with live organisms. The condition has been variously described in the English literature under the names acarophobia, delusion of parasitosis, parasitophobia, dermatophobia and Ekbom's syndrome (Skott, 1975; Berrios, 1985), but we suggest that the term 'syndrome of delusional infestation' is a more accurate description and will be used here.

The syndrome was first described by the French dermatologist Thibierge in 1894 and is well known to dermatologists (Lyell, 1983) but less so to psychiatrists. The typical patient is a woman in late middle age who presents to a dermatologist complaining of cutaneous sensations which are delusionally attributed by her to a skin infestation with creatures such as lice, worms, maggots, fleas, bacteria and fungi. As proof of the infestation she may bring along an envelope containing the alleged organisms, which usually turn out to consist only of skin debris. The dermatologist is unable to find any evidence of infestation, and often the skin of the patient looks normal. However delusional infestation can occasionally be superimposed on a pre-existing skin disorder (Wilson & Miller, 1946; Wilson, 1952; Tullett, 1965; Hopkinson, 1970), follow on from a previous skin infection (Wilson & Miller, 1946; Hopkinson, 1973), or be associated with self-induced dermatoses resulting from attempts to remove the organisms with instruments or chemicals (Klauder, 1936; Wilson & Miller, 1946; Wilson, 1952; Obermayer, 1961; Tullett, 1965; Paulson & Petrus, 1969). The patient holds her conviction with absolute certainty and cannot be persuaded otherwise; any suggestion of referral to a psychiatrist usually meets with a flat refusal. She may go to extraordinary lengths to get rid of the creatures: fumigation of clothing, bedding and furniture may be demanded, and pest control officers (Edwards, 1977) and public health officials (Schrut & Waldron, 1963) consulted. These patients are very difficult to treat, but useful management guidelines have been provided by Gould & Gragg (1976).

The causes of delusional infestation are multiple; in this paper we wish to draw particular attention to the potential role of organic cerebral factors in the aetiology of this syndrome by describing the unusual case of delusional infestation occurring in a woman with dementia.

Case report

The patient is an 82-year-old married childless woman with no previous history of physical or psychiatric illness or family history of dementia. Her husband had noticed early signs of memory impairment and loss of domestic efficiency in his wife over the previous 6 months with an exaggeration of her premorbid personality traits of suspiciousness and possessiveness, but she had shown no evidence of delusional ideation or depression of mood. She was initially seen at home at the request of her general practitioner because of her complaint of skin infestation of 1 month's duration. She claimed that unseen flying insects had been settling on her face at night when she was in bed, and had laid their eggs in her skin and hair. The eggs had hatched and she could feel insects crawling in her skin. She wrapped her head and face in a silk turban at night and washed her hair regularly with vinegar to deter the insects. She spent many hours during the day in front of a magnifying mirror picking at her face with a pin to remove the eggs; periodically she would hold up the pin in triumph and coerce her husband into agreeing that he could see an egg impaled on the end. Her husband was also forced by her to arrange for a pest control officer to spray their bedroom with insecticide. However at no time did her husband believe that his wife was infested. Her general practitioner found no evidence of skin infestation on physical examination and treatment with chlorpormazine 50 mg *t.i.d.* failed to dislodge her delusion.

Mental state examination revealed an elderly slightly suspicious woman with excoriations on her nose and left cheek. She showed no depression of mood, hallucinations or delusions other than those concerning the insects. However she was disorientated in time and had evidence of both short- and long-term memory impairment, scoring only 18 out of 34 points on Hodkinson's Mental Test (1973). Her delusional system was well organised and she lacked insight into her illness, believing that she needed treatment from a dermatologist rather than psychiatric help.

A provisional diagnosis of delusional infestation associated with a possible dementing illness was made and pimozide at a dose of 8 mg daily prescribed. She responded very well to this treatment at home and her delusions completely resolved within a few days. However the drug was stopped after 5 weeks when she showed extrapyramidal side-effects. Physical examination and laboratory and radiological investigations to rule out systemic disease and secondary forms of dementia were normal; an EEG showed a bilateral excess of slower components and a CT brain scan revealed slight ventricular dilatation and cerebral atrophy. There has been a continual decline in her memory, intellect and personality over the ensuing 9 months in keeping with a clinical diagnosis of senile dementia of the Alzheimer type, and she has been regularly admitted to hospital during this period to give her husband some respite. However her symptoms of infestation have not recurred despite the discontinuation of pimozide.

Discussion

Before a diagnosis of delusional infestation is made, it is necessary to ensure that the patient is not actually infested. However as Champion (1983) points out, this is far from simple as scabies and other mite infestations can be easily overlooked, and a thorough physical examination is essential.

Most patients who present with a delusion of infestation are suffering from either a psychiatric illness or an organic disorder; in a small minority, there may be an interplay of both pathologies, or the organic factor may be entirely coincidental.

Delusional infestation occurs most commonly in the context of monosymptomatic hypochondriacal psychosis (MHP), a psychiatric illness characterised by a fixed, usually bizarre, solitary delusional belief of bodily disease or abnormality, which is unaccompanied by disintegration of personality, delusional elaboration or psychotic deterioration (Reilly, 1977; Munro, 1978, 1980). Delusional infestation may also arise in the course of an affective or schizophrenic illness (Wilson & Miller, 1946; Hopkinson, 1970, 1973) when it is accompanied by other relevant psychiatric symptoms, and also in folie à deux (Macnamara, 1928; Partridge, 1950; Wilson, 1952; Paulson & Petrus, 1969; Hopkinson, 1970, 1973; Evans & Merskey, 1972; Mester, 1975).

A clinical picture resembling delusional infestation has been reported in toxic and metabolic disorders such as delirium tremens (Lishman, 1978), cocaine psychosis (Tullett, 1965), amphetamine abuse (Skott, 1975), pellagra (Aleshire, 1954; Fish, 1967), and vitamin B12 deficiency (Pope, 1970), in which pathological cutaneous sensations may be experienced.

Ladee (1966) has reviewed the earlier evidence implicating the role of cerebral-organic factors in the aetiology of some cases of delusional infestation. The syndrome has been observed in patients with cortical and subcortical atrophy who show no intellectual impairment (Campanella, 1969; Schott *et al*, 1973; Heim & Morgner, 1980; Ungvári & Pethö, 1980; Sone, 1983), and in patients with brain tumours (Liebaldt & Klages, 1961; Miller-Kreuser, 1962; Ungvári & Pethö, 1980), alcoholic encephalopathy (Ungvári, 1984) and cerebral infarction (Valbuena Briones *et al*, 1984). Previous cases of delusional infestation associated with dementia have also been described by Wieser & Kayser (1966), Bauer & Mosler (1970) and Ganner & Lorenzi (1975).

The delusion of infestation in our patient, unlike most delusions in dementia, was fixed and well-systematised. Ekbom's (1938) suggestion that the delusion has its origin in true cutaneous sensations which are misinterpreted and not hallucinatory experiences, may be relevant in some cases with an organic aetiology; the abnormal skin sensations of senile pruritus may act as a fertile focus for the development of delusional infestation in the elderly, especially in those with cognitive impairment. Other systemic conditions to exclude in this age group include liver and renal disease, lymphomas, polycythaemia, iron deficiency, thyroid disease, carcinoma and drug reactions, all of which can cause a generalised pruritus without skin lesions (Champion, 1983).

Some workers (Riding & Munro, 1975; Munro, 1978; Reilly *et al*, 1978) have found pimozide to be particularly useful in the treatment of MHP patients with delusions of infestation, although its efficacy in other forms of the syndrome remains debatable. However, our patient's delusion resolved much too quickly for the improvement to be coincidental and suggests a specific beneficial effect of pimozide.

In conclusion, this case illustrates that delusional infestation, especially when it occurs in the elderly, may be symptomatic of an underlying organic disorder.

References

Aleshire, I. (1954) Delusion of parasitosis: report of successful cure with antipellagrous treatment. *Journal of the American Medical Association*, **155**, 15–17.

Bauer, A. & Mosler, A. (1970) Die Behandlung des Dermatozoenwahnes. *Arzneim. Forsch.*, **20**, 884–886.

Berrios, G. E. (1985) Delusional parasitosis and physical disease. *Comprehensive Psychiatry*, **26**, 395–403.

Campanella, G. (1969) Contributo allo studio del delirio dermatozoico (sindrome di Ekbom). *Acta Neurol. (Napoli)*, **24**, 903–927.

Champion, R. H. (1983) Pruritus. *Medicine International*, **27**, 1252–1254.

Edwards, R. (1977) Delusions of parasitosis. *British Medical Journal*, **i**, 1219.

Ekbom, K. A. (1938) Der präsenile Dermatozoenwahn. *Acta Psychiatrica et Neurologica Scandinavica*, **13**, 227–259.

Evans, P. & Merskey, H. (1972) Shared beliefs of dermal parasitosis: folie partagée. *British Journal of Medical Psychology*, **45**, 19–26.

Fish, F. (1974) *Fish's Clinical Psychopathology* (ed. M. Hamilton), 2nd edn. Bristol: John Wright.

Ganner, H. & Lorenzi, E. (1975) Der Dermatozoenwahn. *Psychiatria clin.*, **8**, 31–44.

Gould, W. M. & Gragg, T. M. (1976) Delusions of parasitosis: an approach to the problem. *Archives of Dermatology*, **112**, 1745–1748.

Heim, M. & Morgner, J. (1980) Zur Problematik der chronischen taktilen Halluzinose. *Psychiat. Neurol. med. Psychol. (Leipzig)*, **32**, 405–411.

Hodkinson, H. M. (1973) Mental impairment in the elderly. *Journal of the Royal College of Physicians of London*, **7**, 305–317.

Hopkinson, G. (1970) Delusions of infestation. *Acta Psychiatrica Scandinavica*, **46**, 111–119.

Hopkinson, G. (1973) The psychiatric syndrome of infestation. *Psychiat. clin.*, **6**, 330–345.

Klauder, J. V. (1936) Psychogenic aspects of skin diseases. *The Journal of Nervous and Mental Disease*, **84**, 249–273.

Ladee, G. A. (1966) *Hypochondriacal Syndromes*. Amsterdam: Elsevier.

Leibaldt, G. & Klages, W. (1961) Morphologische Befunde bei einer "isolierten chronischen taktilen Dermatozoenhalluzinose". Versuch einer Deutung. *Nervenarzt*, **32**, 157–171.

Lishman, W. A. (1978) *Organic Psychiatry*. Oxford: Blackwell Scientific Publications Ltd.

Lyell, A. (1983) Delusions of parasitosis. *British Journal of Dermatology*, **108**, 485–499.

Macnamara, E. D. (1928) A note on cutaneous and visual hallucinations in the chronic hallucinatory psychosis. *Lancet*, **i**, 807–808.

Mester, H. (1975) Induzierter "Dermatozoenwahn". *Psychiatria clin.* **8**, 339–348.

Miller-Kreuser, E. (1962) Symptomenbild der chronischen taktilen Halluzinose bei Hypophysenveränderung. *Med. Welt. (Stuttgart)*, **2**, 88–91.

Munro, A. (1978) Monosymptomatic hypochondriacal psychosis manifesting as delusions of parasitosis. A description of four cases successfully treated with pimozide. *Archives of Dermatology*, **114**, 940–943.

Munro, A. (1980) Monosymptomatic hypochondriacal psychosis. *British Journal of Hospital Medicine*, July, 34–38.

Obermayer, M. E. (1961) Dynamics and management of self-induced eruptions. *California Medicine*, **94**, 61–65.

Partridge, M. (1950) One operation cures three people. Effect of prefrontal leukotomy on a case of folie à deux et demie. *Archives of Neurology and Psychiatry*, **64**, 792–796.

Paulson, M. J. & Petrus, E. P. (1969) Delusions of parasitosis: a psychological study. *Psychosomatics*, **10**, 111–120.

Pope, F. M. (1970) Parasitophobia as the presenting symptom of vitamin B12 deficiency. *Practitioner*, **204**, 421–422.

Reilly, T. M. (1977) Monosymptomatic hypochondriacal psychosis: presentation and treatment. *Proceedings of the Royal Society of Medicine*, **70** (suppl. 10), 39–42.

Reilly, T. M., Jopling, W. H. & Beard, A. W. (1978) Successful treatment with pimozide of delusional parasitosis. *British Journal of Dermatology*, **98**, 457–459.

Riding, B. E. J. & Munro, A. (1975) Pimozide in monosymptomatic psychosis. *Lancet*, **i**, 400–401.

Schott, G., Marg, E. & Elsaber, H. (1973) Über eine familiäre Erkrankung an chronischer taktiler Halluzinose. *Psychiat. Neurol. med. Psychol., (Leipzig)*, **25**, 599–605.

Schrut, A. H. & Waldron, W. G. (1963) Psychiatric and entomological aspects of delusory parasitosis. Entomophobia, acarophobia, dermatophobia. *Journal of the American Medical Association*, **186**, 429–430.

Skott, A. (1975) Dermatozoenwahn—Ekboms syndrom. En litteraturöversikt. *Nordisk psykiatrisk Tidsskrift*, **29**, 115–131.

Sone, K. (1983) Über fünf Fälle von sogenanntem "Dermatound Enterozoenwahn"—psychopathologische und neuropsychologische Betrachtungen. *Folia Psychiatrica et Neurologica Japonica*, **37**, 37–55.

Tullett, G. L. (1965) Delusions of parasitosis. *British Journal of Dermatology*, **77**, 448–455.

Ungvári, G. (1984) Behandlung des Dermatozoenwahns durch Neuroleptika. *Psychiat. Prax.*, **11**, 116–119.

Ungvári, G. & Pethö, B. (1980) Zur Pathogenese des Dermatozoenwahn. *Psychiat. Neurol. med. Psychol. (Leipzig)*, **32**, 353–358.

Valbuena Briones, A., Avila Escribano, J. J., De Dios Perrino, C. & Castaneda Sáiz De Jáuregui, M. (1984) Delirio dermatozoico (parasitario). *Med. Clin. (Barc).*, **83**, 249–252.

Wieser, S. & Kayser, H. (1966) Die Psychiatrie des wahnhaften Parasitenbefalls. *Fortschr. Neurol. Psychiat.*, **34**, 5, 257–275.

Wilson, J. W. (1952) Delusion of parasitosis (acarophobia). Further observations in clinical practice. *Archives of Dermatology*, **66**, 577–585.

Wilson, J. W. & Miller, H. E. (1946) Delusion of parasitosis (acarophobia). *Archives of Dermatology*, **54**, 39–56.

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A Case of Resistant Schizophrenia

SIR: It is gratifying that our colleagues at the Joint Hospital (Roberts *et al, Journal,* December 1986, **149,** 789–793) have discovered neuroleptic-resistant schizophrenia. However, had they visited any of the large psychiatric hospitals, which they seem to prefer to call asylums, they would be likely to find a number of patients with this disorder.

Notwithstanding the failure of electroconvulsive therapy in this patient, I would suggest that lithium should be tried. Sometimes 'continuously floridly psychotic behaviour' has a masked affective component, as Crammer suggests in the paper.

We have reported the case of a 27-year-old married lady who required high doses of depot neuroleptics to control episodes of schizophrenia (as diagnosed at that time) associated with very disturbed behaviour and to maintain prophylaxis (Barnes & Bridges, 1980). Her severe symptoms were improved, but certainly not controlled, by the use of ECT. However, there was a good response to lithium, although, ultimately, she was found to be most settled on haloperidol (20 mg t.d.s.) and carbamazepine (400 mg t.d.s.) with procyclidine. Her compliance on this medication is good, and it has kept her out of hospital so far for the past four years. I should add that she was transferred originally to my care at Bexley Hospital from the Bethlem Royal Hospital, where it was considered that she was doing too much damage to both the staff and the fabric and hence "asylum" was needed.

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BARNES, T. R. E. & BRIDGES, P. K. (1980) *British Medical Journal, ii,* 274–275.

Anorexia Nervosa and Dysmorphophobia

SIR: I should like to comment upon Sturmey & Slade's paper (*Journal,* December 1986, **149,** 780–782). Anorexia nervosa and dysmorphophobia are functional disorders which usually occur in young individuals and are characterised by disturbances of the body image. In the former condition subjects characteristically perceive themselves as fatter than they are, as with the reported patient who "considered herself to be generally too 'broad'", whereas in dysmorphophobia, subjects typically complain about one particular aspect of their appearance, such as the nose, chin, mouth, breasts, or penis (Morselli, 1886; Hay, 1970; Thomas, 1984).

In anorexia nervosa and in the reported patient weight loss occurs, and consequently observed appearance cannot be considered normal. In dysmorphophobia there is a subjective feeling of ugliness or physical defect which the patient feels is noticeable to others, although appearance is within normal limits (Morselli, 1886).

I think that it is incorrect to consider that their patient exhibits dysmorphophobia, as the disturbance in body image was not localised, abnormality of body size was present, and there was a primary diagnosis of anorexia nervosa from which the dissatisfaction with appearance developed.

Making these distinctions is important if one wishes to identify and study a homogenous group of dysmorphophobics where an over-valued idea concerning appearance, in the absence of underlying classifiable psychiatric disease, is the principal abnormality.

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References

HAY, G. G. (1970) Dysmorphophobia. *British Journal of Psychiatry,* **116,** 399–406.

MORSELLI, E. (1886) Sulla dismorfofobia e sulla tafefobia. *Bolletino Accademia delle Scienze Mediche di Genova,* **6,** 100–119.

THOMAS, C. S. (1984) Dysmorphophobia: a question of definition. *British Journal of Psychiatry,* **144,** 513–516.

SIR: The report on anorexia nervosa and dysmorphophobia (Sturmey & Slade, *Journal,* December 1986, **149,** 780–782) demonstrates how easy it is to further complicate psychiatric nosology.

Certainly the history suggests that the patient warrants a diagnosis of anorexia nervosa; many of the classical features are present, including a disturbed body image. Dysmorphophobia, however, is confused yet again by an initial emphasis on physical appearance and then referring to it as a social phobia. Here the term is superfluous, because the feature of disturbed body image is a diagnostic criterion of anorexia nervosa. If the authors used this method of 'overlapping nosology', then why didn't they call the report "A study of anorexia nervosa, dysmorphophobia, social phobia, anxiety neurosis, obsessive-compulsive neurosis, and personality disorder, etc."?

It would be interesting to follow up this patient for a time. If her brother has another "schizophrenic attack" (*sic*) and she follows suit then we can label her "schizophrenic" and start all over again!

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Munchausen's Syndrome and Drug Dependence

Sir: London & Ghaffari (*Journal,* November 1986, **149,** 651–654) report on the failure of treatment in a patient with Munchausen's syndrome approached primarily as a problem of dependence on drugs. The demand for pethidine injections is as much a hallmark of this syndrome as is the assertion that the patient is allergic to contrast dyes. The authors state that their patient was in the Royal Army Medical Corps. This is a common assertion (O'Shea *et al.* 1982, 1984). It is rarely true in my experience.

The strongest theory to date of the origins of Munchausen's syndrome suggests that it is a trait learned in childhood: a 'Munchausen mother' induces fictitious diseases in her child; the child later assists the mother in fabricating such illnesses; the young adult goes out into the world seeking mothering with the only language he knows that will procure it – i.e. illness behaviour. Doctors respond with admission, investigations, and treatments, and reject when they find out that the patient has lied (Meadow, 1985; O'Shea, 1984). Most workers now accept that Munchausen patients fall somewhere within the spectrum of psychopathy, and in those cases of Munchausen's syndrome which appear to begin in adult life, scrutiny will nearly always reveal underlying psychopathy.

London & Ghaffari describe the family diseases of their patient in detail, and yet feel that the RAMC was responsible for his being "introduced" to a medical environment. Incidentally, even proxy cases may lie about their nursing experiences (Meadow, 1985).

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References

Meadow, R. (1985) Management of Munchausen by proxy. *Archives of Disease in Childhood,* **60,** 385–393.

O'Shea, B. (1984) More on patients with factitious illness. *American Journal of Psychiatry,* **141,** 140–141.

——, McGennis, A. & Cahill, M. (1984) McIlroy: some of the answers. *Irish Journal of Psychiatry,* **5,** 15–18.

——, ——, Lowe, N. F. & O'Rourke, M. H. (1982) Psychiatric evaluation of a Munchausen's syndrome. *Irish Medical Journal,* **75,** 200–202.

Autocastration and Biblical Delusions in Schizophrenia

Sir: I read with interest the paper by Waugh (*Journal,* November 1986, **149,** 656–659). I remember being called one Sunday to a surgical ward at the Queen Elizabeth Hospital, Woodville, South Australia, as the duty psychiatrist. The patient I was called to see had, the previous day, removed his own penis and genitalia, and also his tongue, with a scalpel. He had also attempted to perforate his ear drums, and there were superficial lacerations to his upper eyelids. It became clear later that he had also considered cutting off his right hand. He was about 30 years old, Chinese-Malaysian, and a medical practitioner.

I saw the patient twice on succeeding days, and he was able to communicate lucidly by means of pencil and paper. Like Waugh's patient, his thought content conveyed a sense of relief at having totally removed his genitals and also his tongue. His reasoning was that he could no longer do evil or speak it. He did not refer to Matthew 19:12, or any other biblical verse specifically, but he clearly felt that he had done what he had in order to atone for masturbation, lustful thoughts, and the use of obscene language. He felt that he had made his peace with the Almighty.

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Sir: I would like to suggest a name for the syndrome described by Waugh. 'Klingsor's syndrome' seems to be the obvious choice. Klingsor was a character in Wagner's *Parsifal* who unmanned himself in a vain effort to gain entry to the brotherhood of Grail Knights. The suggested title thus incorporates both the element of autocastration and the religious motive described by Waugh.

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Parasuicide and unemployment

SIR: I am unable to agree with Platt's concluding remarks (*Journal*, October 1986, **149**, 401–405) "we are faced with incontrovertible evidence... that the overall level of parasuicide could be reduced even more if there were a return to fuller employment." As he points out, we are not in any position to decide between the two types of explanation put forward by Hawton & Rose, i.e. the possibility of an indirect causal link between unemployment and parasuicide and the theory that the psychologically vulnerable may be more likely to become and remain unemployed. If the second theory proves to be correct it does not follow that a return to fuller employment would affect the levels of parasuicide.

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Multi-author papers

SIR: An article published some years ago describing the fatal course of an unusual illness was described by a reviewer as an instance of sixteen men on a dead man's chest. Shaw *et al* offer us nineteen men on a drug company chest (*Journal*, October 1986, **149**, 515–517). The distinction of the contributors is elevated by their unusual qualifications, e.g. MSCPsych. I, PLAB Exam, 1979 and DHO. No MRCPsych Pt I (Failed), but surely a Royal College Journal has a view on the use of these other distinctions? Should not the authors be only those who accept full responsibility for design of the study and intellectual content of the paper? On the latter point, can the conclusion "citalopram is an effective and acceptable antidepressant" be justified by the data presented? The numbers involved are not clearly stated, no significant differences in efficacy were found, and the confidence intervals were not stated.

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NOTE: 'MSCPsych' was a typographical error and should read 'MRCPsych'. *DHO* was italicised, which is editorial style to indicate a post held, in this case District Health Officer – Ed.

Attachment Dynamic in Adult Life

SIR: We agree with Heard & Lake (*Journal*, October 1986, **149**, 430–438) that attachment offers a valuable conceptual tool to identify relevant variables related to the characterisation of neuroses and personality disorders. Attachment, extensively investigated in ethological and developmental studies, focuses on explanatory concepts rather than mere descriptive generalisations. It is surprising, therefore, that DSM–III, Axis II, despite using relationships to others as a central feature of many personality disorders, has neglected the richness of attachment in the delineation of social relationships.

We can also agree that most adults establish 'preferred relationships' with others. We do not agree that all preferred relationships are attachment relationships. Clearly, all of this hinges on how attachment is defined. The authors state: "'Preferred relationships' refers to relationships in which individuals regularly expect to find opportunities for companionable and/or supportive interactions... People who are so classed constitute an individual's attachment network... The concept of preferred relationships in the attachment network circumvents difficulties in describing attachment relationships and affectional bonds in adults".

The implications of this model are that preferred relationships are synonymous with attachment relationships, and an individual's attachment network is simply a subset, defined by intensity and intimacy, of the social support or affiliative network. But this approach to attachment relationships assumed that (a) attachment can be characterised using the same criteria as affiliation (e.g. if affiliative relationships provide companionship, attachment relationships provide preferred or more salient companionship), and (b) attachment and affiliation serve the same functions – attachment, again, doing the job more and better. Both of these assumptions are problematic.

In accordance with the implications of Bowlby's (1969) theory, attachment is restricted to dyadic relationships in which proximity to a special other is sought or maintained to provide a sense of security. The principal function of adult attachment is protection from danger (as it is during childhood) although adults recognise other dangers to existence than those recognised by infants and children: specifically, threats to the individual's self-concept and integrity (Hinde, 1982; West *et al*, 1986). Affiliative relationships have a quite different function, serving to promote exploration and expansion of interests from the secure base provided by attachment.

In our view, Heard & Lake have confused similarity in mechanisms for maintaining attachment and affiliative systems with a congruence of the systems. Identifying the mechanisms used to promote and maintain social relationships is obviously important to the understanding of disorders involving these mechanisms. But differentiating the function and characteristics of particular types of social relationships is equally important for discriminating the variety of disorders. For example, an inability to experience intimacy is experientially and clinically different from an inability to experience security within an intimate relationship. The first we would term a disorder of the affiliative system; the second, of the attachment system. To attempt to 'circumvent' these distinctions hinders rather than aids conceptualisation in this field of research.

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References

BOWLBY, J. (1969) *Attachment and Loss; Attachment*. New York: Basic Books.

HINDE, R. A. (1982) Attachment: some conceptual and biological issues. In *The Place of Attainment in Human Behavior* (eds C. M. Parkes & J. Stevenson-Hinde). New York: Basic Books.

WEST, M., LIVESLEY, J., REIFFER, L. & SHELDON, A. (1986) The place of attachment in the life events model of stress and illness. *Canadian Journal of Psychiatry*, 31, 202–207.

Zinc in Senile Dementia

SIR: I wonder why Ken & Gibb (*Journal*, August 1986, **149**, 221–223) chose patients in their comparison group who were suffering from disorders in which low zinc levels have been reported. Srinivasan (1984) reviews psychiatric disorders in which low zinc levels occur: these include affective disorders, confusional states, and schizophrenia, all of which were included in the comparison group. The study would have been more valuable if a comparison had been made with a healthy, non-psychiatrically ill control group who were matched for age and sex with the dementia group, and it is possible that low zinc levels in dementia could then have been demonstrated.

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SRINIVASAN, D. P. (1984) Trace elements in psychiatric illness. *British Journal of Hospital Medicine*, 32, 77–79.

49 Chromosome Anomaly

SIR: We describe another patient with 49 XXXXY anomaly (*Journal*, February 1986, **148**, 209–210 and 210–212).

Case report: A male, aged 34, has been an in-patient for the last 22 years. He was born with the cord around his neck and was cyanosed at birth. His developmental milestones were delayed. He had recurrent chest infections as a child. His mother was 34 and his father was 39 years old at his birth, and he is the youngest of three siblings.

On physical examination, he is 5 ft ¾ ins in height and his head circumference is 52½ cm. He has a small face, with palpebral fissures slanted upwards and outwards. He is severely myopic, and has left-sided club foot. He has a narrow chest and does not have any facial or body hair, although he does have a few axillary and pubic hairs. His penis is small and his testes are undescended. He had phymosis at birth, which has been operated on in the past.

He is passive, friendly, and attention-seeking, and has a high-pitched voice. He likes music, discos, and parties and is also interested in swimming. He is fully ambulant and his self-help skills are good. He is able to read and write. His IQ was tested in 1981, when the WAIS score was 60. He had unexplained recurrent falls in 1983, and an EEG showed bilateral cortical dysfunction maximal over temporal regions. He had no more falls after he was started on carbamazepine.

It is interesting that despite 49 XXXXY anomaly and some perinatal damage this patient is functioning at the level of mild-to-moderate mental handicap.

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Post-ictal Syndrome after ECT

SIR: James & Simpson (*Journal*, September 1984, **145**, 337–338) invited comment on the observation of a second spontaneous fit after the second ECT given to a young woman suffering from a psychotic depressive illness associated with refusal to eat or drink.

Recently a similar observation was reported locally. A 24-year-old woman suffering a depressive illness had received a 12th right unilateral treatment. The treating doctor reported that about a minute after a tonic-clonic seizure she displayed further arm and leg movements lasting approximately 30 seconds, and concluded that she had undergone a second epileptic seizure.

By chance the young woman was taking part in a study, and thus a 4-channel EEG recording was made of her treatment by an independent observer. The recording showed a clear synchronous centrencephalic spike and wave episode lasting 54 seconds immediately after electrical stimulation. Thereafter the recording showed diffuse slow activity, often in dysrhythmic runs. Approximately 45 seconds later there was a return of muscle potentials on the record, and a period of small muscle twitches and movements were seen. However, there were no further concomitant cerebral epileptic events after the initial grand mal fit. The recording continued for 380 seconds after electrical stimulation.

Motor restlessness and uncoordination are recognised features of the post-ictal syndrome (Livingston *et al*, 1980). In the absence of any epileptic phenomena on the EEG this seems a more likely explanation than that she experienced a second seizure.

The difficulty of differentiating by observation alone the motor restlessness of a post-ictal syndrome from a second epileptic seizure may not, of course, necessarily explain the clinical phenomenon observed by James & Simpson. Certaintly, if spontaneous second seizures are to occur, they would be expected to occur early in treatment as the fit threshold rises during a course of ECT (Sackeim *et al*, 1986). If a second seizure occurs after ECT, it is important to consider possible precipitants such as drugs with convulsant properties, e.g. tricyclic antidepressants and phenothiazines. The woman in the reported case was drug-free, and thus we wondered about other precipitants such as hyperuraemia or menstruation (Hopkins, 1983).

The value of routine EEG monitoring of ECT is a matter of dispute, but it is unlikely that such clinical phenomena will be understood properly without simultaneous EEG monitoring.

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References

HOPKINS, A. (1983) In *Oxford Textbook of Medicine* (eds D. J. Weatherall, J. G. G. Ledingham & D. A. Warrell). Oxford: Oxford University Press.

LIVINGSTON, S., PAULI, L. L. & PRICE, I. (1980) In *Comprehensive Textbook of Psychiatry* (eds H. I. Kaplan, A. M. Freedman & B. J. Sadock). London: Williams & Wilkins.

SACKEIM, H. A., DECINA, P., PROHOUNIK, I. *et al* (1986) Dosage, seizure threshold, and the antidepressant efficacy of electroconvulsive therapy. In *Electroconvulsive Therapy* (eds S. Mehta & H. A. Sackeim). New York: New York Academy of Sciences.

Difficulties in Assessing Tardive Dyskinesia

SIR: Tardive dyskinesia (TD) is a major side-effect of long-term administration of neuroleptics. It is important therefore that its incidence, prevalence, and response to treatment are accurately determined. Such accuracy, however, is bedevilled by difficulties in assessment, and we report a further example of this difficulty.

As part of an (unpublished) open study of the efficacy of a novel psychotropic drug (Glaxo, GRC507/75) in the treatment of TD, ten chronic schizophrenic in-patients with TD were assessed before treatment using the Abnormal Involuntary Movements Scale (AIMS) (US Department of Health, Education and Welfare, 1976) and immediately afterwards were videotaped for one and a half minutes in a standardised way (Kidger *et al*, 1980). The live and video ratings were made by two different psychiatrists; the video rater was unaware that the ratings were before treatment.

As assessed by the AIMS global rating, six patients had mild and four moderate TD; two patients on video had no evidence of TD. The AIMS subscale found that all patients had orofacial and five distal dyskinesia, while the video ratings found only six with orofacial and four with distal dyskinesia.

Videotaping of patients with TD has obvious advantages, especially when carried out serially, as the order of presentation to the rater can be randomised. The present results suggest, however, that it may be less sensitive in mild and moderate cases than the more detailed assessment by AIMS of the 'live patient'. Indeed, our impression was that patients 'froze' in front of the camera. The main drawback to the AIMS is that it does not allow for a quantitative assessment of frequency, amplitude, or duration of movements (Barnes, 1984).

Agreement between live and video ratings was higher in two previous studies (Barnes & Trauer, 1982; Firth & Ardern, 1985). In the former study, however, the patients were unaware of the reasons for videotaping; in the latter, at least one movement was rated 'moderate-severe' in the Abbreviated Rockland Rating Scale. However, evaluation of methods of treatment must focus on patients with mild TD who give informed consent to the study.

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Barnes, T. R. E. (1984) Rating tardive dyskinesia. *British Journal of Psychiatry*, **145**, 338.

—— & Trauer, T. (1982) Reliability and validity of a tardive dyskinesia videotape rating technique. *British Journal of Psychiatry*, **140**, 508–515.

Firth, W. R. & Ardern, M. H. (1985) Measuring abnormal movement in tardive dyskinesia: a pilot study. *British Journal of Psychiatry*, **147**, 723–726.

Kidger, T., Barnes, T. R. E., Trauer, T. & Taylor, P. J. (1980) Subsyndromes of tardive dyskinesia. *Psychological Medicine*, **10**, 513–520.

US Department of Health, Welfare and Education (1976) Abnormal Involuntary Movements Scale (AIMS). In *ECDEU Assessment Manual* (ed. W. Guy). Rockville, Maryland: US Department of Health, Welfare and Education.

Premorbid Adjustment Scale as a Prognostic Predictor for Schizophrenia

It is difficult to formulate a medium or long-term prognosis following the onset of schizophrenia. Standard criteria such as those of type of onset (acute or insidious), predominant symptoms (Huber *et al*, 1980), or the diagnosis of subtype do not correlate satisfactorily with outcome (Bland *et al*, 1976). In the last decade some authors cited the importance of premorbid factors such as socio-familial relationship, adaptation to school, or pseudo-psychopathic or toxicophylic behaviour (Strauss & Carpenter, 1977; Wittenborn *et al*, 1977) in the outcome of these patients, with a marked relationship having been noted between these factors and personal autonomy in later years.

Cannon-Spoor *et al* (1982) recently developed the Premorbid Adjustment Scale (PAS) to evaluate aspects such as withdrawal, social relationships, independence, scholastic performance, and ability to establish socio-sexual relationships. All patients are rated on five sub-scales corresponding to childhood, early adolescence, late adolescence, adulthood, and general, with a total of 26 items. These authors suggested that a high score on this scale may detect patients likely to become chronically hospitalised or at high risk for readmission.

We studied a sample of 30 patients (11 in-patients, 19 out-patients) presenting schizophrenic disorders (DSM–III criteria) independent of subtypes (14 males and 16 females; mean age = 24.9; age range = 17–34). The rating scale was administered retrospectively in the majority of cases by five raters with the patient and his family separately (the minimum correlation between pairs of raters was $r=0.76$ ($P\leqslant 0.0001$)). The minimum duration of illness was 2 years (mean = 3.6). All were admitted to hospital at least once (mean = 3 admissions). Outcome was evaluated using Strauss & Carpenter's scale (1972), which covers duration of time spent outside hospital, social contacts, and time usefully employed over the last year, as well as absence of symptoms in the past month.

Correlation between the two scales, PAS average and Strauss, was statistically significant ($r=0.76$, $P\leqslant 0.001$ for the average score). Similar results were seen on comparing PAS sub-scales with Strauss & Carpenter's outcome scale: childhood, $r=0.49$ ($P\leqslant 0.01$); early adolescence, $r=0.69$ ($P\leqslant 0.001$); late adolescence, $r=0.61$ ($P\leqslant 0.001$); adult, $r=0.73$ ($P\leqslant 0.001$); general, $r=0.75$ ($P\leqslant 0.001$).

Ten patients had been rated as having an acute onset of less than three months, and 20 an insidious onset. Correlation between the two scales was not significant in the acute onset subgroup ($r=0.59$), while the insidious onset sub-group showed a significant correlation ($r=0.67$; $P\leqslant 0.001$).

PAS appears to be a valuable and useful aid both in daily clinical practice and for further studies investigating the factors related to schizophrenia and its outcome.

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References

Bland, R. C., Parker, J. H. & Orn, H. (1976) Prognosis in schizophrenia. *Archives of General Psychiatry*, **33**, 949–954.

Cannon-Spoor, H. E., Potkin, S. G. & Wyatt, R. J. (1982) Measurement of premorbid adjustment in chronic schizophrenia. *Schizophrenia Bulletin*, **8**, 470–484.

Huber, G., Gross, G., Schüttler, R. & Linz, M. (1980) Longitudinal studies of schizophrenic patients. *Schizophrenia Bulletin*, **6**, 592–605.

Strauss, J. S. & Carpenter, W. T. (1972) The prediction of outcome in schizophrenia. I. Characteristics of outcome. *Archives of General Psychiatry*, **27**, 739–746.

—— & —— (1977) Predictor of outcome in schizophrenia. III. Five-year outcome and its predictors. *Archives of General Psychiatry*, **34**, 159–163.

Wittenborn, J. R., McDonald, D. C. & Maurer, H. S. (1977) Persisting symptoms in schizophrenia predicted by background factors. *Archives of General Psychiatry*, **34**, 1057–1061.

Hyponatraemia Masquerading as Malignant Neuroleptic Syndrome

Sɪʀ: We wish to report the case of a patient suffering from acute hyponatraemia with water overload presenting as malignant neuroleptic syndrome.

Case report: A 27-year-old male weighing 76 kg was admitted comatose with tremor, rigidity, pyrexia of 39°C, sinus tachycardia of 140/min, blood pressure 190/80 mmHg, urinary incontinence, and a right extensor plantar response within one day of receiving the eleventh fortnightly dose of clopenthixol decanoate (300 mg by injection) for schizophrenia. Malignant neuroleptic syndrome was tentatively diagnosed.

Laboratory investigations on admission were: white cell count 22.4 × 10/litre (85% neutrophils); Na 116 mmol/litre; measured plasma osmolality 234 mOsm/kg; urea 3.3 mmol/litre and the osmolality of the 2.2 litres of urinary output in the first 4 hours of admission was 108 mOsm/kg. Creatine kinase was 873 i.u./litre (normal range <70), and bilirubin 33 µmol/litre (normal range <17). Blood glucose and CSF examination was normal. A trace of salicylate only was found on screening for other drugs, and lithium was excluded.

Benztropine mesylate (2 mg) and normal saline (1 litre) were commenced. A rapid recovery ensued in the second 12 hours following admission, in association with a urine output of 7.2 litres in the first 24 hours. By the second day a mild degree of Parkinsonism was the only remaining sign, and the white cell count was 11.1 × 10/litre, Na 136 mmol/litre and bilirubin 21 µmol/litre.

The severe hyponatraemia with a calculated excess body water of 7.8 litres and the acute polyuria indicated that primary acute water intoxication was the key. The speedy recovery confirmed this. SIADH induced by a depot drug would have a much longer duration of effect. The raised total CK, pyrexia, and bilirubinaemia resulted from skeletal muscle hyperactivity, with some haemolysis and drug-induced cholestasis probably contributing to the bilirubinaemia. Pyrexia and varying neurological signs are features of acute severe water intoxication (Arieff & Guisado, 1976). Neutrophilia – almost certainly due to stress-related endogenous corticosteroids – pyrexia and autonomic instability are also features of the malignant neuroleptic syndrome (Abbott & Loizou, 1986). Thus plasma sodium is an important investigation in the diagnosis of malignant neuroleptic syndrome.

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References

Aʙʙᴏᴛᴛ, R. J. & Lᴏɪᴢᴏᴜ, L. A. (1986) Neuroleptic malignant syndrome. *British Journal of Psychiatry*, **148**, 47–51.

Aʀɪᴇғғ, A. I. & Gᴜɪsᴀᴅᴏ, R. (1976) Effects on the central nervous system of hypernatremic and hyponatremic states. *Kidney International*, **10**, 104–116.

AIDS-Phobia

Sɪʀ: Five male patients presented to us with psychiatric symptoms associated with the fear that they had AIDS. They were tested by the enzyme-linked immunosorbent assay technique and found to have no antibody to the causative virus infection of AIDS. The common features included: an anxious neurotic premorbid personality; tendency to have low extraversion and high neuroticism scores on the Eysenck Personality Questionnaire; a history of promiscuous heterosexual contact, and symptoms and signs of an anxiety state.

Reports from Western countries have documented a similar condition, under the name 'AIDS-panic' or 'pseudo-AIDS', with obsessive and paranoid features in the premorbid personality (Schwartz, 1983; O'Brien & Hassanyeh, 1985), and a history of homosexual contact and clinical features of anxiety or depression (O'Brien & Hassanyeh, 1985; Miller *et al*, 1985). There are similarities and differences in these cross-cultural reports. Neurotic individuals appear to be more prone to develop AIDS-phobia in India than in the West. Both groups show neurotic reactions as a response to the fear. In the West a history of male homosexual exposure is usually present, as it is the predominant risk factor for AIDS. In India the infection has been found mainly among women prostitutes (Simoes *et al*, 1987), and the public have been warned of the danger of promiscuous heterosexual contact. Although the terms 'AIDS-panic' and 'pseudo-AIDS' have been used for this condition, 'AIDS-phobia' may be a better name, since it is similar to venerophobia.

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References

Mɪʟʟᴇʀ, D., Gʀᴇᴇɴ, J., Fᴀʀᴍᴇʀ, R. & Cᴀʀʀᴏʟʟ, G. (1985) A 'pseudo-AIDS' syndrome following from fear of AIDS. *British Journal of Psychiatry*, **146**, 550–551.

O'Bʀɪᴇɴ, G. & Hᴀssᴀɴʏᴇʜ, F. (1985) AIDS-panic: AIDS induced psychogenic states. *British Journal of Psychiatry*, **147**, 91.

Sᴄʜᴡᴀʀᴛᴢ, R. (1983) AIDS panic. *Psychiatry News*, August 17.

SIMOES, E. A. F., BABU, P. G., JOHN, T. J. *et al* (1987) Evidence for HTLV-III infection among prostitutes in Tamil Nadu. *Indian Journal of Medical Research* (in press).

Art Therapy

SIR: In his review of the book *Art Therapy for Groups* by M. Liebmann (*Journal*, December 1986, **149**, 805), Aveline, in commenting on group dynamics, states: "This imbalance is worrying and reflects a deficiency in the training of art therapists". In fact, much emphasis is placed on group dynamics in the three postgraduate Diplomas in Art Therapy: at Hertfordshire College of Art, St Albans; Sheffield University; and Goldsmiths' College, University of London. I can speak more specifically about the Goldsmiths' course, where five out of eight of the tutors are qualified group psychotherapists as well as art therapists and the other three have extensive experience of group dynamics. Our course is structured around a model of art therapy combined with group analysis, and the students participate in weekly experimental groups, including one focusing solely on group dynamics. We do not neglect individual therapy, but are known for our 'group bias'.

The profession of art therapy is fairly new and is an easy target for misconceptions concerning training and practice. Full information about the courses can be obtained from each of the three centres, and general information on art therapy can be obtained from the British Association of Art Therapy, 13c Northwood Road, London N6.

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SIR: May I comment on Aveline's review of Liebman's book *Art Therapy for Groups: The Handbook of Games and Exercises*. While his criticism of the book's meagre psychodynamic content may be fair comment in the context of the powerful feelings that can be aroused by some of the exercises described, it is misleading to say that this a reflection of the "deficiency in the formal training of art therapists". On the contrary, the Postgraduate Diploma course in Art Therapy at Goldsmiths' College is firmly and extensively based on group dynamics, both theoretical and experiential.

The techniques described in the book demonstrate one approach to art therapy, which can perhaps also be used by other professionals wishing to use art in their work with patients. However, I think it is true to say that qualified art therapists have a deep concern and understanding of the emotional forces which may surface through the use of art, and are fully aware of the implications of individual and group dynamics.

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A HUNDRED YEARS AGO

Lunacy and Pauperism

At a recent Poor Law Conference in Glasgow, Dr Yellowlees read a highly interesting paper on the above theme. He urged that every county or district should have two types of asylums for its pauper insane; one a hospital, fully equipped with the best means of treatment, and receiving all new cases, the size not exceeding 300. Another building, erected at half the cost, should be devoted to chronic cases only, and should receive no patients except from the cure-asylum.

Reference

Journal of Mental Science (July 1884) **30**, 335.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

British Journal of Psychiatry (1987), **150**, 414–423

Book Reviews

Editor: Sidney Crown

The Natural History of Psychiatric Disorder in Children (Maudsley Monographs 29, Institute of Psychiatry). By H. Zeitlin. Oxford: Oxford University Press. 1986. Pp 158. £15.00.

This important book, as its subtitle (*A study of individuals known to have attended both child and adult psychiatric departments of the same hospital*) makes clear, contributes more to the delineation of syndromes than to the prognosis of child psychiatric disorders. The study is based on case records, and the index group consists of 161 adult psychiatric patients who as children had attended the same psychiatric hospital. They were compared with matched groups of adult patients without evidence of childhood disorders, and of child patients not known to have attended psychiatric hospitals as adults. The work has been meticulously done but, because of the nature of the samples, the meaning of the many links established between child and adult pathology is often not at once apparent. This may account for some obscurities in the writing.

The results are a treasure trove of fascinating relationships: for example, the greater continuities of symptoms than of syndromes; the striking continuity from childhood to adult life of obsessional symptoms, greatly over-represented in this index group; and the somewhat less marked continuity of phobias. Most adult depressive neuroses arise *de novo* and most depressed children appear to outgrow their symptoms. However, the greatest continuity of depressive symptomatology was for a specially constructed syndrome based on Pearce's criteria (*Journal of the Royal Society of Medicine*, **71**, 494–500). Depressive mood was an essential component, suggesting that there is no such thing as 'masked depression' in childhood. This reviewer was especially interested in the childhood precursors of schizophrenia: 4 out of 14 adult schizophrenic patients had been given this diagnosis as children; 8, not psychotic in childhood, had presented with conduct disorders with or without emotional and developmental disorders. Two specific features characterised these children: peer isolation and the clinicians' recording of 'incongruous' or unpredictably odd behaviour.

Psychiatric referral in adult life cannot be the only criterion of poor outcome, and disturbed children who later appear at psychiatric hospitals are by no means representative of child psychiatric referrals. For a complete picture of the natural history of child psychiatric disorders we need a replication of Lee Robins' personal follow-up into adult life (*Deviant Children Grown-up*. Williams & Wilkins, 1966), this time of a group of referred children comprising the full diagnostic range, so that we can be as confident about the fate of emotionally disturbed and developmentally delayed children as we are about conduct disordered and delinquent youngsters. Meanwhile, this monograph is an essential purchase for all general and child psychiatry libraries.

Sula Wolff, *Honorary Fellow, Department of Psychiatry, University of Edinburgh*

Education and Training in Psychiatry: A Case Study in the Continuity of Medical Education. Edited by Henry Walton. London: King Edward's Hospital Fund, Oxford University Press. 1986. Pp 231. £12.00.

This book derives from a conference jointly sponsored by the Royal College of Psychiatrists, the Association of University Teachers of Psychiatry, and the now dissolved Association of Psychiatrists in Training. This was held in Cambridge in 1982, and a post-conference meeting was held 18 months later. Fourteen working parties prepared documents for the Cambridge conference, while rapporteurs presented a summary of the discussions on each document.

All this would appear to be a recipe for fragmentation, inconsistency, variable standards of presentation, and dubious conclusions. It is greatly to the credit of the editor that he has brought together a single, cohesive, and up-to-date report on a range of issues to interest what are effectively the recipients or consumers of psychiatric education. The book exemplifies the fact that education is not a short-term process contributing to a basic degree in medicine, nor even a longer-term specialist training, preceding appointment to a consultant post or its equivalent in a psychiatric field. It is a continuing process in which the participants change roles at different stages of the proceedings.

At one moment the young undergraduate, thought to be naïve and undifferentiated, is receiving the words of wisdom, but the next moment that same student is challenging the traditional views held by the teacher, causing thought and interaction between them. The same process continues through general training, specialist training, and refresher courses. Indeed, as exemplified by the title, training is only one aspect of the whole, which is a process of education throughout one's professional career.

The various chapters of the book cover specific educational topics such as teaching psychiatry to medical students, during general professional training, and in higher psychiatric training. There is discussion about what the non-psychiatric doctor needs to know. Specific knowledge about psychiatric syndromes and modes of treatment are obviously part of the basic knowledge, but there are specific psychiatric skills and attitudes best summed up under the general term 'psychological mindedness', which is of general application but on which attention can often be easily focused during the psychiatric clerkship.

Unfortunately, medical students tend to respond to the segmented medical course by segmented thinking. A medical student who showed great interest in psychiatry surprised me by referring to "supratentorial problems" when I was asked to see a patient on a neurology firm. It was made very clear that when doing neurology one thought as a neurologist and abandoned all psychiatric pretensions. As a psychiatric teacher, it gave me pause for thought. It is to be hoped that general professional training can produce a more integrated conceptualisation to the fragmented undergraduate medical training and that this psychological mindedness, already inculcated, can be spread across medical thinking.

Attention is drawn to the lack of clear educational objectives in the field of postgraduate psychiatric training. Objectives and goals are listed and form a useful basis for individual training programmes. Arising out of this list of goals comes a need for some form of assessment. This implies an evaluation by the teachers separate from any formal national examination. The Royal College of Psychiatrists has been very clear that training programmes should form the basis of an adequate education, rather than the acquisition of a diploma which is a measure of the amount of knowledge possessed by an individual over a period of a week or two; such examinations are an inadequate measure of overall qualities, although they may still be the best available in practical terms.

In the section devoted to training settings and, in particular, to liaison psychiatry, attention is again focused on the contribution by the psychiatrist to areas outside the traditional psychiatric hospital, and even the psychiatric unit in the general hospital. It is important to get the balance between the various sites at which psychiatric expertise may be offered. A residual stigma is often felt to attach to the big psychiatric hospital, in spite of occasional changes of name. Wards in general hospitals have also suffered a similar tarnish. For their own benefit, and for the benefit of patients, there is a move to develop Mental Health Services in the community, staffed by non-psychiatrists who obviate the disadvantages of a formal psychiatric service. It remains to be seen whether such manoeuvres overcome the problems and provide a separate and different service from the psychiatric service which continues its medical links, and whether duplication is avoided and two services can be financed. It is to practise in such a changing world that the trainee of today is being educated.

Professor Walton is to be congratulated on producing a neat, comprehensive review of present-day issues, well laid out in short chapters and summarised at the end in a series of emphases. It is a book which should be read by all interested in training in psychiatry, and it is difficult to think of any group of psychiatrists excluded from that description and, indeed, relatively few doctors practising in medicine.

C. P. SEAGER, *Professor of Psychiatry, University of Sheffield*

Advances in Adolescent Mental Health. Vol. 1. Mental Health Disorders in Adolescence. Edited by RONALD A. FELDMAN and ARLENE RUBIN STIFFMAN. London: Jai Press. 1986. In two parts: Part A (pp 283) and Part B (pp 262). £85.00 (Parts A and B).

These two volumes from the Centre for Adolescent Mental Health, Washington University, St Louis (which was set up in 1983) promise to be the first in an annual series of books entitled 'Advances in Adolescent Mental Health'. The editors, who are respectively the Director of the Centre and an Instructor in Social Work in Psychiatry at Washington University, claim that their aim is to report and synthesise pertinent research about all aspects of adolescent mental health, thus attempting to correct earlier inadequate dissemination of knowledge in this field.

A total of twelve completely distinct chapters cover a mixture of clinical syndromes, such as depression in adolescence, anorexia nervosa, and attention deficit disorder in adolescence, and such psychosocial issues as adolescent heterosexual behaviour, cigarette use, alcohol abuse, and suicide, homicide, and accident trends. At first sight this seems a rather random and odd range of topics, not employing any obvious overall system, but this is explained by the editors as a deliberate attempt to tackle the most topical and anxiety-provoking problems presented by this age group first.

The 27 contributors represent very varied disciplines, including paediatrics, psychology, social work, education, and sociology. Only four chapters are from departments of psychiatry. In their introductory chapter, the editors explain that they were deliberately seeking this sort of breadth of coverage but recognise that in doing so they risk foregoing intradisciplinary depth regarding any particular problem. This is all too unfortunately true and is one of my criticisms of the books. Inevitably they vary considerably, but I found the chapters on clinical syndromes disappointingly superficial and coverage of treatment approaches particularly inadequate. However, two chapters which I appreciated, particularly because they tackle subjects not commonly referred to in other books, were those on loneliness, shyness, and withdrawal in adolescence (a familiar enough problem) by Eric Ostrov, a forensic psychologist, and gender role problems by another team of clinical psychologists. Conversely, that on attention deficit disorder, again by a clinical psychologist, reads almost like a foreign language to a British child psychiatrist not in sympathy with the American approach to this syndrome.

Each chapter has a similar format, which is basically that of a literature review, and the bibliography for each runs to seven or eight pages, sometimes more, including 1984 references. This in my view represents the best value of the books: as an up-to-date source of relevant references.

My strongest criticism has to be of the cost of these volumes. One can understand why they are so expensive: they are beautifully produced, and cloth bound. However, with smaller print and thinner paper the contents could comfortably occupy a single volume, and I really do not understand why it was felt necessary to create two. It is difficult to envisage many individuals paying this sort of sum, and few professional libraries in these economically stringent times are likely to feel they can afford to. In terms of value for money, there is absolutely no comparison with the child and adolescent psychiatrists' "bible" edited by Rutter and Hersov.

PATRICIA AINSWORTH, *Lecturer/Consultant in Child and Adolescent Psychiatry, Prestwich Hospital, Manchester*

GABA and Mood Disorders: Experimental and Clinical Research (LERS Monograph Series, Vol. 4). Edited by G. BARTHOLINI, K. G. LLOYD and P. L. MORSELLI. New York: Raven Press. 1986. Pp 222. $32.50.

This book presents the proceedings of a symposium held in Paris in 1984. A substantial number of the papers describe efficacy studies of drugs synthesised by the company which has sponsored this series of monographs. The presentation is uneven, and there is little sign of editing, so that overlap and repetition are common. A significant number of the chapters contain pronouncements with no reference, or with a reference to another chapter of the book (often written by a co-worker at Synthélabo).

The book launches into the complex neuropharmacology of GABA agonists, but interpretation is hampered by the lack of explanatory chapters on the anatomy and physiology of the GABA system in the CNS. The specificity and selectivity of GABA agonists to the GABA system is not discussed. Given the fact that GABA is a major inhibitory neurotransmitter for at least one-third of CNS synapses, it is striking what little clinical effect GABA agonists have in normal subjects. It is likely that their lack of effect relates to powerful local inactivation processes for GABA in the CNS, but this important issue is not tackled. It is suggested that GABA agonists may produce effects only when GABA is pathologically altered, but the evidence put forward that this is the case in affective illness is unconvincing.

However, important messages are contained in this volume. It becomes clear that manipulation of the GABA system can modulate a wide range of other neurotransmitters and their function. Moreover, well-known physiological and pharmacological changes in other transmitters and their receptors can be markedly altered by drugs acting on the GABA system. Interesting evidence is presented that the antidepressant effect of 'classical' antidepressants and ECT relates to changes in GABA function and GABA receptors. The antidepressant efficacy of GABA agonists appears to be equivalent to conventional antidepressants, and the clinical effects seem to be rapid and associated with few side-effects. An elegant chapter on carbamazepine by Post is included, but the evidence that this drug's interesting clinical effects are mediated by GABA was underwhelming.

On balance, the negative aspects of this volume outweigh the positive. It appears to be aimed at neuropharmacologists and biological psychiatrists, but misses both targets and will satisfy neither group. The best parts of the book have appeared or will appear in papers in refereed journals.

I. N. FERRIER, *MRC Clinical Scientist and Honorary Consultant Psychiatrist, MRC Neuroendocrinology Unit, Newcastle upon Tyne*

The Biology of Depression. Edited by J. F. W. DEAKIN. London: The Royal College of Psychiatrists. 1986. Pp 224. £7.50.

Twenty years ago Coppen wrote a lengthy review entitled 'The biochemistry of affective disorders', in which he classified the possible abnormalities underlying these disorders under amine, electrolyte, and endocrine changes. There has been remarkably little conceptual change since then and although Coppen's 1967 review is not quoted by any of the authors in this collection of 11 papers, Schildkraut's catecholamine hypothesis (1965) is cited by six of them. This compact little book derives from the proceedings of a one-day meeting of the Biological Group of the Royal College of Psychiatrists, held at Manchester University in 1985, and provides a valuable update on the present stage of British research into depressive illness.

Deakin and Crow set the scene with a comprehensive but concise review of monoamine neuropharmacology, concluding with yet another reformulation of the catecholamine hypothesis: "depression results from hypoactivity of catecholaminergic reward systems, and anxiety from hyperactivity of serotoninergic punishment systems, the two systems interacting with each other such that anxiety and depression represent separate but correlated dimensions of disturbance". In spite of its ingenuity, however, one can't help wondering how testable this is, and the philosophical problem of whether any identified biological changes are primary or secondary remains. Three of the subsequent papers deal with amine receptors, three with neuroendocrinology, and one with electrolyte changes. Additional papers cover genetic and physiological studies, and Paykel provides an authoritative appraisal of current antidepressant treatments and the management of resistent depression. The studies of receptor binding sites in post-mortem brain, platelets, and rat brain have yielded largely inconsistent or negative findings. However, the growing link with neuroendocrinology looks promising, particularly clonidine-stimulated growth hormone release and tryptophan-stimulated prolactin release as *in vivo* indicators of central α_2 adrenergic and 5HT receptor function respectively.

Evidently there is still a long way to go before we can really talk about 'the biology of depression'. Nevertheless, the editor is to be congratulated on the production of a most useful book which, apart from being very good value for money, should be essential reading for anyone planning clinical research in depression and highly recommended for psychiatric trainees preparing for the MRCPsych.

DAVID J. KING, *Reader in Psychopharmacology, The Queen's University of Belfast*

Principles into Practice: A Developmental Study of a Community Mental Health Service. By AILEEN MCDERMOTT. Manchester: Manchester Free Press. 1986. Pp. 69. £3.00.

This little booklet is simplicity itself and in being so it speaks directly to the professional. It says he must improve accessibility, informality, be people-(client)-centred, and encourage consumer involvement and integration of services. The account given is of a development in the centre of Manchester of a service for young people. It says that girls especially would have been helped if it had been in local neighbourhoods. It extols the service using cafes, pubs, etc., to help people feel at ease and says that, despite even that, some still fear approaching any services, let alone psychiatric clinics with their professionalism and bureaucracy.

The contribution of volunteers and the conflicts between them and "workers" is discussed. The introverts who need peace are mentioned, as is the fact that they need defending from the over-intrusive. The pleasantness and value of using ordinary meeting places together is mentioned. There is some criticism of structural programmes into which patients are to be fitted, and more emphasis is required on "Being" rather than "Doing", we are told. There must in addition, it is insisted, be little division in our approach to people classified as copers or non-copers: we all need respect, friendship, time, and security. Going for a walk, cooking a meal, having a drink, visiting the cinema, etc. together is worthwhile, whether or not intensely personal questions are raised. In an under-employed society surely there is time for this, and maybe it is as cost-effective as "treatment". The technical language of professional groups produces to some extent their cases – its avoidance goes some way to helping people.

The short time this sensible work takes to read is as rewarding as it is thought-provoking and refreshing. We are clearly helped to ask whether we do need to remain so respectable and pompous.

F. A. JENNER, *Professor, University Department of Psychiatry, Sheffield*

Psychopharmacology of Sexual Disorders. Edited by MARK SEGAL. London: John Libbey. 1985. Pp. 171. Price £26.00.

This is a short book containing contributions from 29 authors from eight different nations whose main objective is to increase awareness of possible psychopharmacological components in human sexual dysfunction.

It is divided into two sections, Basic Science and Clinical Science, is very readable and fulfils its objectives. The Basic Science section gives an outline of the animal experimental work being done in this area. The second section looks at what is known at a clinical level in the psychopharmacology of psychosexual disorders. In the final analysis this does more to highlight what is not known rather than what is known.

The book makes two important contributions to this field. It stresses the fact that thirty, rather than ten percent of patients who present with sexual dysfunction will have a physical or organic cause for it and emphasises the heterogeneous nature of sexual disorders with a novel form of classification in Chapter 10. This classification is based on ethology rather than the more traditional symptom orientated method.

The biggest fault of the book is its attempt to cover too many topics in too short a space. This, however, gives the authors the opportunity to put forward many useful suggestions in the area of research into sexual disorders. This is probably its greatest forte and makes it highly recommendable to workers in the field.

ETHNA C. O' GORMAN, *Consultant Psychiatrist and Senior Lecturer, Queen's University of Belfast*

Handbook of Psychiatric Diagnostic Procedures. Vol. II. By RICHARD C. W. HALL and THOMAS P. BERESFORD. Lancaster, Lancs: MTP Press. 1985. Pp. 359. £39.50.

This book opens with an excellent account of clinical EEG, condensing the conceptual and methodological issues, the findings, the diagnostic yield, and the limitations of EEG into a succinct, readable chapter. The application of EEG to sleep studies is equally comprehensive but the parasomnias are dealt with dogmatically, while ignoring recent advances in child psychiatry.

Further chapters consider the identification of melancholia with sleep studies and the shortened REM latency found in endogenous depression.

Darwin's neglected work on facial emotions has only recently been revived. Facial electromyography during states of affective imagery is one demonstrative legacy which this handbook addresses in a chapter on psychomotor function. It has exciting implications for diagnosis, prognosis, and social communication. Two chapters cover evoked potentials (EP) and brain spiking; there is one on CT scanning. Between descriptions of the structural changes in syndromes and indications for CT is a rather disappointing section on CT scan research.

I am not sure how many psychiatrists would read the second half of this book, which is devoted to the laboratory evaluation of anorexia, alcoholism, drug abuse, sexuality, geriatrics, and dementia. There is a poor chapter on the diagnosis of dementia, both clinically and psychometrically.

In a chapter on the paraphilias, a series of patients is presented of whom three-quarters (mainly paedophiles) had CT scan or chromosal abnormalities or elevated testosterone levels. The discussion bypasses selection factors and issues of validity, and typifies a conceptual narrow-mindedness evident in the book.

Three future directions are singled out: computerised practice, PET scanning (a good introduction) and the endorphins – three techniques "likely to be available in the near future". A handbook for the library, notable for its survey of electrophysiology.

ROBIN JACOBSON, *Lecturer, Institute of Psychiatry, London*

The Psychiatric Emergency: Its Recognition and Management. Edited by EUGENE O'NEIL. New York: The Haworth Press. 1986. Pp. 128. $24.95.

Of all the emergencies that may occur in general practice and within hospitals, the ones that generate most anxiety seem to be psychiatric. Any book which helps explain the problems and as a consequence diffuse the anxiety must be welcome. This small book, the proceedings of a conference, does not pretend to be exhaustive but has a series of lectures of considerable value. I particularly like the description of the work of the social worker as an initial assessor in a hospital emergency room and the way in which the authors tactfully coped with the doctor/PSW relationship. The lecture on drug emergencies is a concise and clear description of this large area of practice although the need to be over-inclusive results in a failure to stress the common presentations. The fact that manic patients may require much higher doses of psychotropic drugs should be corrected and the brief, almost naive chapter on affective disorders is in strict contrast to the more obsessional classifications in a chapter on Alzheimer's disease. The final chapter on alternatives to hospital in-patient care is of limited use to British practitioners and stresses the need for a broader survey of the problems that present urgently. If anything it emphasises the frustration of doctors and patients alike in mobilising alternatives to in-patient care. The book is useful for family doctors and social workers as well as hospital practitioners. It could be recommended more if the scope were expanded and the price less.

BERNARD ADAMS, *Consultant Psychiatrist, University College Hospital, London*

Recent Advances in Psychogeriatrics – I. Edited by TOM ARIE. Edinburgh: Churchill Livingstone. 1985. Pp. 235. £25.00.

How much psychogeriatrics is advancing depends on how one defines an advance. Interest has grown, even in academic departments, and books have multiplied disproportionately. There has been much repetition and much that is of indifferent quality. This book, however, is a useful and up-to-date compendium of reviews of many of the fields in Old Age Psychiatry; the topics are relevant and the literature surveyed contemporary.

Chapters deal with structural regeneration in the brain; epidemiology; survival in dementia; the genetics of dementia; the heterogeneity of Alzheimer's disease; the reversible dementias; quality of care in institutions; affective disorders in old age; measurement; new radiological techniques; international comparisons; education; and WHO programmes.

Many of the contributors have made significant advances in research and in practice and they write with authority. Each chapter has an extensive bibliography and the references, some going up to 1985, are abundant and wide-ranging.

LOÏC HEMSI, *Consultant Psychiatrist, St George's Hospital, London*

Law, Psychiatry and Morality: Essays and Analysis. By ALAN A. STONE. Washington, DC: American Psychiatric Association. 1986. Pp 277. £12.95 pb.

Dr Stone, a Professor of Law and Psychiatry at Harvard University, is an example of an exceedingly useful academic hybrid, unknown in this country but by no means exceptional in the USA. Because of his legal expertise Dr Stone is accustomed to the casuistry and double-talk of lawyers, and, when the necessity arises, can play them at their own game. This facility makes his book all the more valuable to forensic psychiatrists, untutored in the law, who are accustomed to call a spade a spade rather than a triple-edged offensive weapon.

Although I found the whole of this book eminently readable there are chapters or essays of outstanding importance, such as, for example, Chapter I: Political Misuse of Psychiatry: A Tale of Two Generals. In it Dr Stone compares the fate of two army generals, Petro Grigorenko of the Soviet Union and Edwin Walker of the United States, both charged with crimes against the state. As Dr Stone explains, "It should be emphasised that both generals' 'fixed ideas' attacked the most delicate political question of their nation, the corruption of the Soviet Revolution and the racism of the world's greatest democracy". It could be equally emphasised that also on trial was the integrity of the psychiatrists, or indeed of psychiatry itself, in their individual countries. Did the psychiatrists involved succumb to the temptation of becoming political tools of the Politburo on the one hand or the Kennedy administration on the other?

Dr Stone examines the evidence with scrupulous fairness. His conclusion from a legal standpoint is that, "It is the American legal system's constitutional safeguards which protected General Walker. It is the absence of such safeguards that led to the 'psychiatric terrorism' in General Grigorenko's case". And from the standpoint of psychiatry he makes a fervent plea that "psychiatrists should bend over backwards to avoid complicity, and the danger of becoming over-zealous lackeys".

Of comparable importance is Chapter IV: The Trial of John Hinkley. Hinkley, it will be remembered, attempted the assassination of President Reagen. His trial and the verdict, not guilty by reason of insanity, rocked public confidence in American psychiatry and in the American criminal justice system, although it seems clear that, as the law stood, the jury reached a just decision. Nevertheless, it was felt by the public at large that, due to the machinations of the psychiatrists, the would-be assassin of a popular President had got away with it. It would not be idle to compare Hinkley's trial with that of Sutcliffe, the Yorkshire Ripper, in 1981. It is exceedingly doubtful if the jury on this occasion reached a just decision, but the public's anxiety and lust for revenge was assuaged, and it was agreed, except by the majority of psychiatrists, that Sutcliffe had got his just deserts.

Dr Stone ends this chapter with a grim warning. He writes, "Psychiatry is held hostage by the psychiatrists who testify in courts whatever their standards and whatever the test of insanity may be... the reputation and credibility of our profession is in their hands...".

In chapters IV–VII Dr Stone paints a terrifying picture of the legal minefield in which mental health workers at all levels have to pick their way. So perilous has the situation become that, at least in the public mental hospital sector, to treat or not to treat has become a legal decision and not a psychiatric one. We may well ask how near we have come to matching this state of affairs in this country, and how much nearer will we come if certain of our civil libertarians continue to increase their influence on mental health legislation.

If I hesitate to recommend this book wholeheartedly it is only because the hazards of the practice of psychiatry are so painfully portrayed and so off-putting that many potential entrants to the field might adhere to the advice of an American Appeal Judge, Judge Aldisert: "If you are contemplating a position as an attendant in a mental hospital, seek another job!"

HENRY R. ROLLIN, *Emeritus Consultant, Horton Hospital, Epsom*

The Diagnosis and Treatment of Drug and Alcohol Abuse. Edited by SIDNEY COHEN and JAMES F. CALLAHAN. New York: The Haworth Press. 1986. Pp 305. $39.95.

This volume brings up to date previous monographs issued by the United States National Institute on Drug Abuse. It is a guide to practical issues in the recognition, assessment, and management of drug and alcohol abusers. It is designed for nurses and doctors and is very medically oriented, but could be read by psychologists, social workers, and lay counsellors wishing to understand the practical and medical aspects of managing these conditions.

It is clearly laid out, well indexed, and has a strong cover, a clear large typeface, and durable paper. It would survive well and be useful in a busy clinic or ward.

It is richer and fills more of a gap when dealing with drug abusers than with alcohol abusers, although it is unfortunate that in a book published in 1986 there was not more on cocaine and on the HTLV-III virus. Methadone maintenance treatment (which the Federal Drug Administration permits if an abuser has been dependent on heroin for two years) is treated favourably, although, given the format of the book, not debated in detail. There is a guide to the use of clonidine in opiate withdrawal. The future potential of naltrexone, the long-acting opiate antagonist, in preventing relapse is touched on: so long as naltrexone is taken regularly, the euphoric effect of opiates is blocked. Overdosage of commonly abused drugs is usefully dealt with.

The alcoholism sections are useful, but are not as rich in detail as the drug abuse sections. Whereas all manner of skin lesions and disorders are described in drug abusers, the injected conjunctivae and excessive skin capillarisation of the alcoholic are not mentioned. Hypertension is erroneously said not to occur in alcohol withdrawal.

The last 61 pages detail training resources in the United States, with names and addresses of trainers and lists of training materials. This is of little or no relevance to readers outside North America. There are other sections which make the book unlike a standard text, but which are more useful. Each part of the book is preceded by a true/false or MSQ set of questions, and you are invited to test yourself again after reading each section. This is a helpful aid to study. The model assessment interview is detailed and would well provide a basis for a clinic wishing to devise its own assessment schedule. It is suggested that the counsellor records subjective data: (a) "does he like the client?"; (b) "does the client scare him?"; (c) "is the client a hustler?"; (d) "is he a nice person?". As this might suggest, the level of psychological sophistication in the book is not high. Some important psychosocial aspects of drug and alcohol abuse are not mentioned at all, such as marital and sexual problems and the needs of the family.

Nevertheless, for the trainees in a Drug Abuse clinic, or at a Casualty Department or Infectious Diseases unit where drug abusers are frequent attenders, this would be a useful introduction. The well-endowed Department of Psychiatry Library could also consider buying a copy if it is lacking a textbook on the topic.

JONATHAN CHICK, *Consultant Psychiatrist, Royal Edinburgh Hospital*

Science and Service in Mental Retardation. Edited by JOSEPH M. BERG. London: Methuen. 1986. Pp 475. £45.00.

The International Association For The Scientific Study of Mental Deficiency was founded in 1964 and, among many other activities, has promoted a conference every three years. This book, produced commendably quickly, contains part of the proceedings of the conference co-sponsored by the World Health Organization and held in New Delhi in 1985. This venue provided the opportunity for the conference to relate current knowledge and practice to the services in the Third World. The Association commends the multidisciplinary approach and attempts to bring together research and practice in the vast area of primary prevention, ranging from biological studies, through the characteristics and needs of the handicapped person, to service development in all parts of the world.

The contents of the book have been sensibly restricted to 47 out of some 200 papers presented at the conference. The result is a range of interesting papers covering epidemiology, clinical and biological studies, behavioural and psychiatric aspects, educational considerations, preventive and therapeutic interventions, retardation in adulthood, and perspectives in developing countries. The editors are to be congratulated on the balance of the papers chosen, which demonstrates the growing points of the specialty. While biological research aimed at disease identification and primary prevention must continue, there is no doubt that people with a mental handicap will always be with us. There is evidence that services are becoming more sophisticated throughout the world and that the developing countries have the opportunity to avoid the tragic errors of service delivery made by those that are supposedly more advanced.

These triennial conferences and the subsequent publication are unique and mark the progress made on the international scene of our sub-speciality. Long may they continue to provide a universal knowledge base, a sense of purpose, and a chance to renew our dedication and commitment to people with a mental handicap throughout the world.

JOAN BICKNELL, *Professor of the Psychiatry of Mental Handicap, St George's Hospital Medical School, London*

Obsessive Compulsive Disorders: Theory and Management. Edited by MICHAEL A. JENIKE, LEE BAER and WILLIAM E. MINICHIELLO. Bristol: Wright. 1986. Pp 196. £16.00.

Sir Aubrey Lewis's concise, clear, and assertive writings on obsessional disorders provided a dependable guide and a standard for over 30 years. It is reasonable to expect new publications on this subject to add to or improve Lewis's account, and to incorporate the information gathered since the resurgence of interest that took place in the seventies. Many parts of this book are considerably less adequate than the writings by Lewis, some sections are misleading, and all but two chapters make little or no reference to recent research findings.

The book is divided into 11 chapters that are intended to cover aetiology, epidemiology, behaviour therapy, somatic treatments, psychotherapy, prognosis, related disorders, the biology of the disorder, and the management of the individual patient. Dr Jenike, the first-named and presumably the senior editor, wrote six of the eleven chapters and was co-author of two others.

After reading the first few chapters of the book it became evident that Lewis's work had either been forgotten or was being ignored. As there is no author index, it was necessary to make a count of the number of references to his work, and the total for the entire volume is two – both minor.

Most of the contributions are unsatisfactory. Jenike's chapter on aetiology consists of a brief examination of several possibilities, none of which are considered in detail. His concluding remarks contain the following statement:

> "The fact that OCD (obsessive-compulsive disorder) is refractory to non-behavioural psychotherapeutic manoeuvres but is responsive to certain medications and surgical techniques lends support to a neurologic hypothesis for OCD" (page 18).

Even if that statement were accurate, which is debatable, it does not follow that a neurological hypothesis would be supported. But in any event, the statement fails to consider the effects of behavioural methods of treatment and the theoretical consequences of this evidence. This omission is especially strange, as on page 60 of the same book Baer & Minichiello state that, "in general, these studies have found that 60% to 70% of patients with compulsive rituals were much improved after behavioural treatment", and on page 38, Rasmussen & Tsuang note that, "over the past 15 years, significant advances have been made in the behavioural and pharmacological treatment of OCD".

Jenike opens his chapter on psychotherapy with the admission that "traditional psychodynamic psychotherapy is not an effective treatment for patients with obsessive-compulsive disorder as defined in the DSM". Not easily dissuaded, he then proceeds to give ten pages of advice on how to conduct such psychotherapy. In this chapter, as elsewhere, the information and conceptual approach are out-of-date.

More seriously, Jenike's recommendation of psychosurgery may be misleading. In a chapter that repeats most of the old information, but fails to refer to the large volume by E. Valenstein (*The Psychosurgery Debate.* San Francisco: W. F. Freeman, 1980) devoted exclusively to psychosurgery, Jenike concludes that, "for the patient with disabling OCD, who fails to respond to more conservative treatment, psychosurgery is a viable option" (page 105). This conclusion is not justified by the older evidence, and the one recent study (Tippin, J. & Henn, M. (1982) *American Journal of Psychiatry*, **139**, 1601–1603) which is used to bolster his conclusion consists of no more than five highly selected cases which are incompletely and unsatisfactorily described and analysed.

The chapter on epidemiology by Rasmussen & Tsuang is sound and informative, as is the critical review of behaviour therapy written by Baer & Minichiello. The outcome of the ambitious epidemiological study referred to by Rasmussen is awaited with great interest. These two chapters deserve to be published in better company.

STANLEY RACHMAN, *Professor, University of British Columbia, Canada*

Drug-Induced Diseases in the Elderly. By F. I. CAIRD and P. J. W. SCOTT. Amsterdam: Elsevier Biomedical Press. 1986. Pp 210. US$70.25, Dfl. 190.00.

In no branch of medicine are adverse drug reactions (ADRs) more important than in (psycho-)geriatrics. In the late 1970s an estimated 12% of admissions to geriatric units in the UK were at least partly due to ADRs. The incidence in hospital and general practice is probably falling due to wider understanding of the problems and better education of doctors, but as the authors state, there is "no reason at all for complacency".

This book is sub-titled "*A critical survey of the literature*", which is not strictly true. A great deal of the literature has been read, no doubt with a critical eye, and reference lists are lengthy, but most of the text could not be described as a critical survey. In fact, it is much better and more useful than that. After some short, pithy introductory chapters on epidemiology, mechanisms, and management, the remaining three-quarters of the book deal with specific ADRs, first by systems (neuropsychiatric, gastro-intestinal, cardio-vascular, etc.) and then by main drug groups (psycho-pharmacological agents, anti-convulsants, etc.). Most ADRs are therefore covered twice, once from each standpoint.

The text is set out with exemplary clarity. Paragraphs are short and numbered for easy reference. Tables are numerous, easily understood, and give information at a glance. The index is comprehensive. Thus what we have here is an excellent reference dictionary on ADRs in the elderly, which I have already found useful in clinical practice. I should like to have recommended it for every medical library and (psycho-)geriatric ward in the country. However, I fear that the publishers have ruled this out by setting a price so unreasonably high as to go beyond most means in these straightened times.

ROBIN JACOBY, *Consultant Psychiatrist, The Bethlem Royal Hospital, Kent*

Behavioral Neurology: A Practical Approach. By HOWARD S. KIRSHNER. Edinburgh: Churchill Livingstone. 1986. Pp 230. £24.50.

What is behavioural neurology? It is a discipline which does not appear to exist in the UK, at least under this name. Apparently it is flourishing in the USA, and this is the second textbook in recent years to be published from that country with this title. The author tells us that behavioural neurology is "the study of the effects of brain disease on human behaviour and higher cortical functions. Behaviour, in this context, includes both specific, localised brain functions such as language, memory calculations, and visuospatial ability, and the more difficult to define qualities of intelligence, mood and personality". After a brief chapter on the neurological approach to mental state examination, more than half the book is taken up·with aphasias, alexias, agraphias, apraxias, agnosias, and disorders of the right hemisphere. There follows chapters on the frontal lobes, amnesias, dementia and ageing, delirium and acute confusional states, and finally behavioural aspects of epilepsy.

A comparison with the book on behavioural neurology by Pincus & Tucker reveals considerable discrepancies and uncertainties as to the scope of behavioural neurology in the USA. Pincus & Tucker include chapters on schizophrenia, depression, psychosis, movement disorders, sleep, limbic system, and violence, none of which are covered in Kirshner's book. Indeed, the great bulk of this monograph is a more detailed review of 'disorders of intellectual functioning' (using the Pincus & Tucker terminology) without specific consideration of organic brain syndromes in children, early childhood autism, or 'attention deficit disorder'. Some aspects of this volume are also covered in greater detail by Benson in a monograph in the same series on aphasia, alexia, and agraphia.

The author advises us that behavioural neurology should be distinguished from two related fields, neuropsychology and biological psychiatry. The former he defines as "the branch of psychology which deals with assessment of brain disease by standardised psychological tests". The latter is defined as "the study of physical or organic abnormalities underlying primary psychiatric diseases such as depression and schizophrenia, in which no primary brain abnormality is proved" (a view which is clearly not shared by Pincus & Tucker). There is in this book, as the author admits, much from the field of neuropsychology, which he regards as complementary to the subject of behavioural neurology.

We are told that the history of behavioural neurology has had two distinct phases. It began with the descriptions of the great 19th century neurologists. It lapsed in the early and middle years of the 20th century under the influence of psychological theories and religious beliefs. We are now in the second golden era of behavioural neurology, which began after World War II when the late Dr Norman Geschwind, as much as any individual, rediscovered the important descriptions of the 19th century behavioural neurologists and renewed the study of the higher cortical functions. Perhaps the author is describing a purely American phenomenon and he does not know that the 19th century clinical findings and descriptions, which were after all European, were not lost sight of or forgotten in Europe (although it must be admitted that there is a danger of this happening now in the UK).

This volume is indeed an update of the 19th century clinico-anatomical approach to selected aspects of neurology. It contains a liberal number of neuro-anatomical diagrams, modern CT scan pictures, and tables relating clinical symptoms to focal lesions. The main emphasis is diagnostic, with relatively few excursions into therapy. What is lacking is any 'functional' component to the picture, using the word in its 19th century neurological sense. For example, there is not an EEG tracing in sight, even in the chapter on behavioural aspects of epilepsy. Clinical practice so often reveals that, even with the help of the modern CT scanner, the strict relationship of clinical syndromes and symptoms to local lesions is not always so straightforward as this book suggests.

E. H. Reynolds, *Consultant Neurologist, Maudsley Hospital, London*

Transcultural Psychiatry. Edited by John L. Cox. Beckenham: Croom Helm. 1986. Pp 337. £29.50.

This is a multi-author text which sets out to be a contemporary critique of transcultural psychiatry. The book is divided into 18 chapters, and the various authors have in the main drawn on information not only from their clinical, research, or anthropological standpoints but from their intimate knowledge of the various socio-cultural groups described. The essential material includes: the epidemiology of mental illness across cultures; migration; family therapy across cultures; psychiatric services for ethnic minorities; depression in ethnic minorities; and politics and the impact of racism on mental illness. The book dispels the common myth that the mainstay of transcultural psychiatry is a cluster of culture-bound syndromes presenting in ethnic minorities (from far-away lands), often with bizarre or dramatic manifestations. The salient point is that no society is entirely homogenous and that there are equally important cultural determinants to the presentation and outcome of disease in, say, a first generation West Indian living in Britain as in an Irishman who has emigrated to England.

The significance of this is often inadequately explored or underestimated in the mental state in clinical practice. The difficulty in applying 'orthodox' descriptive psychopathology to individuals of varying cultural backgrounds is also illustrated. For instance, in the definition of a delusion (a false, unshakeable belief held against all evidence to the contrary, and also out of keeping with the patient's social and cultural background), how sure can one be in reliably eliciting this phenomenon without being *au fait* with every culture? A similar point is also made with regard to nosology.

Racism, prejudice, and the paucity of an adequate psychiatric service for ethnic minorities makes for sombre but imperative reading, and warns of the dangers in underestimating the level of distress caused by deprivation and isolation which pervades the life of many minority groups.

The multiplicity of contributors with their differing styles makes the book a little disjointed, and many readers may prefer to use it more as a source of references (with which the book is studded) than as a source of basic information. This, however, would be a pity, as on the whole the book is well written and has a definite clinical bias. It succeeds in providing an excellent review of essential aspects in transcultural psychiatry. As a result, most workers in psychiatry would benefit from reading this book, and it would no doubt stimulate in some an interest in medical anthropology.

B. A. Johnson, *Registrar in Psychiatry, The London Hospital (St Clement's)*

Genetic Risk. By Stephen Thomas. London: Penguin. 1986. Pp 182. £3.95

This book has been written by a layman, a one-time consumer of genetic counselling, for other consumers or potential consumers. It provides a clear and well set-out discussion of the basis of human genetics and the application of genetics in clinical practice. There are useful chapters on pre-natal testing, abortion, contraception, and sterilisation, as well as artificial reproduction. There is a well considered chapter, entitled 'Decisions', which describes the practical aspects, the ethical problems, and the potential benefits of genetic counselling. In the main the book deals with single gene disorders and chromosomal abnormalities, with relatively little on multifactorial conditions. It is therefore only of limited use to a reader whose specific aim is to obtain a straightforward guide to genetics as applied in psychiatry. However, it is well researched, is carefully and clearly written, and can be confidently recommended to those without a medical or biological background who wish to obtain a brief, balanced overview.

PETER MCGUFFIN, *Professor of Psychological Medicine, University of Wales, Cardiff*

Neurology and Neurobiology, Vol. 17: Two Hemispheres—One Brain. Functions of the Corpus Callosum. Edited by FRANCO LEPORE, MAURICE PTITO and HERBERT H. JASPER. New York: Alan R. Liss. 1986. Pp 565. £89.00.

This volume is one of a well-known series and contains the proceedings of a conference held in Montreal in 1984. It is well produced, adequately illustrated, and contains a series of chapters which span the fields of philosophy via anatomy to neuropsychology. I particularly enjoyed the introductory chapters by Sperry and Bogen, both of whom give overviews of the conceptual issues involved in considering the behavioural results of commissurotomy. To the casual observer, patients who undergo this procedure appear normal. How can it be that the result of severing some 200 million nerve fibres which join one half of the brain to the other is a splitting of consciousness which is only demonstrable using controlled lateralised testing? The results from such tests indicate that the subjects have a consciousness in each hemisphere and each show appropriate reactions to social events. This finding is expanded by the work of Jeeves, which describes work on acallosal patients. The puzzle of how separate consciousnesses are fused in normal people is the starting point for other chapters which aim to show that the corpus callosum is not just for holding the two hemispheres together.

The anatomy of the corpus callosum is particularly well served in historical perspective (Collonier) and in detailed reviews of the topography of commissural fibres. The relevance of callosal anatomy to the thorny problems of laterality in humans is discussed by Witleson in 'Wires of the mind'. Functions of the corpus callosum are dealt with in depth in the sections on electrophysiology, animal behaviour, and human neuropsychology.

The last section of the book contains a series of useful reviews critically assessing investigations using particular techniques (e.g. cerebral blood flow, visual masking) or theoretical approaches (e.g. complementary specialisation).

In summary, this is a useful book. The introduction will be of interest to any general reader intrigued by notions of brain and mind. There is enough detail in other sections to make this a handy reference work. Its price takes it beyond the reach of many who would profit from it, and probably ensures that it will only be found on library shelves.

GARETH W. ROBERTS, *Clinical Research Centre, Northwick Park Hospital*

Suicide. Edited by ALEC ROY. London: Williams & Wilkins. 1986. Pp 205. £27.00.

Suicide is statistically a relatively rare event which is difficult to predict even after intensive individual assessment, and the practical problems which beset research into it are no less formidable. Nevertheless, the assessment of a suicide risk and its prevention is an important aspect of day-to-day clinical practice. This volume is a welcome addition to the literature on the subject.

A dozen or so distinguished researchers into suicide have contributed as many chapters over a wide range of topics relevant to suicidal behaviour. Although there is inevitably a certain amount of redundancy of material from one chapter to another, each is closely linked with the wider literature which on the whole is commendably up-to-date. Topics range widely from epidemiological, genetic, and biological themes, through a systematic consideration of psychotic syndromes and physical illness, to issues concerned with management and prevention. Much of the material is thought-provoking, and the underlying approach of each author is discernible in each chapter: this adds to the authoritative quality of the volume as a whole. Much information is packed into the deceptively slim size and it is no quick read.

Those who need rapid access to reference material for teaching purposes will certainly find this book useful. It also deserves attention from anyone with a special interest in suicide and its prevention. Entirely written by psychiatrists, its emphasis is on medical and other clinical aspects of the problem. It is to be hoped that its second edition will also include more detailed consideration of the environmental and relationship factors which play such an important part in deciding whether or not a vulnerable individual will commit suicide.

H. G. MORGAN, *Professor of Mental Health, University of Bristol*

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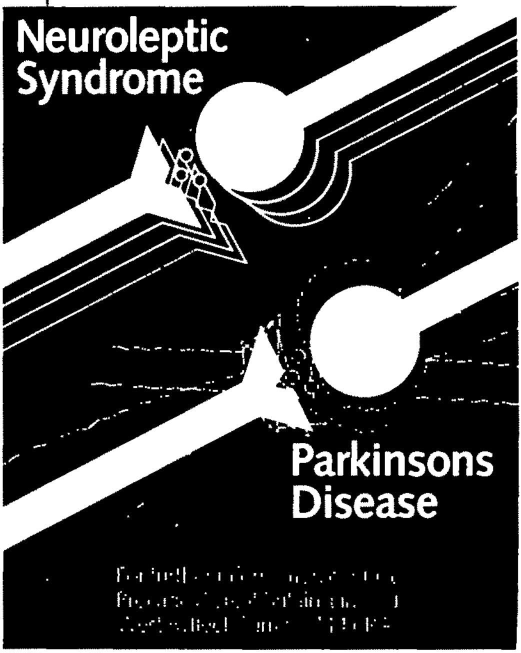

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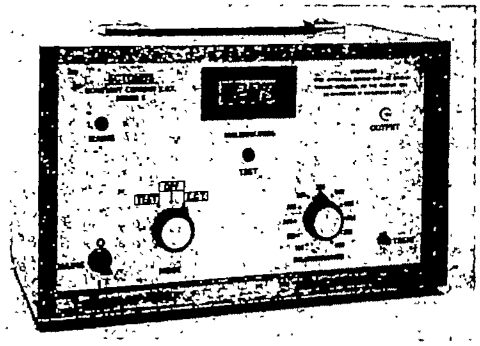

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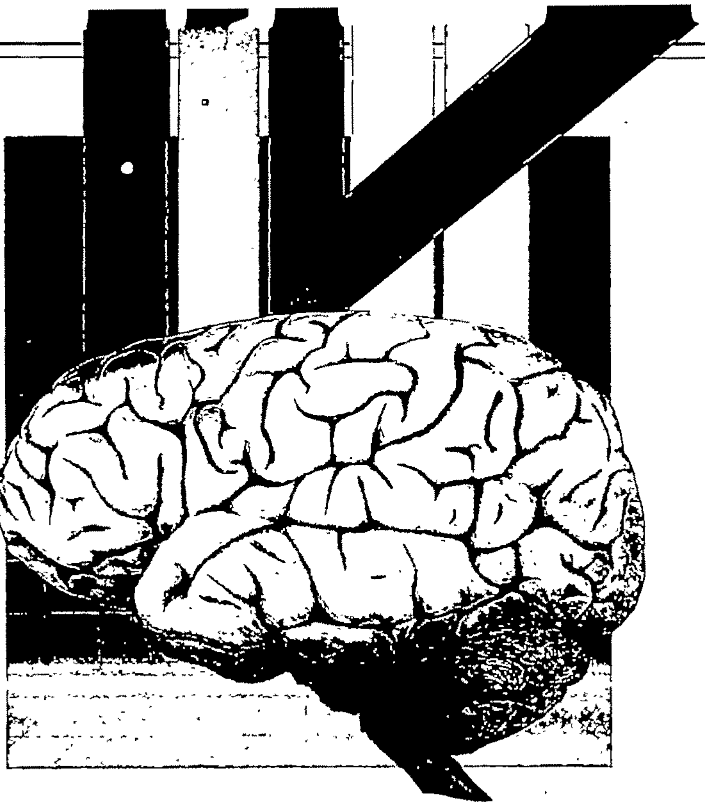

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British Journal of Psychiatry (1987), **150**, 425–427

Annotation

Neuropsychiatry

D. ROGERS

The last 20 years have seen a rapidly growing interest in disorder of brain function in psychiatric illness but this is only the latest phase in a steady development of ideas over the last three centuries. Throughout this time, biological and psychological formulations of psychiatric disorder have co-existed and, at different periods, one or other has been the focus of attention. Thomas Willis, who coined the word 'neurology' 300 years ago, felt that psychiatric disorder represented brain disorder, but it was only during the nineteenth century, as understanding of brain function made its first real progress, that this opinion became more explicit and accepted. By the second half of the century, it was axiomatic that psychiatric disorder was synonymous with brain disorder (Griesinger, 1845; Maudsley, 1873; Ferrier, 1878); all that was lacking was the actual neurophysiological basis of mental disorder.

Ironically, as this neurophysiological knowledge then gradually accumulated, the focus of attention shifted to investigation of abnormal mental states divorced from cerebral function. Thus, as the first intimation of chemical mediation of nerve impulses (Elliot, 1904) and the concept of extrapyramidal motor disorder (Wilson, 1912) appeared, Bleuler (1911) put forward his psychological concept of schizophrenia. As the electroencephalogram was introduced (Berger, 1929) and the insights into neurotic and psychotic phenomena provided by epidemic encephalitis described (von Economo, 1931), the psychological approach pioneered by Freud, Adler, and Jung, which sought to construct causal explanations out of the stuff of subjective experience, was claimed to be more successful than the physiological approach (Hart, 1932). At the same time as the limbic system was being proposed as a cerebral basis for emotion (Papez, 1937), Lewis (1934), in his classic papers on depression, condemned the physiological and neurological conceptions of the previous generation of psychiatrists and the 'biological tendencies' of those contemporary psychiatrists who maintained an interest in the central nervous system.

Ey and his colleagues (1947), in the first extensive critical study of the relationship of neurology and psychiatry, concluded that this hinged on conflicting conceptualisations of the relationship of psychiatric disorder and the brain. For some, psychiatric disorders were disorders of the nervous system, neurologists specialising in disorders below the brainstem and psychiatrists in those above; this could be called the neurological paradigm. For others, psychiatric disorder was radically different from cerebral disorder, psychiatry having nothing in common with neurology; this could be called the psychological paradigm. These paradigms represent conceptually different approaches to understanding psychiatric disorder. Either approach is valid but they are mutually exclusive like the two different views of a Necker cube. The same psychiatric disorder can be viewed in terms of either paradigm. Individuals make their own decision as to which approach provides a more satisfying understanding of a particular disorder. Majority opinion is subject to an historical process. After the turn of the century, most psychiatrists favoured the psychological rather than the neurological paradigm, and most neurologists left the study of behavioural disorder to philosophy.

About 30 years ago, the neurological paradigm in psychiatry started to re-establish itself. Between 1950 and 1970, the main theory of the genesis of infantile psychosis changed from a psychogenic to a neurophysiological one (Kolvin *et al*, 1971). Anderson (1952) described a personal conversion from one paradigm to the other, and how the symptoms in adult patients with schizophrenia that she had previously conscientiously tried to interpret psychodynamically now appeared quite similar, if not identical, with known organically determined symptoms, as some had consistently maintained (Kleist, 1960). From this time the neurological paradigm became an increasingly orthodox position (Symonds, 1960; Hunter, 1973; Kiloh, 1982), and technological advance again stimulated interest in the investigation of major mental disorder. The rise of biological psychiatry was evidence of this new interest, although its concentration on biological processes managed to avoid the neurological implications of the change in approach. Neurologists developed a new interest in behaviour which grew to become 'behavioural neurology'. Some (Damasio, 1984) completely excluded psychiatry from participation in this new speciality, although others

(Stevens, 1982) pointed out that it was in fact another name for neuropsychiatry. The modern revival of neuropsychiatry is bringing psychiatry and neurology together again after a century of separation; 'we are back where Griesinger started' (Caine & Joynt, 1986).

Conceptions of neurological disorder have also evolved. Not all neurological disorder is constant and irreversible. Basal ganglia disorders typically fluctuate in severity and the extensor plantar response after an epileptic fit is fully reversible. Neurological features of psychiatric illness are typically variable in expression and can be completely reversible, including such 'hard' neurological signs as extensor plantar responses in schizophrenia (Claude *et al*, 1927). Neurological features fluctuate in severity with the severity of psychiatric features of both neurological and psychiatric disorder (Modai *et al*, 1985; Tucker & Silberfarb, 1978). Thus the more severe the psychiatric disorder the more neurological it will appear. Certain neurological disorders, for example oculogyric crises, basal ganglia diseases, epilepsy and spasmodic torticollis can be precipitated, exacerbated, improved or completely resolved by psychosocial factors (Delbeke & van Bogaert, 1928; Brenner *et al*, 1947, Fenwick, 1982; Jayne *et al*, 1984). There have long been suggestions that symptoms produced by physical trauma to certain areas of cerebral tissue might also arise from transient and reversible lesions in the same areas, including those produced by emotional trauma (Marinesco & Dragenesco, 1929; Vujic, 1952; Whitty, 1956). A proper neuropsychiatric assessment always considers the contribution of psychosocial factors.

The conceptual issues discussed above have important practical consequences. The terms 'organic' and 'functional' both have two distinct meanings. Organic can mean structural or it can mean physical in origin; functional can mean physiological or it can mean mental in origin. Two issues are involved: one is descriptive – the presence or absence of recognisable pathological change, and the other philosophical – the choice between the two paradigms for psychiatric disorder. These two issues are completely independent and bear no necessary relationship to each other. Ambivalent use of the terms organic and functional, however, allows this distinction to be blurred. Thus, the difference between organic and functional psychoses theoretically refers to their presumed physical or mental origin, but in practice hinges on the presence or absence of recognisable cerebral disorder. This means that in clinical and research investigations of functional psychosis, when the investigation reveals brain disorder in particular cases, these are excluded from the study (Johnstone *et al*, 1986*a*, *b*). It allows the same textbook (Pincus & Tucker, 1974) to accept minor neurological abnormalities as commonly found in schizophrenia (p. 108), but give the absence of major neurological deficits as one of the important criteria for its diagnosis (p. 86). It makes the preliminary elimination of organic states a requirement for psychiatric assessment with precise diagnostic instruments such as the Present State Examination (Wing *et al*, 1974) and Research Diagnostic Criteria (Spitzer *et al*, 1978), although the criteria for this important first step are not specified. These paradoxical positions have been consistently criticised (Gibbs, 1938; Ferraro, 1943; Stevens, 1982; Murray *et al*, 1985), but are still orthodox. A basically philosophical issue is interfering with empirical research, since the cases excluded from investigation are likely to be the very ones to throw most light on the cerebral basis of psychiatric disorder.

A neuropsychiatric approach is an approach to psychiatric disorder in general. It is not limited to psychiatric disorder associated with known cerebral disorder. With such an approach there is no absolute demarcation between neurological and psychiatric disorder and disorders can only be classified as neurological or psychiatric in terms of who manages them. Neurological examination and investigation is performed on patients with psychiatric disorder for the information it provides on the disorder and not only to define those cases needing referral to a neurologist. Abnormalities detected are no longer described as incidental or non-contributory. Neurological dysfunction is incorporated as a component of psychiatric syndromes. Medication is not divided into neurotropic and psychotropic. Needless to say, a neurological approach does not in any way preclude a caring or supportive approach to the patient, from either neurologist or psychiatrist.

In England and Wales there are currently ten times the number of consultant psychiatrists (including mental handicap) as consultant neurologists (DHSS, 1983). Most central nervous system disorder is managed by psychiatrists. Thirty years ago, neurology could lay claim to the study of the neural basis of all the psychological functions of man (Brain, 1958). Now, a neurological approach is becoming increasingly relevant to the practice of psychiatry. It is both permissible and desirable. However, even in some university centres, psychiatrists are not permitted to order essential neurological investigations on their out-patients because they are not considered competent enough by their non-psychiatric colleagues (Hays, 1985). In future, neuropsychiatry must form a major part of the training of psychiatrists.

References

Anderson, C. M. (1952) Organic factors predisposing to schizophrenia. *The Nervous Child*, **10**, 36–42.

Berger, J. (1929) Uber des elektrenkepalogramm des menschen. *Arch. Psychiat. Nervenkr.* **87**, 527–570.

Bleuler, P. E. (1911) *Dementia praecox oder die Gruppe der Schizophrenien*. Liepzig: Deuticke.

Brain, R. (1958) Neurology: past, present, and future. *British Medical Journal*, **i**, 355–360.

Brenner, C., Friedman, A. P. & Merritt, H. H. (1947) Psychiatric syndromes in patients with organic brain disease 1. Diseases of the basal ganglia. *American Journal of Psychiatry*, **103**, 733–737.

Caine, E. D. & Joynt, R. J. (1986) Neuropsychiatry . . . again. *Archives of Neurology*, **43**, 325–327.

Claude, H., Bourguignon, G. & Baruk, H. (1927) Signe de Babinski transitoire dans un cas de demence precoce. *Revue Neurologique*, **1**, 1078–1081.

Damasio, A. R. (1984) Behavioural neurology: research and practice. *Seminars in Neurology*, **4**, 117–119.

Delbeke, R. & Bogaert, L. van (1928) Le probleme general des crises oculogyres au cours de l'encephalite edidemique chronique. *L'Encephale*, **23**, 855–890.

DHSS Medical Manpower Division (1983) Medical and dental staffing prospects in the NHS in England and Wales, 1982. *Health Trends*, **15**, 35–39.

Economo, C. von (1931) *Encephalitis Lethargica, its Sequelae and Treatment*. (Translated by K. O. Newman). London: Oxford University Press.

Elliott, T. R. (1904) On the action of adrenalin. *Journal of Physiology*, **31**, 20–22.

Ey, H., Ajuriaguerra, J. de & Hecaen, H. (1947) *Les rapports de la Neurologie et de la Psychiatrie*. Paris: Hermann & Cie.

Fenwick, P. (1982) Precipitation and inhibition of seizures. In: *Epilepsy and Psychiatry* (ed. E. H. Reynolds & M. R. Trimble). Edinburgh: Churchill Livingstone.

Ferraro, A. (1943) Pathological changes in the brain of a case, clinically diagnosed dementia praecox. *Journal of Neuropathology and experimental neurology*, **2**, 84–94.

Ferrier, D. (1878) *The localisation of cerebral disease*. London: Smith, Elder & Co.

Gibbs, F. A., Gibbs, E. L. & Lennox, W. G. (1938) The likeness of the cortical dysrhythmias of schizophrenia and psychomotor epilepsy. *American Journal of Psychiatry*, **95**, 255–269.

Griesinger, W. (1845) *Die pathologie und Therapie der psychischen Krankheiten*. Stuttgart: Krabbe.

Hart, B. (1932) Psychology and psychiatry. *Proceedings of the Royal Society of Medicine*, **25**, 187–200.

Hays, P. (1985) Implications of the distinction between organic and functional psychoses. *Acta Psychiatrica Scandanavica*, **71**, 620–625.

Hunter, R. (1973) Psychiatry and Neurology. Psychosyndrome or Brain Disease. *Proceedings of the Royal Society of Medicine*, **66**, 359–364.

Jayne, D., Lees, A. J. & Stern, G. M. (1984) Remission in Spasmodic Torticollis. *Journal of Neurology, Neurosurgery and Psychiatry*, **47**, 1236–1237.

Johnstone, E. C., Crow, T. J., Johnson, A. L. & Macmillan, J. F. (1986*a*) The Nortwick Park study of first episodes of schizophrenia 1. Presentation of the illness and problems relating to admission. *British Journal of Psychiatry*, **148**, 115–120.

——, ——, Macmillan, J. F., Owens, D. G. C., Bydder, G. M. & Steiner, R. E. (1986*b*)—A magnetic resonance study of early schizophrenia. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 136–139.

Kiloh, L. G. (1982) The brain and psychiatry. *Australian and New Zealand Journal of Psychiatry*, **16**, 34–42.

Kleist, K. (1960) Schizophrenic symptoms and cerebral pathology. *Journal of Mental Science*, **106**, 246–255.

Kolvin, I., Ounsted, C. & Roth, M. (1971) Cerebral dysfunction and childhood psychoses. *British Journal of Psychiatry*, **118**, 407–414.

Lewis, A. (1934) Melancholia: A historical review. *Journal of Mental Science*, **80**, 1–42.

Marinesco, G. & Draganesco, S. (1929) Forme nouvelle de maladie familiale d'origine extrapyramidale caracterisee par des crises paroxystiques d'hypertonie. Ses rapports avec l'hysterie. *Revue Neurologique*, **1**, 275–283.

Maudsley, H. (1873) *Body and Mind: an enquiry into their Connection and Mutual Influence, specially in reference to Mental Disorders*. London: Macmillan.

Modai, I., Karp, L., Liberman, U. A. & Munitz, H. Penacillamine therapy for schizophreniform psychosis in Wilson's disease. *Journal of Nervous Mental Diseases*, **173**, 698–701.

Murray, R. M., Lewis, S. W. & Reveley, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, **i**, 1023–1026.

Papez, J. W. (1937) A proposed mechanism of emotion. *Archives of Neurology and Psychiatry*, **38**, 725–743.

Pincus, J. H. & Tucker, G. J. (1974) *Behavioral Neurology*. New York: Oxford University Press.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research Diagnostic Criteria: rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.

Stevens, J. (1982) The neuropathology of schizophrenia. *Psychological Medicine*, **12**, 695–700.

Symonds, C. (1960) Disease of mind and disorder of brain. *British Medical Journal*, **ii**, 1–5.

Tucker, G. J. & Silberfarb, P. M. (1978) Neurological dysfunction in schizophrenia: significance for diagnostic practice. In: *Psychiatric Diagnosis: Exploration of Biological Predictors* (ed. H. S. Askikal & W. L. Webb). New York: SP Medical and Scientific Books.

Vujic, V. (1952) Larvate encephalitis and psychoneurosis. *Journal of Nervous and Mental Disease*, **116**, 1051–1064.

Whitty, C. W. M. (1956) Mental changes as a presenting feature in subcortical cerebral lesions. *Journal of Mental Science*, **102**, 719–725.

Wilson, S. A. K. (1912) Progressive lenticular degeneration: a familial nervous disease associated with cirrhosis of the liver. *Brain*, **34**, 295–509.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *Measurement and classification of psychiatric symptoms. An Instruction Manual for the PSE and Catego Program*. London: Cambridge University Press.

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Review Article

Amnesia:
Organic and Psychogenic

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This paper describes the clinical features of selected examples of organic and psychogenic amnesia, and it discusses the nature of the dysfunction that these amnesias entail. The anterograde component of organic amnesia involves a severe impairment in acquiring (or learning) new information, rather than accelerated forgetting, and this may reflect an underlying limbic or neurochemical dysfunction. Retrograde amnesia has a basis which is (at least partially) independent of anterograde amnesia – in some patients, it appears to involve a failure to reconstruct past experience from contextual cues, and this may reflect a superimposed frontal dysfunction. Two types of confabulation are discussed, one of which ('provoked') is a normal response to poor memory, and the other ('spontaneous') appears to reflect incoherent, context-free retrieval, associated with more severe frontal pathology. It is argued that many cases of psychogenic amnesia may resemble organic amnesia, in that they result from an impaired acquisition of information at the time of initial input, perhaps thereby predisposing the subject to subsequent retrieval difficulties.

> The stream of thought flows on; but most of its segments fall into the bottomless abyss of oblivion. . . . Selection is the very keel on which our mental ship is built. And . . . its utility is obvious. If we remembered everything, we should on most occasions be as ill off as if we remembered nothing.
>
> (William James, *The Principles of Psychology*, 1890)

> We oftentimes find a disease quite strips the mind of all its ideas, and the flames of a fever in a few days calcine all those images to dust and confusion, which seemed to be as lasting as if graven in marble.
>
> (John Locke, *Essay concerning Human Understanding*, 1690)

In recent years, the truth of these two maxims has been demonstrated in Luria's brilliant case histories, which have illustrated the crippling consequences of an extraordinarily absorptive and retentive memory (Luria, 1969), as well as the devastating effects of a 'shattered' one (Luria, 1976). Memory plays a central organising and selective role in our mental life. Moreover, its anomalies of function were the starting point for psychoanalysis; its impairment is an early and common sign of organic dysfunction; and its disruption in dementia is one of the most prevalent and distressing of psychological disabilities. For these various reasons, the study of memory and amnesia should be of immense interest to the general psychiatrist.

Regrettably, progress in this topic has sometimes been impeded by the narrow perspectives of particular disciplines, and by their conflicting uses of terminology. Because of this, the present review will begin with a brief outline of the terminology to be employed here; and it will then consider the topic in four sections:

(1) clinical aspects of organic amnesia
(2) the nature of the deficits in organic amnesia
(3) the neuropathology of amnesia
(4) psychogenic amnesia.

The paper will summarise the principal features of selected examples of organic and psychogenic amnesia, and it will discuss the main theories which purport to account for these disorders in terms of the registration, retention, or retrieval of information. In addition, it will draw attention to resemblances between organic and psychogenic amnesia, in the hope that the extensive research on the nature of the former may also shed some light on the latter.

Terminology

Experimental psychologists distinguish between a *primary* (or *short-term*) memory of very limited capacity, which holds information for a few seconds only, and a *secondary* (or *long-term*) memory of much greater capacity and durability. *Secondary memory*, therefore, encompasses all material recalled beyond a period of a few seconds, and items within it may have occurred within the remote or recent past. *Remote* and *recent memory* tend to be very loosely defined, but generally refer to the relative *age* of particular memories; whereas *retrograde* and *post-traumatic amnesia* are precisely defined in terms of the *onset* of an injury or illness. *Retrograde amnesia* (RA) refers to memory loss for events occurring before the onset of a lesion, whilst *post-traumatic amnesia* (PTA) refers to memory loss for events following this, and, with respect to head trauma, it ends with the return of the continuous registration of 'personal' memories. *Anterograde amnesia* (AA) refers to an impairment in learning new material; it is commonly used synonymously with PTA, although, in traumatic cases, such an impairment may persist long after the termination of PTA.

There have been various attempts to investigate the subcomponents of primary and secondary memory. *Working memory* refers to a particular model of primary memory, distinguishing its subcomponents and emphasising its information-processing activity. Secondary memory has been considered in terms of *episodic* (personal) memory and *semantic* (conceptual) memory, or, alternatively, in terms of *conscious recollection* and memory for skills (*procedural* memory): these will be discussed below. Traditionally, primary and secondary memory were thought of as separate *stores* operating in series: nowadays, greater emphasis is placed upon the *processes* they entail, and some authorities argue that they operate in parallel rather than in series.

The present paper will use the term *psychogenic amnesia* to refer to all instances in which memory loss is presumed to have a psychological, rather than an organic, basis. This term allows for the facts that (i) *both* organic and psychogenic amnesia involve disordered 'function', and that (ii) some *combination* of conscious and unconscious ('hysterical') factors may precipitate a psychologically based amnesia.

The following two cases illustrate the way that some of these concepts will be used in the present paper.

A.B. is a 62-year-old Korsakoff patient whose Wernicke encephalopathy occurred in 1970, after many years of heavy drinking. He performs at a normal or near-normal level at tests of primary memory, such as digit span. He can find his way about the hospital (procedural memory), although he is unable to describe ('recollect') his route and he soon gets lost if he leaves the immediate vicinity. He shows severe impairment at tests of secondary memory which require him to learn new material (AA). He recalls virtually nothing about recent events, either in his personal experience (episodic memory) or requiring conceptual knowledge, such as the name of the present Prime Minister (semantic memory). He also performs very poorly on a remote memory test, which asks about news events from the late 1930s to the early 1980s – although there is relative sparing of memories preceding the mid-1950s. Obviously, both RA (pre-1970) and AA (post-1970) have contributed to this man's remote memory impairment.

By contrast, C.D. is a 24-year-old man who suffered a moderately severe concussion injury in a road traffic accident. He developed an RA of 10–15 minutes and a PTA of 24 hours. During the period of his PTA, his performance at tests of primary memory was impaired, but this recovered soon afterward. When reassessed some weeks after his accident, his recall of recent and remote events appeared completely intact except that his RA and PTA remained unchanged, i.e. there was a discrete gap in his episodic memory. He also showed a mild impairment on formal learning tests (AA).

Clinical aspects of organic amnesia

The amnesic states seen in clinical practice can be classified along two dimensions – organic *vs* psychogenic, and discrete (time-limited) episodes *vs* persistent memory impairment. Table I provides examples of amnesic disorders falling within the four quadrants of this classification. Obviously, there is some overspill between the quadrants: for example, head injury and hypoglycaemia often give rise to discrete amnesic 'gaps', but sometimes they may produce more persistent memory impairment as well. The clinical characteristics of selected examples of organic amnesia will be briefly discussed in the present section.

Lishman (1987) has described in detail the clinical assessment and investigation of organic amnesia. Some clinical tests of memory, e.g. the use of a

TABLE I
Amnesic states

Discrete episode	*Persistent impairment*
Organic	
Toxic confusional state	Drug toxicity
Head injury	Amnesic syndrome, specific
Epilepsy	Dementia, global
Alcoholic 'black-out'	
Hypoglycaemia	
Transient global amnesia	
Post-ECT	
Psychogenic	
Fugue states	Pseudodementia
Situation-specific e.g. for an offence	

memory and orientation scale and recall of a passage of prose, appear to be more discriminating than others (Kopelman, 1986*a*); and formal neuropsychological assessment is usually essential.

Head injury

Head injury is the classical instance of a discrete, organic amnesia. The familiar pattern of memory loss consists of a brief period of retrograde amnesia (RA), a longer period of post-traumatic amnesia (PTA), and islets of preserved memory within the amnesic gap (Russell & Nathan, 1946). Occasionally, PTA may occur without any RA, although this is more common in cases of penetrating lesions. Sometimes, there is a particularly vivid memory for images or sounds occurring immediately before the injury, on regaining consciousness, or during a 'lucid' interval between the injury and the onset of PTA.

As is well known, the duration of PTA is assumed to reflect the degree of underlying diffuse brain pathology. It is predictive of eventual cognitive outcome (Brooks, 1984), psychiatric outcome (Lishman, 1968), and social outcome (Russell & Smith, 1961), although these relationships are weaker than is often assumed. There appears to be a relationship with age such that older subjects tend to have a longer PTA and more serious deficits at a given PTA (Russell & Smith, 1961), whereas in subjects under 30 PTA is sometimes found to be less effective as a predictor of subsequent memory impairment (Brooks, 1972).

Following a mild head injury, a neurotic ('post-traumatic') syndrome sometimes ensues in which 'forgetfulness' is a prominent complaint. The aetiology of this syndrome remains controversial, but it is clear that the symptoms often persist long after the settlement of any compensation issues. In more severe head injury, the ability to learn new material is the slowest cognitive deficit to recover, and the pattern of residual memory deficit resembles, in many respects, that seen in the Korsakoff or 'amnesic' syndrome (Brooks, 1984; Baddeley *et al*, 1987; Merskey & Woodforde, 1972).

Alcoholic black-out

Goodwin *et al* (1969) described discrete episodes of 'memory loss for significant events' which occurred in 64% of a sample of 100 hospitalised alcoholics. These 'black-outs' were always associated with prolonged alcohol abuse and severe intoxication, and were of two types. In the 'fragmentary' type, the subject became aware of his memory loss only after being told about an event later: there were 'islets' of preserved memory, and the amnesia tended to shrink through time in a manner analogous to the RA of a head injury. In the 'en bloc' variety, the subject usually became aware of the memory loss on awakening from sleep with a sense of 'lost time': the amnesic gap had a definite starting point, islets of preserved memory were rare, and the memories were very seldom recovered. In a minority of cases, the subjects 'came round' while alert and awake, realising that they had no recollection of what they had been doing: sometimes they had travelled, paid cheques, stayed in hotels, in a similar manner to a 'fugue state'. Three subjects reported 'en bloc' black-outs lasting between 2 and 5 days. In addition, 'state-dependent' phenomena were reported by 61% of the patients who had black-outs: commonly, the subjects would hide money when drunk, be unable to find it when sober, and would retrieve it again when intoxicated. The authors cited a 47-year-old woman who wrote letters when drinking, was unable to decipher them when sober, and would resume writing as soon as she had had a few drinks: "It was like picking up the pencil where I had left off."

The diagnosis of an alcoholic black-out can be important in medico-legal cases in which a defendent may claim amnesia for his offence, as will be discussed below.

ECT

This is an iatrogenic form of 'discrete' amnesia, and the adverse effects of ECT on the recall of personal memories were noted from an early stage (e.g. Janis & Astrachan, 1951). In recent years, the pattern of the memory deficit has been carefully mapped out. Verbal memory appears to be particularly sensitive to disruption, and unilateral ECT to the non-dominant hemisphere produces considerably less memory impairment than bilateral ECT (Squire, 1977), making it important to identify the non-dominant hemisphere by a valid procedure (Kopelman, 1982). Squire and his colleagues have found that subjects tested within a few hours of ECT show a retrograde impairment for information from the preceding 1–3 years, a pronounced anterograde deficit on recall and recognition tests, and accelerated forgetting of newly learned information (Squire, 1977, 1981; Squire *et al*, 1984). Although these deficits have been attributed by some to cholinergic depletion, they are more widespread than would be expected on such a basis, and would seem more likely to be attributable to gross metabolic disruption.

Follow-up studies, 6–9 months after ECT, reveal that memory performance on objective tests returns to normal, apart from a persistent loss of material acquired within a few hours of the convulsions (Squire, 1977; Freeman *et al*, 1980; Squire & Slater, 1983; Frith *et al* 1983). However, complaints of memory impairment persist (Squire, 1977; Freeman *et al*, 1980; Frith *et al*, 1983), and are still evident after 3 years (Squire & Slater, 1983). Squire & Slater (1983) found that the complaints focus upon the period for which there has been an initial, authentic (retrograde and anterograde) amnesia. Freeman *et al* (1980) reported that the subjects who complain most do, in fact, perform poorer on objective tests than those who do not complain, but Squire & Slater (1983), using similar tests, did not find this. Squire & Slater (1983) and Frith *et al* (1983) both indicate that the 'complainers' are the patients who have recovered least well from their depression.

Korsakoff syndrome

The Korsakoff syndrome is the classical example of a 'persistent' organic amnesia. It is best defined as "an abnormal mental state in which memory and learning are affected out of all proportion to other cognitive functions in an otherwise alert and responsive patient" (Victor *et al*, 1971).

Lawson (1879) and Korsakoff (1889) both described profound amnesia occurring as a consequence of extensive alcohol abuse, but they also recognised that other factors could produce an identical syndrome. Although Korsakoff indicated that recent memories tend to be more severely affected than remote, he emphasised that the retrograde component to the amnesia is very variable and sometimes extends back many years or decades. Subsequent clinical and experimental studies have confirmed the extensiveness of the remote memory impairment, but tend to find that there is indeed a relative sparing of the most distant memories, whether tested in terms of the recognition or recall of famous faces and news events, or the recall of personal memories (Butters & Albert, 1982; Squire *et al*, 1984; Baddeley & Wilson, 1986).

Korsakoff (1889) also drew attention to the patients' disorientation in time and their inability to recall the temporal sequence of events. The disorientation in time can result in an under-estimation of the time spent in hospital (Moll, 1916) and of the patient's own age (Zangwill, 1953), rather similar to that described in some schizophrenic patients. The failure to recall the temporal context of events may contribute to confabulation, which often involves the inappropriate and jumbled recall of genuine events, rather than the fabrication of fictions (Korsakoff, 1889; Victor *et al*, 1971). "Telling of a trip she had made to Finland before her illness . . . [the patient] mixed into her story her recollections of the Crimea, and so it turned out that in Finland people always eat lamb and the inhabitants are Tartars" (Korsakoff, 1889).

It is commonly stated that both Wernicke and Korsakoff failed to recognise the relationship of their two syndromes (e.g. Victor *et al*, 1971). However, Korsakoff (1889) did in fact notice confusion, ataxia, nystagmus, and opthalmoplegia in some of his patients. Describing patients seen in an acute neurological service, Victor *et al* (1971) reported that Wernicke signs preceded the amnesia in 96% of Korsakoff cases; whereas the recorded prevalence in psychiatric series tends to be rather lower. Moll (1916) suggested that the disorder can have either an acute or an insidious onset; and recent studies have confirmed that the initial clinical manifestations of the disorder may range from acute coma (Torvik *et al*, 1982), through the classical acute Wernicke syndrome, to an insidious onset (Cutting, 1978*a*). Moreover, the characteristic neuropathology is identified in many cases at autopsy who have survived in the community without being diagnosed in life (Harper, 1983; Torvik *et al*, 1982). This latter finding provides some support for those who believe that the clinical Korsakoff syndrome may represent an extreme form of the memory impairment commonly found in non-Korsakoff alcoholics (e.g. Butters & Albert, 1982).

Alzheimer-type dementia

Memory impairment is a prominent early symptom in dementing patients, but, unlike the above disorders, it takes place in the context of widespread cortical atrophy and multiple cognitive deficits. Morris and Kopelman (1986) have suggested that the pattern of memory impairment in Alzheimer-type dementia can be viewed as the outcome of 'information-processing' deficits superimposed upon an amnesic syndrome; and there is evidence that this pattern may differ from that seen in normal aging (Huppert & Kopelman, 1987).

There is a profound impairment in learning new information but, surprisingly, the forgetting rate of 'acquired' information is normal in early cases, once adequate learning has been accomplished, a pattern which is consistent with that expected on the basis of cholinergic depletion (Kopelman, 1985*a*). There is also an extensive impairment of remote memory, although, to date, objective tests have failed to establish evidence of any relative sparing of the most distant memories (Wilson *et al*, 1981). Superimposed on this pattern is a substantial impairment at tests of primary memory, e.g. digit span (Miller, 1973; Kopelman, 1985*a*); and there may also be impairment in certain aspects of semantic memory, requiring knowledge of facts or concepts (Weingartner *et al*, 1983). Furthermore, the impact of these various memory deficits upon the patients' daily lives is likely to be potentiated by an interaction with the effect of their other cognitive defects. For example, the impairment of primary memory is most severe in younger patients, who have the most advanced neuropathology, and this deficit may exacerbate the effect of any subtle dysphasic difficulty (Kopelman, 1986*b*). Similarly, as discussed below, the combination of amnesia and severe frontal dysfunction may precipitate very florid confabulation in some cases.

Clinical trials of cholinergic 'replacement' therapy to alleviate the memory disorder in dementia have tended to be a little more successful than trials of other pharmacological agents, although all are disappointing. These studies have been reviewed in detail elsewhere (Hollander *et al*, 1986; Kopelman & Lishman, 1986; Kopelman, 1986*c*).

Confabulation

Berlyne (1972) distinguished between 'momentary' confabulation, which is fleeting and has to be provoked, and 'fantastic' confabulation which is sustained, wide-ranging, grandiose, and spontaneous. These might better be termed 'provoked' and 'spontaneous' confabulation as the former is seen only in response to questions probing the subject's memory, whereas the latter is readily evident in his or her everyday conversation.

Provoked confabulation is common in amnesic patients when given memory tests and may represent a normal response to a faulty memory; it may be related to the distortions and intrusions commonly produced by healthy subjects when they remember something poorly (Bartlett, 1932). In a series of 16 Alzheimer and 16 Korsakoff patients described elsewhere (Kopelman, 1985*a*,*b*), the present author obtained instances of provoked confabulation in seven Alzheimer and eight Korsakoff cases, usually in the immediate or delayed (45 minutes) recall of a prose passage. Moreover, when 17 healthy subjects were asked to recall this passage after a 1-week delay, eight subjects showed similar distortions or confabulations, e.g. in recalling a woman whose purse had been stolen in a high street, it was said that she was a thief who was stopped by the police, or that the episode took place near a railway station. (A paper reporting these data in more detail is in preparation.)

Spontaneous confabulation, on the other hand, is a pathological phenomenon, which may result from the superimposition of frontal lobe pathology on an organic amnesia. This suggestion was first made by Luria in 1976, and, subsequently, a number of other researchers have identified an association between confabulation and the presence of either frontal lobe pathology or evidence of frontal dysfunction (Stuss *et al*, 1978; Kapur & Coughlan, 1980; Baddeley & Wilson, 1986). According to Luria (1976), this kind of confabulation may sometimes involve the inappropriate recall of real memories, jumbled in temporal sequence (cf. Korsakoff, 1889; Victor *et al*, 1971), whereas Berlyne (1972) emphasised the fabrication of fictions: it seems likely that the latter can be grafted on to the former. Several authors have noted that the confabulations can be preoccupying, bizarre, and held with firm conviction, in which case they may be distinguishable from delusional memories only in that they occur in the context of an organic amnesia rather than a psychosis. Possibly because of the association with fairly severe frontal lobe pathology, spontaneous confabulation appears to be much commoner in the more advanced stages of Alzheimer's disease than in the chronic phase of Korsakoff's syndrome. In Berlyne's (1972) series, it occurred in eight out of 62 dementing patients, but in only one Korsakoff patient who was described as having had a frontal lobe syndrome following a head injury. In the present author's series, it was seen in only two cases, both of whom were Alzheimer patients.

Kapur and Coughlan (1980) described a 48-year-old building contractor who had an (anterior communicating) aneurysm clipped following a subarachnoid haemorrhage. CT scan showed a well-defined area of left frontal hypodensity. An initial right-sided weakness recovered rapidly, but he was left with a pronounced personality change including irritability, apathy, and sexual disinhibition. Performance at delayed recall and 'frontal' tests was poor, but he did surprisingly well at recognition tests. Confabulation was prominent and spontaneous: "He would claim . . . in the morning to have fictitious business appointments when in fact he was attending a day centre, would frequently dress for dinner in the evening in the mistaken belief that guests were coming . . . When questioned about holidays or outings over the past few days, he would again report events that bore no relation to actual happenings. When the correct version of events was brought to his attention, he would either seem puzzled . . . or place the confabulated event in another temporal context".

Nature of the deficits in organic amnesia

Neuropsychologists have devoted considerable attention to trying to identify the nature of the memory deficits in organic amnesia. With regard to anterograde amnesia, many theories have been propounded but they can be broadly classified under four main headings, corresponding to the stage or aspect of memory processes considered impaired. Some theories postulate deficits in the 'acquisition' processes occurring during and shortly after the initial 'registration' of information, whereas others propose impairments in subsequent 'retention' or 'retrieval'.

Faulty encoding

These theories propose that there is a deficit in the psychological processes involved in the initial 'registration' of information. In particular, it has been suggested that, whilst amnesic patients are able to 'encode' the direct, sensory properties of information, they have difficulty in 'processing' its more meaningful (semantic) qualities. For example, Korsakoff patients perform particularly badly at learning word pairs (e.g. hungry–thin) whose recall is normally facilitated by thinking of semantic links between the words (Cutting, 1978*b*). On the other hand, this does not seem to be true of all cases (Squire, 1982), and giving instructions or orienting tasks which encourage the extraction of meaning from a stimulus produces, at most, a relatively small enhancement in the amnesic patients' subsequent recall of that stimulus (Meudell *et al*, 1979; McDowall, 1981). It seems unlikely that a failure to encode semantic information is the fundamental deficit in amnesia, although it remains plausible that there exists some impairment in initial 'encoding' which is at present poorly defined and measured.

Faulty consolidation

A second type of hypothesis proposes that there is impairment in the physiological processes which are assumed to occur shortly after initial registration and to establish ('consolidate') information in memory into some relatively permanent form (e.g. Meudell *et al*, 1979; Moscovitch, 1982). These processes were traditionally thought to involve the 'transfer' of information from primary to secondary memory, operating during a time-period of something less than a minute. The hypothesis was suggested by the finding that primary memory (holding information for a few seconds) is relatively intact, in the presence of profound amnesia, in patients with hippocampal lesions, head injury, and in some studies of Korsakoff patients (e.g. Milner, 1966; Brooks, 1984; Kopelman, 1985*b*). In one particularly intriguing study, Lynch and Yarnell (1973) interviewed six concussed American footballers within 30 seconds of their injury and at intervals thereafter. Although they were initially able to give a lucid account of events occurring just before the blow, they subsequently developed a 'relatively complete' retrograde amnesia.

An attraction of consolidation theory is that the properties and time-course of action of the cholinergic neurotransmitter system might appear to make it an excellent 'candidate' as the physiological substrate of the process (see Kopelman, 1985*a*, 1986*c*). A problem is that such a rapid consolidation process cannot explain why a retrograde amnesia of more than a few seconds can occur; and Squire *et al* (1984) have recently postulated 'consolidation' lasting as long as two years to try to account for this difficulty (see below).

Accelerated forgetting

A third possibility focuses attention upon 'storage' (retention) rather than learning processes. In recent years, it has been suggested that patients who have hippocampal lesions show faster forgetting, even after material has been adequately learned (Huppert & Piercy, 1979; Squire, 1981). In one version of this theory, the accelerated forgetting appears to be viewed as being superimposed upon (and arising independently of) any 'acquisition' deficit (Huppert & Piercy, 1979); whereas, in another version, it is seen as a consequence of a faulty (and protracted) consolidation process (Squire *et al*, 1984).

There is some evidence that normal aging causes a slight increase in the forgetting rate of visuospatial information (Huppert & Kopelman, 1987), and that the gross metabolic disruption following ECT produces accelerated forgetting (Squire, 1981). However, there is surprisingly little evidence that structural lesions produce accelerated forgetting. Patients who have the Korsakoff syndrome, penetrating and non-penetrating head injury, and Alzheimer-type dementia have all been shown to exhibit a normal forgetting rate (relative to age-matched controls), once efforts have been taken to ensure that adequate initial learning has taken place (Huppert & Piercy, 1978; Squire, 1981; Kopelman, 1985*b*; Baddeley *et al*, 1987). It seems that the clinical impression of faster forgetting in such patients arises because information has never been adequately absorbed at initial input.

Faulty retrieval

Two types of retrieval hypothesis have been postulated. 'Pure' retrieval hypotheses postulate a retrieval deficit arising independently of any failure in 'acquisition' processes. For example, Warrington & Weiskrantz (1973) postulated that amnesic patients are unable to suppress inappropriate responses during recall or recognition tasks. They noted that amnesic patients sometimes respond erroneously to memory tests with what had been the correct replies to previous tests (even if these had not been recalled at the appropriate time); and, secondly, that the provision of retrieval cues (e.g. initial letters of words to be recalled) can improve their performance. On the other hand, it has been shown that healthy subjects also exhibit these phenomena when given recall tests at relatively long retention intervals (e.g. a week) – suggesting that they may be a consequence of poor memory, rather than its cause (e.g. Woods & Piercy, 1974). Moreover, restricting the number of choices in a recognition or cued recall test does not necessarily improve amnesics' performance, relative to controls, in the way that this hypothesis predicts that it should (Huppert & Piercy, 1976; Warrington & Weiskrantz, 1978).

A modified retrieval hypothesis stresses evidence that retrieval processes are dependent upon the nature of initial encoding, in terms of a network of cognitive and affective associations, and it suggests that retrieval deficits arise in consequence of initial encoding deficits. However, when couched in these terms, the distinction between an encoding and retrieval deficit becomes essentially linguistic (Squire, 1980).

Retrograde amnesia

In summary, the evidence reviewed above would seem to suggest that the deficit in anterograde amnesia occurs at the stage of memory 'acquisition,' although the precise nature of this fault, in terms of (psychological) encoding or (physiological) consolidation, remains to be specified. Accelerated forgetting does not seem to occur in cases of structural lesions, and any retrieval deficits appear to take place as a consequence of the initial 'acquisition' deficit.

Such an explanation fails to account for retrograde amnesia and for its temporal gradient, with relative sparing of more remote memories in, for example, the Korsakoff syndrome. However, various clinical studies have suggested that retrograde and anterograde amnesia may occur, and show improvement or deterioration, independently of each other (e.g. Goldberg *et al*, 1982; Kapur *et al*, 1986). Differing anatomical substrates for the two aspects of amnesia have been postulated, but there is very little agreement on which site produces retrograde amnesia.

At a psychological level, several factors have been implicated. Squire *et al* (1984) cited the occurrence of a relatively short (1–2 years) retrograde impairment in some head injury cases, post-ECT confusional states, and a bitemporal lobectomy patient, as evidence for disruption of a prolonged 'consolidation' process. Much more commonly, the length of retrograde amnesia in head injury is a few seconds or minutes only (Russell & Smith, 1961), and it certainly seems very plausible that such a short RA might result from a disrupted physiological process, such as 'consolidation'. However, the more extensive remote memory impairment in Korsakoff and Alzheimer cases requires a different explanation.

Butters & Albert (1982) demonstrated a loss of recent memories in chronic alcoholics, which they attributed to a progressive anterograde impairment during the period of heavy drinking. Korsakoff patients perform substantially worse than other alcoholics at remote memory tests, and Butters & Albert suggested that, superimposed on the alcoholics' loss of recent memories, Korsakoff patients exhibit a 'generalised' retrograde impairment, which has an abrupt onset at the time of the Wernicke encephalopathy. Alzheimer patients may manifest a similarly generalised retrograde loss, in which a relative sparing of the most remote memories may be more apparent than real (Wilson *et al*, 1981). It has been suggested that this generalised, retrograde loss consists of a 'general impairment in reconstructing past memories' (Squire *et al*, 1984); and, in this connection, it may be relevant that various authors have emphasised the role of contextual cues (e.g. time, order, place) in the 'reconstruction' (conjuring up) of past memories (e.g. Baddeley, 1982).

Memory for context

There is now evidence that Korsakoff patients and some (but not all) other amnesic patients have particular difficulty in recalling the context of information (Huppert & Piercy, 1976; Mayes *et al*, 1985). In studies of anterograde amnesia, they show a disproportionate difficulty in recalling the temporal sequence of events, the place where something was learned (spatial context), and the source of information even when the information itself has been recalled. In studies of retrograde amnesia, they exhibit problems in dating or ordering events.

There are problems of method in most of these studies, but evidence is accumulating that it is the presence of concomitant frontal lobe pathology in amnesia which may underlie the failure to recall the context of information (Squire, 1982; Mayes *et al*, 1985). Since some degree of frontal dysfunction is common in Korsakoff and Alzheimer patients, it seems reasonable to suppose that this failure to recall contextual information may contribute to their difficulty in retrieving from remote memory – a hypothesis currently being tested by the author. As mentioned above, frontal dysfunction and impaired recall of temporal context have also been implicated as factors producing 'spontaneous' confabulation: it seems plausible that spontaneous confabulation may reflect more extensive frontal lobe pathology, resulting in the extremely incoherent (context-free) retrieval of past memories and associations.

Memory without awareness

Whatever the fundamental deficit in organic amnesia, certain aspects of memory *do* appear to be relatively preserved in amnesia, and these appear to be those components which do not require conscious recollection of personal experiences. In the nineteenth century, James Mill, Henry Maudsley and William James had all emphasised that an essential component of memory is the "additional consciousness that we have thought or experienced (something) before" (James, 1890). However, at the turn of the century, Claparede (1911) demonstrated that "the feeling that (a memory) belongs to the person's experience can be absent in situations in which retention is evident". He cited a Korsakoff patient whose finger he pricked with a pin, and who avoided shaking Claparede's extended hand the next day without knowing why. This lady was able to learn her way around the hospital, e.g. to the bathroom, but was completely unable to describe her route.

Modern experiments have confirmed that severely amnesic subjects can show normal conditioning (Weiskrantz & Warrington, 1979), and can acquire and retain affective reactions despite their profound impairment of 'conscious recollection' (Johnson *et al*, 1985). They can also acquire and show retention in certain skills, e.g. performing jigsaw puzzles or reading back-to-front writing, in the absence of any recall or recognition of having performed the tasks before. In a particularly striking example, Starr & Phillips (1970) described a pianist who was taught a new piece of music but the next day had no recall of it: on being hummed the first few bars, he was able to continue playing the piece, although still unable to recall the name of the piece or that he had been taught it before. Such skill (or 'procedural') learning does not necessarily appear to depend upon well-established premorbid learning, and most researchers postulate that it depends upon a subsystem of memory independent of that which subserves 'conscious recollection' (Moscovitch, 1982; Squire *et al*, 1984).

Probably related is the 'priming' phenomenon, in which a learning episode has a transient, facilitative effect on the performance of a subsequent task. For example, viewing a series of words during a study session makes it especially likely that amnesic subjects will give those words in response to some subsequent test, even though their performance at a recognition test of those words may be severely impaired. Shimamura & Squire (1984) propose that priming involves the activation of 'pre-existing elements or processing structures', which, in turn, may be necessary for skill learning to develop.

A further suggestion, put forward in different ways by Gillespie (1937) and Tulving (1972), is that 'semantic memory' is selectively spared in organic amnesia, i.e. memory for facts, concepts, and language (as opposed to memory for personal experiences or events). Unfortunately, the status of semantic memory as an independent memory subsystem remains equivocal, because the tests used to examine it have usually required the recall of material which has been 'over-learned' premorbidly. Moreover, the concept of 'semantic memory' is itself something of a conglomerate, in that the memory system which subserves our use of language (commonly spared in amnesia) may well differ from that which enables us to name the present or past Prime Minister (characteristically affected in amnesia).

The neuropathology of amnesia

Neuroanatomy and neurophysiology

Table II lists the principal types of pathology which give rise to an amnesic syndrome. Consideration of these pathologies has led many authorities to postulate that two types of lesion are particularly likely to disrupt the formation of new memories: bilateral hippocampal lesions and diencephalic lesions.

TABLE II
Aetiology of the amnesic syndrome

Thiamine deficiency	—alcohol —malnutrition —malabsorption
Head injury	
Post-encephalitis	—e.g. herpes simplex
Anoxia	—e.g. CO poisoning
Vascular lesions	—e.g. thalamic infarction —subarachnoid haemorrhage
Tumour (third Ventricle)	
TB meningitis	
(Bilateral) temporal lobectomy	

Scoville & Milner (1957) reported moderate or severe amnesia in eight patients who had undergone bilateral temporal lobectomy for management of psychosis or epilepsy, the amnesia being most severe in three cases in whom the removal of the hippocampi was most extensive. No amnesia was seen in a case who had a unilateral operation only, nor in a case in whom the hippocampi were spared. One of the most severe cases (H.M.) has been described in detail (e.g. Milner, 1966). He had a retrograde component to his amnesia extending back 2 or 3 years before his operation, and a profound anterograde amnesia. More than a decade after his operation, he was unable to find his way to the home in which he had lived for several years; he performed the same crossword puzzle repetitively without showing practice effects; and he mourned afresh every time he learned that his uncle had died. However, his immediate memory span, his IQ, and his ability to hold conversation were all normal (Milner, 1966). Patients who have suffered a herpes simplex encephalitis sometimes manifest a similarly severe amnesia, and the neuropathology typically involves a profound necrosis of bilateral, medial temporal structures as well as some involvement of the orbitofrontal regions (Hierons *et al*, 1978). The hippocampi and the temporal lobes are, of course, particularly implicated in the neuropathology of Alzheimer's disease (Tomlinson & Corsellis, 1984); and the temporal poles are especially vulnerable to bilateral contusions in head injury (Teasdale & Mendelow, 1984).

Diencephalic involvement was first implicated from autopsy studies of Korsakoff cases. Malamud & Skillicorn (1956) and Victor *et al* (1971) noted that the pathology occurred in the paraventricular and peri-aqueductal grey matter, particularly in the mammillary bodies and medial-dorsal nucleus of the thalamus. Victor *et al* (1971) pointed out that all 24 of their cases, in whom the medial-dorsal nucleus of the thalamus was implicated, had a history of memory impairment (Korsakoff syndrome), whereas five cases in whom it was not affected had a history of Wernicke features only. By contrast, the mammillary bodies were implicated in all the Wernicke and Korsakoff cases examined. Amnesia in association with unilateral or bilateral involvement of the thalamic nuclei, with apparent *sparing* of the mamillary bodies, has been described in cases of tumour, trauma, and infarction (McEntee *et al*, 1976; Squire & Moore, 1979; Speedie & Heilman, 1982). However, Mair *et al* (1979) have described two amnesic cases whose autopsies showed lesions in the mammillary bodies and the midline of the thalamus, but *not* in the medial dorsal nuclei. Mair *et al* suggested that the lesions they described might 'disconnect' a critical circuit running between the temporal lobes and the frontal cortex.

A potential difficulty for the view that the diencephalon and hippocampi are critical in memory formation is that lesions of the fornix bundle, which transmits connecting fibres between them, do not always seem to produce amnesia in animals and man. However, the more recent animal studies suggest deficits consistent with those seen in human amnesia: namely, a relative sparing of primary memory and the learning of affective associations, together with a profound impairment of secondary memory as measured on recognition tests (Owen & Butler, 1981; Carr, 1982). A more serious problem, documented by Markowitsch (1984), is that lesions within the hippocampi and thalamic nuclei themselves do not always give rise to amnesia and, when they do, there is usually concomitant involvement of other nuclei. Whilst acknowledging that there may be 'nodal points' in the neural substrate of memory, Markowitsch argues that the thalamic nuclei and hippocampi do not represent discrete 'functional entities', but are embedded in complex neural circuits and 'neuronal assemblies'. Similarly, Mishkin (1978) has argued that *combined* lesions within the hippocampal (medial limbic) and amygdaloid (basolateral limbic) circuits are required to produce severe amnesia; and it seems plausible to suppose that neurotransmitter depletion within these circuits, which contain cholinergic pathways, may be critical in producing amnesia. The dorsomedial thalamus and medial temporal lobes are the anatomical locations ('nodal points') where these two circuits converge, and are, therefore, particularly vulnerable to the effect of discrete, structural lesions (Speedie & Heilman, 1982).

Neurochemistry

Recent attention has focused upon the neurochemistry of amnesia. In the Korsakoff syndrome, the role of thiamine deficiency was confirmed by the De Wardener & Lennox (1947) study of prisoners of war in South East Asia; and, more recently, Blass & Gibson (1977) have postulated a hereditary abnormality of transketolase metabolism which predisposes some alcoholics to this disorder. However, this latter study was based on only four cases, and other studies indicate wide variability in transketolase activity amongst Korsakoff and other alcoholics (Leigh *et al*, 1981).

In Alzheimer's disease, three sources of evidence suggest that cholinergic depletion contributes to the memory disorder:

(a) neuropathological studies demonstrating reduction in cholinergic enzymes and synthesis
(b) pharmacological studies examining the effect of cholinergic 'blockade' in healthy subjects
(c) clinical trials administering cholinergic agents to Alzheimer patients.

These studies have been reviewed in detail elsewhere (Kopelman, 1986*c*), and it seems likely that cholinergic depletion accounts for some, but not all, of the memory deficits seen in Alzheimer-type dementia. A small study has suggested that there may also be depletion of cholinergic neurons in Korsakoff's syndrome (Arendt *et al*, 1983), and the pattern of the anterograde memory deficit in this disorder would indeed be consistent with such an observation: however, there is insufficient neuropathological evidence to place any great weight on this hypothesis as yet.

The role of other neurotransmitter systems in memory and dementia remains to be elucidated. An impairment of the ascending noradrenergic pathways in Korsakoff's syndrome has been implicated by McEntee *et al* (1984), but their results have not been replicated (Martin *et al*, 1984). Moreover, animal studies implicate the adrenergic system in mechanisms mediating attention and arousal, and trials of vasopressin, which modulates adrenergic activity, indicate an effect on arousal and mood rather than memory (Kopelman & Lishman, 1986).

Psychogenic amnesia

Diagnosis and phenomenology

Table I includes psychologically based amnesias, giving fugue states and situation-specific memory loss as examples of 'discrete' amnesias, and depressive pseudodementia as a more persistent impairment. In both discrete and persistent amnesia, the differentiation of psychogenic from organic causation can be surprisingly difficult. In the discrete type, the diagnosis usually relies on such features as the rate and circumstances of the onset, loss of personal identity (rare in organic amnesia except in advanced dementia), and whether new learning is affected (often spared in psychogenic amnesia, although not necessarily so). In the persistent type, the past and family history, the results of physical investigations, the current pattern of deficit, and the patient's response to it, all provide important clues, but the diagnostic problem can remain perplexing.

The phenomenology of psychogenic amnesia often bears interesting resemblances to organic amnesia. For example, there may be islets or fragments of preserved memory within the amnesic gap. A woman, who was due to meet her husband to discuss divorce, recalled that she was 'supposed to meet someone' (Kanzer, 1939). A young man, who slipped into a fugue following his grandfather's funeral, recalled a cluster of details from the year (1979) which he described (after recovery) as having been the happiest of his life (Schachter *et al*, 1982). The subject may adopt a detached attitude to these memory fragments, describing them as 'strange and unfamiliar' (Coriat, 1907). In many cases, semantic knowledge remains intact, e.g. foreign languages and the names of streets, towns, and famous people (Kanzer, 1939; Schachter *et al*, 1982), whereas in others it is also implicated (Coriat, 1907; Abeles & Schilder, 1935; Kanzer, 1939). Similarly, performance at verbal learning tests has been reported as unaffected (Abeles & Schilder, 1935), mildly impaired (Schachter *et al*, 1982), or more severely impaired (Gudjonsson & Taylor, 1985). Memory for skills is often preserved (e.g. Coriat, 1907), but in the Padola case (Bradford & Smith, 1979) retention of a rudimentary knowledge of aerodynamics and of other skills (e.g. solving jigsaw puzzles) was taken as evidence against an organic amnesia – an interpretation in conflict with the recent findings regarding the relative preservation of 'procedural' memory, reviewed above. Sometimes, memory retrieval may be facilitated by chance cues in the environment (e.g. Abeles & Schilder, 1935; Schachter *et al*, 1982) but deliberate cueing is often unsuccessful (Coriat, 1907; Kanzer, 1939), and the results of amytal abreaction are very variable.

Fugue states

'Fugue states' refer to a syndrome characterised by the abrupt onset of a loss of personal ('episodic') memory and of the sense of personal identity, associated with a period of wandering. The episode usually resolves within a few hours or days, after which the subject is left with a residual amnesic gap for the period of the fugue. Fugue states are rare, but their study has helped to elucidate the factors predisposing to psychogenic amnesia. These are listed in Table III.

There is always a severe precipitating stress, and fugues are reported much more commonly in wartime (Sargant & Slater, 1941). However, they also occur following marital or emotional discord or at times of financial difficulty. A history of depressed mood just before the onset of the fugue is virtually always present, including those cases in which there is some other, formal psychiatric diagnosis (Berrington *et al*, 1956). Occasionally, the fugue may even represent a 'flight from suicide' (Stengel, 1941). For example, Abeles & Schilder (1935) described a

TABLE III

Psychogenic amnesia: factors predisposing to fugue states

1. Precipitating stress e.g.	—marital discord	(Kanzer, 1939)
	—financial problems	(Kanzer, 1939)
	—offending	(Wilson *et al*, 1950)
	—war stress	(Sargant & Slater, 1941; Parfitt & Gall, 1944)
2. Depressed mood		(Kanzer, 1939; Stengel, 1941; Berrington *et al*, 1956)
3. Suicide attempts		(Stengel, 1941; Berrington *et al*, 1956)
4. Past history of head injury		(Abeles and Schilder, 1939; Berrington *et al*, 1956)
5. Past history of alcohol abuse		(Kanzer, 1939; Berrington *et al*, 1956)
6. Epilepsy		(Stengel, 1941)
7. Other neurosis		(Sargant & Slater, 1941)
8. Other organic disorder		(Kennedy & Neville, 1957)
9. Tendency to lie		(Stengel, 1941; Wilson *et al*, 1950; Berrington *et al*, 1956)

woman who deserted her husband for another man: after a week she determined to return to her family but, as she descended the Underground stairway, she was contemplating suicide. "Instead amnesia developed", the authors tersely reported. Stengel (1941) claimed that suicide never occurs during a fugue, but may take place as the subject emerges from the fugue. A past history of an organic amnesia is also common, arising, for example, from a head injury, an alcholic black-out, or from epilepsy. Berrington *et al* (1956) reported that 16 of their 37 cases had previously experienced a severe head injury, and a further three cases head injury of uncertain severity. On the other hand, epilepsy is reported less commonly than is generally supposed, Stengel giving the highest figure (10%). In summary, it appears that some subjects, who have experienced an earlier organic amnesia, are predisposed to developing a psychogenic memory loss, in the presence of depressed mood and a severe precipitating stress. On the other hand, several authors have remarked that these patients tend to be rather unreliable personalities with either a tendency to lie or a legal motive for wishing to have a fugue state substantiated.

E.F. was a 46-year-old man who described 12 to 15 episodes of 'going blank' during the previous 5 years. He said that these episodes lasted 2 to 36 hours, and that, on 'coming round', his feet were often sore, he was a long way from home, and he had no idea of the time or what had been happening during the previous hours. For example, he found himself on one occasion near the Thames, 10 miles from his home, with his clothes sopping wet. There was a history of epilepsy since he was 19, ECT and bilateral leucotomy at 33, major cardiac surgery, recurrent depression, and two serious suicide attempts. He had recently married for the third time: his wife was many years younger than himself and was pregnant. Marital difficulties were suspected, but were vehemently denied by both partners. Mr E.F. asked for a psychiatric report after being charged with driving whilst disqualified, without any insurance, and whilst under the influence of alcohol: his defence was that he had been in a fugue state.

Amnesia for offending

A common form of psychogenic amnesia with important implications is that claimed for a criminal offence. Most commonly, this has been described in association with homicide, in which it is claimed in 30–40% of cases (Table IV). A study in Broadmoor Hospital (Hopwood & Snell, 1933) and another in Brixton remand prison (Taylor & Kopelman, 1984) showed that amnesia also occurs in other violent crimes, although less commonly. In these studies, amnesia was found rarely or not at all in non-violent crime: this may have reflected the locations of the studies, but there is evidence that in violent crime recall by victims and eyewitnesses is also impaired. Taylor & Kopelman (1984) found that amnesia for an offence occurs in three types of circumstance:

(a) homicide cases in which the victim is closely related to the offender (a lover, wife, or family member), and the offence is unpremeditated and takes place in a state of high emotional arousal

TABLE IV

Homicide studies

	n	*% Amnesic*
Leitch (1948)	51	31
Guttmacher (1955)	36	33
O'Connell (1960)	50	40
Bradford & Smith (1979)	30	47
Taylor & Kopelman (1984)	34	26
Parwatikar *et al* (1985)[1]	105	23

[1] Pre-trial evaluations only; rate for convicted homicides not given.

(b) chronic alcohol abusers who commit a crime when severely intoxicated and whose victims are much less commonly related to them
(c) a small number of schizophrenic patients who commit criminal damage when floridly psychotic.

G.H. was a 40-year-old man, who had been born in Egypt, and was Jewish by religion. He had lived in England for 20 years, and was married to a 33-year-old Englishwoman. There was a son aged 9 and a daughter aged 3. The wife was having an affair with a musician, and G.H. was being treated for depression as an out-patient at a teaching hospital. He was taking an antidepressant and a benzodiazepine. On the day of the offence, the couple had a furious row during which G.H. threatened to kill the musician. His wife shared a bedroom with her daughter, and the last thing G.H. remembered was going to kiss the child goodnight. He reported that he could not remember what happened after that until the police arrived, but that he had been told that he phoned the police himself. He was charged with the murder of his wife by stabbing.

Depression and pseudodementia

A more persistent, psychologically based memory impairment is that occurring in a depressive pseudodementia, the clinical features of which have been described elsewhere. Memory impairment appears to be common in depression, although in the majority of cases it is relatively mild and poorly correlated with the severity of the depression (e.g. Kopelman, 1986*a*). The nature of the impairment in those cases which masquerade as 'dementia' is unclear, but one suggestion is that the disorder involves the more 'effortful' aspects of memory processes, and is closely associated with deficits in attention, motivation, and drive (Cohen *et al*, 1982). Affective disorder also gives rise to some particularly interesting state-dependent phenomena: depressives show a relative facility in recalling unpleasant events (Lishman, 1972; Lloyd & Lishman, 1975); this increased accessibility of unpleasant memories has a diurnal variation (Clark & Teasdale, 1982); and patients with mood swings show superior performance at memory tests if mood is consistent between learning and recall than if the mood changes (Weingartner *et al*, 1977).

Mechanisms in psychogenic amnesia

What mechanisms underlie psychogenic amnesia? One possibility is that many instances of psychogenic amnesia may result from faulty encoding of information at initial input (Kopelman, 1985*a*) and that deficits in retrieval may arise in consequence of this. In support of this view, it should be noted that psychogenic amnesia commonly occurs in states of abnormal mood or extreme arousal, in which normal cognitive processing might well be compromised. Fugues arise in the context of depressed mood and severe stress; amnesia for offences in situations of extreme emotional arousal, severe intoxication, or florid psychosis; and memory disorder in depression in association with impaired attention and motivation. This view, emphasising a disturbance in memory 'acquisition', suggests that the impairment in psychogenic amnesia mimics that in organic amnesia—of which there is so often an earlier history.

A second possibility is, of course, that psychogenic amnesia is an example of 'motivated forgetting' or repression. There is a considerable literature purporting to test the 'repression hypothesis' by showing that subjects tend to recall 'pleasant' events more readily than 'unpleasant' (e.g. Meltzer, 1930) and that this phenomenon cannot be accounted for by the relative affective intensity of the memories (Master *et al*, 1983). Although Freud (1915/17) wrote that "it is an undoubted fact that disagreeable impressions are easily forgotten", this kind of experimental study has often been criticised on the grounds that it fails to take account of the role of 'antagonistic impulses' in engendering repression—"an attempt at flight by the ego from libido which is felt as danger" (Freud, 1915/17). Experimental tests of repression in this sense tend to rely on very crude laboratory analogues, but Erdelyi & Goldberg (1979) have reviewed a number of concepts in contemporary cognitive psychology which appear to resemble repression. In this connection, the recent observations in organic amnesia of memory without awareness, and especially of preserved affective reactions in the presence of profound amnesia, are particularly intriguing.

A third possibility, strongly advocated by Bower (1981), is a (primary) retrieval deficit. Bower suggested that the 'discrete' psychogenic amnesias may reflect mood-state dependent phenomena, similar to those described in depression. According to this view, the experiences of a 'dissociative state' would be retrieved if the subject could be restored to the same subjective state in which they originally occurred. Fugue states and amnesia for offences are both associated with depressed mood and extreme states of arousal (Berrington *et al*, 1956; Taylor & Kopelman, 1984); and the latter are also associated with heavy abuse of alcohol in which state-dependent phenomena have commonly been described (Goodwin *et al*, 1969). Against this view, the state-dependent phenomena seen in laboratory experiments tend to be small and undramatic; mood disorder and alcohol abuse are common, whereas psychogenic amnesia is rare; and the only direct test of the hypothesis has failed to support it (Wolf, 1980).

Conclusion

This review has attempted to summarise the clinical features and the pattern of the memory dysfunction in selected examples of organic and psychogenic amnesia. It has also surveyed various theories which purport to explain the nature of the dysfunction.

It appears that the anterograde amnesia produced by structural lesions, including the widespread neuropathology of Alzheimer's disease, involves a severe impairment in acquiring (or learning) new information, and any retrieval deficits arise as a consequence of this. Contrary to clinical impression, the forgetting rate of new information is not accelerated, once adequate learning has been accomplished – although elderly people do show somewhat poorer retention of visuospatial material than younger subjects, and the metabolic disruption which follows ECT may produce accelerated forgetting. The precise deficit in psychological 'encoding' or physiological 'consolidation', which underlies this acquisition (or learning) deficit, remains to be elucidated. However, the simultaneous implication of two limbic circuits has been postulated as critical for the occurrence of anterograde amnesia, and it is known that these pathways contain cholinergic fibres. Future research is likely to clarify further the role of neurotransmitter depletion in producing amnesia.

Both psychological and pathophysiological evidence suggest the partial independence of the retrograde and anterograde components of amnesia. Retrograde amnesia may result from a number of factors, including a failure to make use of contextual cues (with regard to time, order, place, and source) in 'reconstructing' past experience. There is controversy as to where the critical lesions lie which produce retrograde amnesia, but there is evidence that frontal lesions disrupt the recall of contextual information. Moreover, two types of confabulation have been postulated, one of which ('provoked') may be a normal response to poor memory, but the other ('spontaneous') is characterised by the extremely incoherent (context-free) retrieval of memories and associations, and appears to reflect more extensive frontal pathology.

Finally, it has been postulated that many instances of psychogenic amnesia may resemble organic in that they result from the impaired acquisition of information at initial input, perhaps thereby predisposing the subject to subsequent retrieval difficulties in consequence of this. Such a view does not exclude the role of other possible mechanisms: organic amnesia provides a model for 'memory without awareness', and state-dependent phenomena may occur as occasional instances of retrieval difficulty.

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References

Abeles, M. & Schilder, P. (1935) Psychogenic loss of personal identity. *Archives of Neurology and Psychiatry*, **34**, 587–604.

Arendt, T., Bigl, V., Arendt, A. & Tennstedt, A. (1983) Loss of neurons in the nucleus basalis of Meynert in Alzheimer's diseases, Paralysis Agitans and Korsakoff's disease. *Acta Neuropathologica (Berlin)*, **61**, 101–108.

Baddeley, A. D. (1982) Domains of recollection. *Psychological Review*, **89**, 708–729.

——, Harris, J. E., Sunderland, A., Watts, K. & Wilson, B. (1987) Closed head injury and memory. In *Neurobehavioral Recovery from Head Injury* (ed. H. Levin). Oxford University Press.

—— & Wilson, B. (1986) Amnesia, autiobiographical memory and confabulation. In *Autobiographical Memory* (ed. D. Rubin). Cambridge: Cambridge University Press.

Bartlett, F. (1932) *Remembering*. London: Cambridge University Press.

Berlyne, N. (1972) Confabulation. *British Journal of Psychiatry*, **120**, 31–39.

Berrington, W. P., Liddell, D. W. & Foulds, G. A. (1956) A reevaluation of the fugue. *Journal of Mental Science*, **102**, 281–286.

Blass, J. P. & Gibson, G. E. (1977) Abnormality of a thiamine-requiring enzyme in patients with Wernicke-Korsakoff syndrome. *New England Journal of Medicine*, **297**, 1367–1370.

Bower, G. H. (1981) Mood and memory. *American Psychologist*, **36**, 129–148.

Bradford, J. & Smith, S. M. (1979) Amnesia and homicide: the Padola case and a study of thirty cases. *Bulletin of the American Academy of Psychiatry and the Law*, **7**, 219–231.

Brooks, N. (1984) Cognitive deficits after head injury. In *Closed Head Injury Psychological, Social and Family Consequences* (ed. N. Brooks) Oxford: Oxford University Press.

—— (1972) Memory and closed head injury. *Journal of Nervous and Mental Disease*, **155**, 350–355.

Butters, N. & Albert, M. (1982) Remote memory, retrograde amnesia, and the neuropsychology of memory. In *Human Memory and Amnesia* (ed. L. S. Cermak). Hillsdale, NJ: Lawrence Erlbaum.

Carr, A. C. (1982) Memory deficit after fornix section. *Neuropsychologia*, **20**, 95–98.

Claparede, E. (1911) Recognition and 'me-ness'. (Recognition et moiite) *Archives Psychologique, Geneve*, **11**, 79–90.

Clark, D. M. & Teasdale, J. D. (1982) Diurnal variation in clinical depression and accessibility of memories of positive and negative experiences. *Journal of Abnormal Psychology*, **91**, 87–95.

Cohen, R. M., Weingartner, H., Smallberg, S. A., Pickar, D. & Murphy, D. L. (1982) Effort and cognition in depression. *Archives of General Psychiatry*, **39**, 593–597.

Coriat, I. H. (1907) The Lowell case of amnesia. *Journal of Abnormal Psychology*, **2**, 93–111.

Cutting, J. (1978*a*) Relationship between Korsakoff's syndrome and "alcoholic dementia". *British Journal of Psychiatry*, **132**, 240–251.

—— (1978*b*) A cognitive approach to Korsakoff's syndrome. *Cortex*, **14**, 485–495.

De Wardener, H. E. & Lennox, B. (1947) Cerebral beriberi (Wernicke's encephalopathy): review of 52 cases in a Singapore P.O.W. hospital. *Lancet*, **i**, 11–17.

Erdelyi, M. H., Goldberg, B. (1979) Let's not sweep repression under the rug: toward a cognitive psychology of repression. In *Functional Disorders of Memory* (eds J. F. Kihlstrom & F. J. Evans). Hillsdale, NJ: Lawrence Erlbaum.

Freeman, C. P. L., Weeks, D. & Kendell, R. E. (1980) Electroconvulsive therapy. *British Journal of Psychiatry*, **137**, 8–37.

Freud, S. (1915/17) *Introductory Lectures*. Harmondsworth: Penguin.

Frith, C. D., Stevens, M., Johnstone, E. C., Deakin, J. F. W., Lawler, P. & Crow, T. J. (1983) Effects of ECT and depression on various aspects of memory. *British Journal of Psychiatry*, **142**, 610–617.

Gillespie, R. D. (1937) Amnesia. *Archives of Neurology and Psychiatry*, **37**, 748–764.

Goldberg, E., Hughes, J. E. O., Mattis, S. & Antin, S. P. (1982) Isolated retrograde amnesia: different aetiologies, same mechanisms? *Cortex*, **18**, 459–462.

Goodwin, D. W., Crane, J. B. & Guze, S. B. (1969) Phenomenological aspects of the alcoholic 'blackout'. *British Journal of Psychiatry*, **115**, 1033–1038.

Gudjonsson, G. H. & Taylor, P. J. (1985) Cognitive deficit in a case of retrograde amnesia. *British Journal of Psychiatry*, **147**, 715–718.

Guttmacher, M. S. (1955) *Psychiatry and the Law*. New York: Grune & Stratton.

Harper, C. (1983) The incidence of Wernicke's encephalopathy in Australia: a neuropathological study of 131 cases. *Journal of Neurology, Neurosurgery and Psychiatry*, **46**, 593–598.

Hierons, R., Janota, I. & Corsellis, J. A. N. (1978) The late effects of necrotizing encephalitis of the temporal lobes and limbic areas: a clinico-pathological study of 10 cases. *Psychological Medicine*, **8**, 21–42.

Hollander, E., Mohs, R. C. & Davis, K. L. (1986) Cholinergic approaches to the treatment of Alzheimer's disease. *British Medical Bulletin*, **42**, 97–100.

Hopwood, J. S. & Snell, H. K. (1933) Amnesia in relation to crime. *Journal of Mental Science*, **79**, 27–41.

Huppert, F. A. & Piercy, M. (1976) Recognition memory in amnesic patients: effect of temporal context and familiarity of material. *Cortex*, **12**, 3–20.

—— & Piercy, M. (1978) Dissociation between learning and remembering in organic amnesia. *Nature*, **275**, 317–318.

—— & Piercy, M. (1979) Normal and abnormal forgetting in organic amnesia: effect of locus of lesion. *Cortex*, **15**, 385–390.

—— & Kopelman, M. D. (1987) Rate of forgetting in normal ageing: a comparison with dementia. Submitted for publication.

James, W. (1890) *Principles of Psychology*, Vol. 1. New York: Holt.

Janis, I. L. & Astrachan, M. (1951) The effects of electroconvulsive treatments on memory efficiency. *Journal of Abnormal and Social Psychology*, **46**, 501–511.

Johnson, M. K., Kim, J. K. & Risse, G. (1985) Do alcoholic Korsakoff's syndrome patients acquire affective reactions? *Journal of Experimental Psychology, Language, Memory, Cognition*, **11**, 22–36.

Kanzer, M. (1939) Amnesia: a statistical study. *American Journal of Psychiatry*, **96**, 711–716.

Kapur, N. & Coughlan, A. K. (1980) Confabulation and frontal lobe dysfunction. *Journal of Neurology, Neurosurgery and Psychiatry*, **43**, 461–463.

——, Heath, P., Meudell, P. & Kennedy, P. (1986) Amnesia can facilitate memory performance: evidence from a patient with dissociated retrograde amnesia. *Neuropsychologia*, **24**, 215–222.

Kennedy, A. & Neville, J. (1957) Sudden loss of memory. *British Medical Journal*, **ii**, 428–433.

Kopelman, M. D. (1982) Speech dominance, handedness and electro-convulsions. *Psychological Medicine*, **12**, 667–670.

—— (1985*a*) Multiple memory deficits in Alzheimer-type dementia: implications for pharmacotherapy. *Psychological Medicine*, **15**, 527–541.

—— (1985*b*) Rates of forgetting in Alzheimer-type dementia and Korsakoff's syndrome. *Neuropsychologia*, **23**, 623–638.

—— (1986*a*) Clinical tests of memory. *British Journal of Psychiatry*, **148**, 517–525.

—— (1986*b*) Recall of anomalous sentences in dementia and amnesia. *Brain and Language*, **29**, 154–170.

—— (1986*c*) The cholinergic neurotransmitter system in human memory and dementia: a review. *Quarterly Journal of Experimental Psychology*, **38A**, 535–573.

—— & Lishman, W. A. (1986) Pharmacological treatments of dementia (non-cholinergic). *British Medical Bulletin*, **42**, 101–105.

Korsakoff, S. S. (1889) Psychic disorder in conjunction with peripheral neuritis. Translated and republished (1955) by M. Victor and P. I. Yakovlev. *Neurology*, **5**, 394–406.

Lawson, R. (1879) On the symptomatology of alcoholic brain disorders. *Brain*, **1**, 182–194.

Leigh, D., McBurney, A. & McIlwain, H. (1981) Erythrocyte transketolase activity in the Wernicke-Korsakoff syndrome. *British Journal of Psychiatry*, **139**, 153–156.

Leitch, A. (1948) Notes on amnesia in crime for the general practitioner. *Medical Press*, **219**, 459–463.

Lishman, W. A. (1968) Brain damage in relation to psychiatric disability after head injury. *British Journal of Psychiatry*, **114**, 373–410.

—— (1972) Selective factors in memory, 2: affective disorder. *Psychological Medicine*, **2**, 248–253.

—— (1987) *Organic Psychiatry: the psychological consequences of cerebral disorder*. 2nd ed. Oxford: Blackwell Scientific. (In press).

Lloyd, G. C. & Lishman, W. A. (1975) Effect of depression on the speed of recall of pleasant and unpleasant experience. *Psychological Medicine*, **5**, 173–180.

Luria, A. R. (1969) *The Mind of a Mnemonist*. London: Jonathan Cape.

—— (1976) *The Neuropsychology of Memory*. New York: John Wiley.

Lynch, S. & Yarnell, P. R. (1973) Retrograde amnesia: delayed forgetting after concussion. *American Journal of Psychology*, **86**, 643–645.

Mair, W. G. P., Warrington, E. K. & Weiskrantz, L. (1979) Memory disorder in Korsakoff's psychosis: a neuropathological and neuropsychological investigation of two cases. *Brain*, **102**, 749–783.

Malamud, N. & Skillicorn, S. A. (1956) Relationship between the Wernicke and Korsakoff syndrome. *Archives of Neurological Psychiatry*, **76**, 585–596.

Markowitsch, H. J. (1984) Can amnesia be caused by damage of a single brain structure? *Cortex*, **20**, 27–45.

Martin, P. R., Weingartner, H., Gordon, E. K., Burns, R. S., Linnoila, M., Kopin, I. J., Ebert, M. H. (1984) Central nervous system catecholamine metabolism in Korsakoff's psychosis. *Annals of Neurology*, **15**, 184–187.

Master, D., Lishman, W. A. & Smith, A. (1983) Speed of recall in relation to affective tone and intensity of experience. *Psychological Medicine*, **13**, 325–331.

Mayes, A. R., Meudell, P. R. & Pickering, A. (1985) Is organic amnesia caused by a selective deficit in remembering contextual information? *Cortex*, **21**, 167–202.

McDowall, J. (1981) Effects of encoding instructions on recall and recognition in Korsakoff patients. *Neuropsychologia*, **19**, 43–48.

McEntee, W. J., Biber, M. P., Perl, D. P. & Benson, D. F. (1976) Diencephalic amnesia: a reappraisal. *Journal of Neurology, Neurosurgery and Psychiatry*, 39 436–441.

——, Mair, R. G. & Langlais, P. J. (1984) Neurochemical pathology in Korsakoff's psychosis: implications for other cognitive disorders. *Neurology*, 34, 648–652.

Meltzer, H. (1930) Individual differences in forgetting pleasant and unpleasant experience. *Journal of Educational Psychology*, 21, 399–409.

Merskey, H. & Woodforde, J. M. (1972) Psychiatric sequelae of minor head injury. *Brain*, 95, 521-528.

Meudell, P. R., Mayes, A. R. & Neary, D. (1979) Is amnesia caused by a consolidation impairment? In *Research in Psychology and Medicine* (eds D. J. Osborne, M. M. Gruneberg & J. R. Eiser). London: Academic Press.

Miller, E. (1973) Short- and long-term memory in patients with presenile dementia (Alzheimer's disease). *Psychological Medicine*, 3, 321–324.

Milner, B. (1966) Amnesia following operation on the temporal lobes. In *Amnesia* (eds C. W. M. Witty & O. Zangwill). London: Butterworth.

Mishkin, M. (1978) Memory in monkeys is severely impaired by combined but not by separate removal of amygdala and hippocampus. *Nature*, 273, 297–298.

Moll, J. M. (1916) The amnesia of Korsakoff's syndrome with alcoholic aetiology: an analysis of 30 cases. *Journal of Mental Science*, 61, 423–437.

Morris, R. G. & Kopelman, M. D. (1986) The memory deficits in Alzheimer-type dementia: a review. *Quarterly Journal of Experimental Psychology*, 38A, 575–602.

Moscovitch, M. (1982) Multiple dissociations of function in amnesia. In *Human Memory and Amnesia* (ed. L. S. Cermak). Hillsdale, NJ: Lawrence Erlbaum.

O'Connell, B. A. (1960) Amnesia and homicide. *British Journal of Delinquency*, 10, 262–276.

Owen, M. J. & Butler, S. R. (1981) Amnesia after transection of the fornix in monkeys: long-term memory impaired, short-term memory intact. *Behavioural Brain Research*, 3, 115–123.

Parfitt, D. N. & Gall, C. M. C. (1944) Psychogenic amnesia: the refusal to remember. *Journal of Mental Science*, 90, 511-527.

Parwatikar, S. D., Holcomb, W. R. & Menninger, K. A. (1985) The detection of malingered amnesia in accused murderers. *Bulletin of the American Academy of Psychiatry and Law*, 13, 97–103.

Russell, W. R. & Nathan, P. W. (1946) Traumatic amnesia. *Brain*, 69, 280–300.

—— & Smith, A. (1961) Post-traumatic amnesia in closed head injury. *Archives of Neurology*, 5, 4–29.

Sargant, W. & Slater, E. (1941) Amnesic syndromes in war. *Proceedings of the Royal Society of Medicine*, 34, 757–764.

Schacter, D. L., Wang, P. L., Tulving, E. & Freedman, M. (1982) Functional retrograde amnesia: a quantitative case study. *Neuropsychologia*, 20, 523–532.

Scoville, W. B. & Milner, B. (1957) Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery and Psychiatry*, 20, 11–21.

Shimamura, A. P. & Squire, L. R. (1984) Paired-associate learning and priming effects in amnesia: a neuropsychological study. *Journal of Experimental Psychology: General*, 113, 556–570.

Speedie, L. J. & Heilman, K. M. (1982) Amnesic disturbance following infarction of the left dorsomedial nucleus of the thalamus. *Neuropsychologia*, 20, 597–604.

Squire, L. R. (1977) ECT and memory loss. *American Journal of Psychiatry*, 134, 997–1001.

—— (1980) Specifying the defect in human amnesia: storage, retrieval, and semantics. *Neuropsychologia*, 18, 368–372.

—— (1981) Two forms of human amnesia: an analysis of forgetting. *Journal of Neuroscience*, 1, 635–640.

—— (1982) Comparisons between forms of amnesia: some deficits are unique to Korsakoff's syndrome. *Journal of Experimental Psychology, Learning, Memory, Cognition*, 8, 560–572.

—— & Moore, R. Y. (1979) Dorsal thalamic lesion in a noted case of human memory dysfunction. *Annals of Neurology*, 6, 503–506.

—— & Slater, P. C. (1983) ECT and complaints of memory dysfunction a prospective three-year follow-up study. *British Journal of Psychiatry*, 142, 1–8.

——, Cohen, N. J. & Nadel, L. (1984) The medial temporal region and memory consolidation: a new hypothesis. In *Memory Consolidation* (eds H. Weingartner & E. Parker). Hillsdale, NJ: Erlbaum Associates.

Starr, A. & Phillips, L. (1970) Verbal and motor memory in the amnesic syndrome. *Neuropsychologia*, 8, 75–88.

Stengel, E. (1941) On the aetiology of the fugue state. *Journal of Mental Science*, 87, 572–599.

Stuss, D. T., Alexander, M. P., Lieberman, A. & Levine, H. (1978) An extraordinary form of confabulation. *Neurology*, 28, 1166–1172.

Taylor, P. J. & Kopelman, M. D. (1984) Amnesia for criminal offences. *Psychological Medicine*, 14, 581–588.

Teasdale, G. & Mendelow, D. (1984) Pathophysiology of head injuries. In *Closed Head Injury: Psychological, Social, and Family Consequences* (ed. N. Brooks). Oxford: Oxford University Press.

Tomlinson, B. E. & Corsellis, J. A. N. (1984) Ageing and the dementias. In *Greenfield's Neuropathology* (eds J. H. Adams, J. A. N. Corsellis & L. W. Duchen) 4th ed. London: Edward Arnold.

Torvik, A., Lindboe, C. F. & Rogde, S. (1982) Brain lesions in alcoholics. *Journal of Neurological Science*, 56, 233–248.

Tulving, E. (1972) Episodic and semantic memory. In *Organisation of Memory* (eds E. Tulving & W. Donaldson). New York and London: Academic Press.

Victor, M., Adams, R. D. & Collins, G. H. (1971) The Wernicke-Korsakoff Syndrome. Philadelphia: F. A. Davis Co.

Warrington, E. K. & Weiskrantz, L. (1973) Short- and long-term memory defects in man. In *The Physiological Basis of Memory* (ed. J. A. Deutsch). London: Academic Press.

—— & Weiskrantz, L. (1978) Further analysis of the prior learning effect in amnesic patients. *Neuropsychologia*, 16, 169–177.

Weigartner, H., Miller, H. & Murphy, D. C. (1977) Mood-state-dependent retrieval of verbal associations. *Journal of Abnormal Psychology*, 86, 276–284.

——, Grafman, J., Boutelle, W., Kaye, W. & Martin, P. R. (1983) Forms of memory failure. *Science*, 221, 380–382.

Weiskrantz, L. & Warrington, E. K. (1979) Conditioning in amnesic patients. *Neuropsychologia*, 17, 187–194.

Wilson, G., Rupp, C. & Wilson, W. W. (1950) Amnesia. *American Journal of Psychiatry*, 106, 481–485.

Wilson, R. S., Kaszniak, A. W. & Fox, J. H. (1981) Remote memory in senile dementia. *Cortex*, 17, 41–48.

Wolf, A. S. (1980) Homicide and blackouts in Alaskan natives. *Journal of Studies of Alcohol*, 41, 456–462.

Woods, R. T. & Piercy, M. (1974) A similarity between amnesic memory and normal forgetting. *Neuropsychologia*, 12, 437–445.

Zangwill, O. L. (1953) Disorientation for age. *Journal of Mental Science*, 99, 698–701.

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Lecture

Temperament, Personality and Personality Disorder

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It is now over 2000 years since Hippocrates put forward his idea that there were four body 'humours', each associated with a characteristic personality style: black bile with melancholic; blood with sanguine; yellow bile with choleric; and phlegm with phlegmatic (Mora, 1980). The proposition that human beings are constitutionally different in ways that shape their personality style caught on with the lay public. Indeed the notion of body humours determined the very words that we use to describe such styles. Yet the view has not had such a smooth journey in scientific circles. There is a surprising lack of uniformity in both concept and terminology, as is all too evident in the controversies surrounding the terms 'temperament', 'personality' and 'personality disorder'.

Paradoxically, personality psychology went out of fashion just at the time that the study of temperament was becoming firmly established. But the recent upsurge of research and of writing on temperament has not brought general agreement. Kagan *et al* (1987) see temperament in terms of those behavioural styles that have a strong biological basis; somewhat similarly Buss & Plomin (1986) defined temperament as inherited personality traits present in early childhood. By contrast, Bates (1980) suggested that much of temperament lay in parental percepts rather than child behaviour; Mischel (1968) argued that situational influences were so strong that the role of personality traits could be ignored; Stevenson-Hinde & Hinde (1986) accepted that there is an individual component to behaviour but pointed out that most behaviour as observed is a property of social interactions rather than just one person; and Graham & Stevenson (1986) maintained that temperament represents only subclinical manifestations of psychiatric disorder. Where does the truth lie? What value have these concepts for clinical practice if their meaning and basis is in such dispute? My purpose in this paper is to attempt some clarification of the rather complex mesh of ideas that fall under the general rubric of temperament, personality and personality disorder. In so doing, I shall seek to provide tentative answers to these difficult questions and, more hesitantly, I shall suggest some possible ways forward.

Temperament

The modern study of child temperament can be said to have begun with the New York Longitudinal Study initiated in the 1950s by Thomas *et al* (Thomas *et al*, 1963; Thomas & Chess, 1977; Chess & Thomas, 1984). The themes of current interest in temperament (Plomin, 1983) reflect those emphasised in that pioneering investigation. These include a focus on: 1) individual differences in development rather than normative trends; 2) the child as an active agent who shapes his own environment as well as being influenced by his circumstances; 3) possible neurobiological bases for behaviour, including genetic influences; and 4) socioemotional, rather than cognitive, aspects of development. Of course, their work should not be seen in isolation. It had its roots in Pavlov's (1927) typology of nervous systems, in Sheldon's (1942) study of body types and temperament, in Lacey & Lacey's (1958*a,b*) psychophysiological investigations and in Gesell's (1937) earlier studies of children's behavioural attributes, to mention but a few examples.

Before considering some of the controversial issues surrounding the concept of temperament, it is important to draw attention to some of the many matters that are no longer controversial because there is ample supporting empirical evidence. First, it is abundantly clear that individual children differ markedly from one another in a host of different ways that can be measured with reasonable reliability (Buss & Plomin, 1975; Berger, 1985; Dunn, 1980; Campos *et al*, 1983). This has been shown, for example, with features as varied as activity level, autonomic reactivity, behavioural inhibition, and sociability. Second, it has been well demonstrated that these differences *matter* in terms of their implications for later development and for psychiatric risk.

As the psychopathological importance of temperament rests on that point it may be as well to spell out some of the main findings. To begin with, it has been found that interview measures of temperament predict the later development of emotional and

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behavioural disorders in both 'normal' (Rutter *et al*, 1964; Thomas & Chess, 1977; Dunn & Kendrick, 1982; Kagan *et al*, 1987) and high risk samples (Graham *et al*, 1973; Wolkind & De Salis, 1982). Similar findings derive from studies using questionnaire measures (Bates *et al*, 1985; Maziade *et al*, 1985); these have also shown an association between 'difficult' temperamental features and a higher rate of accidents, sleep difficulties and of infantile colic (Huttunden & Nyman, 1982; Carey, 1986). There are good reasons for accepting the validity of these findings. Thus, some of the associations between temperament and disorder have spanned quite long time periods; 5 years in the study by Maziade *et al* (1985). Dunn & Kendrick (1982) found that temperamental characteristics were more strongly associated with an *increase* in emotional disturbance following the birth of a sibling than with emotional disturbance at the time of the temperamental assessment prebirth. Also, Graham *et al* (1973; Rutter, 1978) found that temperamental features (measured by parental interview) predicted behavioural deviance at school a year later. In other words, the children's temperament has been found to predict *changes* of behaviour in different contexts at a later date.

The third topic that is no longer controversial is relevant to the matter of psychiatric risk; namely that children's temperamental features influence the ways in which *other* people respond to them. It may suffice to give just a few examples of findings in this area. Thus, Lee & Bates (1985) found that temperamentally difficult infants elicited more confrontations with their mothers as well as a greater tendency for these to result in conflict. We (Rutter, 1978) found that children with adverse temperamental features were more likely to be the target of parental criticism. Stevenson-Hinde & Hinde (1986) noted that children with more negative mood were more likely to have mothers who were irritable and teachers who were reactively hostile. It might be suggested that these associations reflected the impact of others on the child rather than the reverse. Of course, effects are likely to be bidirectional but several sets of data show the importance of child effects. First, this is evident from clinical studies in which beneficial drug effects on children's hyperactivity have been followed by changes in parental behaviour (Barkley, 1981). Second, it has been demonstrated by experimental investigations in which children have been trained to behave in contrasting ways (Brunk & Henggeler, 1984). Third, it is implicit in the differences in parental behaviour associated with organically determined child handicaps (Cross *et al*, 1985).

All of this adds up to a strong case for the clinical importance of individual differences in children's temperamental attributes. However, it is necessary to go on to consider just what is this temperament that has clinical implications. The first point to underline is that, necessarily, temperamental characteristics are abstractions, rather than directly observable discrete behaviours (Rutter *et al*, 1964). This is so for several somewhat different reasons. Most measures of temperament rely on someone's ratings of the children's behaviour. Inevitably, such ratings are bound to be influenced by errors and biases in the perceptions and attributions of the person making the rating. There is good reason to suppose that such biases may be present. For instance, it has been found that adults, when presented with a wrongly labelled, ambiguously dressed crying infant, are more likely to attribute anger if told that the infant is a boy and fear if a girl (Condry & Condry, 1976; Haviland & Malatesta, 1981). Nevertheless, the relatively good agreement between ratings and directly observed discrete behaviours (Bates, 1986; Dunn & Kendrick, 1980) are reassuring that the measures reflect far more than biased perception. Indeed, Bates (1980) who first raised the bias issue with respect to the measurement of 'difficult' temperament has now concluded that temperament measures reflect both objective and subjective factors (Bates & Bayles, 1984).

A further problem is that observations and ratings have to be based on the situations in which the child has been present with the rater or observer. This is relevant because it is known that people's behaviour is influenced by the situations in which they find themselves (Mischel, 1979; Rutter & Giller, 1983). Behaviour as observed is bound to reflect *both* personal traits and situation-specific reactions. As an extension of the same point, behaviour in a social context (and most temperamental features are ones that are evident in social situations) is bound to reflect the properties of that context, including the temperamental traits of the *other* person as well as influences stemming from the history of previous interactions (Asher, 1983; Dodge, 1980). As I have noted already, temperament is an abstraction, not an observable variable. Of course, there are ways and means of getting closer to that abstraction. The use of multiple measuring devices and multiple situations provides a good means of overcoming all three sources of measurement distortion (Epstein, 1979, 1983). Moreover, structural equation modelling is a statistical procedure that enables one to study 'latent' variables (Duncan-Jones *et al*, 1986). The requirement simply is to specify just what is conceptualised as the latent variable. Thus, one could specify that it is that aspect of the behaviour that is pervasive over situations and consistent over time, once measurement errors have been excluded.

Therein lies the difficulty, however. At first it seems obvious that some definition of that sort must be what one wants. But is it? Let us take emotional reactivity as an example of a temperamental attribute that is included by virtually all researchers and which shows both substantial heritability and stability over time (Buss & Plomin, 1975). Suomi's (1983) studies of rhesus monkeys show clearly that individual differences in autonomic reactivity remain moderately consistent over the period from infancy to puberty, but *only* when measured in stressful situations. In other words, it has required the 'press' of particular situations to bring out an important temperamental trait. It would be seriously misleading to suppose that personal traits have to be pervasive over all situations.

Kagan's human data (Kagan *et al*, 1986, 1987; Reznick *et al*, 1986) bring out two other crucial points. First, the behavioural manifestations of the same trait change over the course of development. Behavioural inhibition in an infant may be evident in the seeking of proximity to mother, but in a school-age child by a particular style of social interaction and a cautious approach to new tasks. Also the situations required to elicit inhibition change as the tasks that are meaningful to the child also alter with developmental progression. The very process of development makes it unlikely that an underlying predisposition will be expressed in the same way throughout the whole of childhood (Campos *et al*, 1983). Moreover, the finding of a similar factor structure at different ages does not mean that the behaviours have the same meaning, as the data on type A behaviours illustrate (Steinberg, 1986).

Second, composites or constellations of behaviour may be more meaningful than single dimensions considered individually. This is *not* just a matter of broad measures being better than narrow ones or of factors being superior to the components of the factor. Rather, it is an indication that in some circumstances, the association of two behaviours may change their meaning. For example, Kagan *et al* (1987) found that their heart rate measures (treated in dimensional terms) added nothing to the variance accounted for by their behavioural measure of inhibition. But, the subgroup of children with both behavioural inhibition *and* a high stable heart rate were strikingly different from the remainder. Magnusson (1987) noted the same with respect to the role of autonomic measures in relation to aggressivity. However, it should not be thought that the function necessarily resides in the combination of physiological and behavioural measures. Magnusson (1987) found that it also applied to the combination of the two behavioural measures of restlessness and aggressivity.

Where do these considerations leave us? Two related points need to be discussed: 1) what sort of concept do we want to be covered by the term 'temperament'?; and 2) depending on the answer to that question, which traits or qualities should be included within the concept? Of course, that question can only be answered by considering which qualities (and why) should *not* be included.

It is most appropriate to start with the views that suggest that temperament should not be separately conceptualised at all; rather it should be seen as a mini-disorder (Stevenson & Graham, 1982; Graham & Stevenson, 1986). The main rationale for this argument is that the factor structure of behavioural deviance in the general population closely parallels that in clinical groups, that most child psychiatric disorders represent quantitative rather than qualitative departures from normality, and that some of the items in temperamental scales sound like lesser variants of those in psychiatric measures (high activity level or high emotional reactivity would be examples). The value of studying the behavioural transitions from normality to abnormality can certainly be accepted (Rutter, 1986*a*; 1987*a*) but still the equation of temperament with psychiatric disorder must be rejected. First, extremes of temperamental traits do not in themselves constitute disorder. Second, most of the symptoms of child psychiatric disorder are not part of temperamental concepts (this would be so, for example, with antisocial behaviour, specific phobias, encopresis, or feelings of guilt or hopelessness). Third, the patterns of correlations with *change* in behaviour rather than with current disorder (see above) do not fit easily with the hypothesis. Fourth, the suggestion bypasses the evidence on the indirect path by which temperament may lead to disorder via influences on other people's reactions. Fifth, the notion fails to take account of the repeated finding that the effects of temperament may vary by sex. For example, Buss (1981) found that high activity was associated with positive interactions between fathers and sons but negative interactions between fathers and daughters. Conversely, Stevenson-Hinde & Hinde (1986) found that shyness in boys was associated with largely negative interpersonal interactions whereas in girls the correlates were largely positive. It seems that the extreme of a trait often does not lead directly to psychopathology, rather its effects are dependent on the social meaning that is given to the behaviour. Finally, the importance of temperament does not lie just in psychiatric risk; moreover the same trait may carry both positive and negative implications according to circumstances. For example, high activity proved protective to infants raised in a poor institution (Schaffer, 1966) and a 'difficult' temperament was protective to Masai infants during the East African famine (De Vries, 1984) - presumably in both cases because they demanded and received more attention and/or food. In other situations, however, high activity and a difficult temperament are psychiatric risk factors. It is not helpful to view temperament as subclinical psychiatric disorder; that constitutes an unacceptable broadening of the concept of disorder and fails to account for key empirical findings. The issue, however, is one to which I must return when discussing personality disorder.

The more usual concept of temperament is exemplified by Buss & Plomin's (1986) suggestion, following the tradition of Allport (1937), that it forms a subclass of personality traits that emerge early in life and which show high heritability. Previously, stability over time had also been regarded as an essential feature (Buss & Plomin, 1975; Plomin, 1983). Like McCall (1986) I find that definition equally unsatisfactory. Much of behaviour has a substantial genetic component, appears early and shows a reasonable degree of consistency over time, at least after infancy (and temperamental traits correlate very weakly from infancy to middle or later childhood). Moreover, as Plomin (1986) has pointed out elsewhere, heritability for most characteristics tends to rise with age, typically being very low in the early infancy period. Also, it is evident that traits that are genetically distinct may lead to a single phenotype as a result of environmental effects (Martin *et al*, 1979). The definition both requires too much and too little. Its demands are too high in expecting pervasiveness over situations and stability over time. As already noted, some traits require particular environments in order that they be manifest. Also, it must be expected that behaviour will change with development. I've already commented on the fact that the manifestations of behavioural inhibition do so. Its requirements with respect to heritability are unhelpful in being non-discriminating. It is not suggested that temperament is insusceptible to environmental influences; indeed no human characteristic is. Thus, height is a characteristic with very high heritability but yet the effects of nutrition are considerable (Tizard, 1975). Moreover, which human behaviour does *not* have a substantial genetic component? That does not seem to differentiate temperament in theory or in practice; empirically the heritability of temperamental traits is not particularly high in comparison with that of other behaviours. The key requirement is to decide how to separate temperament from other aspects of personality or of human function more generally.

The New York group argued that temperament represented the *how* of behaviour, that is the style as distinct from the *why* or motivational component and the *what* of content or capacity. As Graham & Stevenson (1986) noted, the how-what differentiation is not easy to apply in practice as people's behavioural styles lead them to choose one activity over another (the highly active child is more likely to be found on the football field or up a tree than in the library!). Nevertheless, the distinctions seem helpful in excluding from temperament behaviours that are defined in terms of content (thus emotional reactivity is included but specific fears are not) or of motivation or cognitive components (so that, negative mood is allowable but helplessness or depression is not). There is also an implicit focus on simple stylistic, non-motivational, non-cognitive features rather than complex behavioural tendencies. Activity level is included but obsessionality, generosity and suspiciousness are not.

Many theorists have also suggested that there should be a fairly direct connection with neurobiological processes. For example, Kagan *et al* (1987) suggest that their characteristic of behavioural inhibition is a function of a low threshold of responsivity in limbic and hypothalamic structures. In support, they point to the systematic associations with autonomic reactivity and cortisol levels. While this theoretical approach has not been typical of researchers in the field of child temperament, it has been characteristic of many personality theorists in the adult field. Moreover, it is striking that they tend to focus on much the same set of traits. Thus Buss & Plomin (1986) argue for three main temperamental traits: emotionality, activity and sociability, with uncertainty over a possible fourth, impulsivity. The first letters of the traits form the acronym EASI. While not identical, these appear closely comparable to Eysenck's (1967, 1982) neuroticism, extraversion and psychoticism; to Tellegen's (1985) negative and positive emotionality and constraint; to Gray's (1983) behavioural inhibition, impulsivity and fight/flight; and Cloninger's (1987) harm avoidance, novelty seeking and reward dependence. Cloninger (1987) has recently extensively reviewed the evidence on the neurobiological correlates of these traits and argued that variation in each dimension is strongly related to activity in a specific central monoaminergic pathway: harm avoidance with high serontonergic activity, novelty seeking with low basal dopaminergic activity and reward dependence with low basal noradrenergic activity. There will be debate on both the specific ways the traits are defined and on the specific neurobiological correlates. Nevertheless, it can be accepted that a good case has been made out for some kind of correlates, and for a small set of traits that could reasonably be regarded as temperamental in terms of the concepts discussed here. Furthermore, there seem to be no good reasons for regarding them as part of temperament in childhood but personality in adult life.

Several further issues require to be addressed, however. First, there is the question of whether the traits are best conceptualised in dimensional or categorical terms. Both Kagan *et al* (1987) and Magnusson (1987) have noted that at least some traits may be better dealt with in terms of physio-behavioural composites that are qualitatively distinct rather than a continuum. The matter has been too little explored up to now for any decision on the matter. What may be said is that we would be unwise unquestionably to assume that traits have to be dimensional.

The second major issue concerns what needs to be left out of temperament if the traits are to be conceptualised as simple, non-motivational, non-cognitive stylistic characteristics. I have already noted that this approach rules out most of the personality traits that appear in adult psychiatric classifications - such as schizoid, paranoid, histrionic and obsessional. However, also it raises questions about some characteristics that have played a prominent part in child temperament research. For example, should 'difficultness' be accepted as a temperamental feature? As already discussed, it has predictive validity but is it temperament? The term is unfortunate as it suggests that the concept is defined in terms of other people's reactions. However, that is misleading because, in fact, it is based on a constellation of child behaviours. It remains uncertain how it fits into the concept of temperament. If it can be defined in terms of a composite of simple behavioural styles, if that composite has substantial neurobiological correlates, and if there is substantial consistency over time perhaps it should be classed as a temperamental trait. The fact that it is a composite should not be a reason for exclusion. However, if its unity lies only or mainly in terms of other people's reactions that would seem to rule it out. The evidence is not enough to enable any decision (but see Korn, 1984).

The second trait that raises queries is aggressivity; in this case because most temperamental researchers leave it out rather than because it is included. Of course, it sounds as if motivational components are inherent but are they? Is it a derivative of the temperament traits noted already or is it a trait in its own right? If stability over time is used as a criterion for temperament, aggresivity must rank as a serious contender, as Olweus' (1979) review of the evidence indicates. Buss & Plomin (1975) ruled it out first of all on the lack of evidence for high heritability. However, it is not that there are data showing a low genetic component, rather there is little evidence one way or the other. However, such evidence as there is shows substantial heritability (Rushton *et al*, 1986). Buss & Plomin (1975) also argue that aggressiveness can be explained in terms of the combination of three temperamental features: activity, emotionality and impulsivity. If that were so, it would be a reason for rejecting aggressivity as a simple, stylistic temperamental attribute but, once more the necessary evidence is lacking.

Where does that leave the concept of temperament? We may conclude that there are strong grounds for assuming the validity of the general notion that there are simple, non-motivational, non-cognitive, stylistic characteristics that represent meaningful ways of describing individual differences between people. For the most part, such differences appear early in childhood, show substantial stability over time after the pre-school years (although the particular behavioural manifestations may change), represent predictable modes of response (although these may require particular situations to bring them out), and possibly have fairly direct neurobiological correlates. As used in this way, the term temperament seems as applicable to adult traits as to child characteristics. There is continuing debate over the best way of conceptualising such basic traits but there is general agreement that some variant on the features of emotionality, activity and sociability is needed. There may be other traits (such as impulsivity or aggressivity) and also it may be that some traits are better viewed in categorical terms rather than dimensional ones. Finally, it is possible that some behavioural composites should be included. The 'difficultness' composite remains a contender but there is continuing uncertainty on the unity of the concept as a biological construct. It should be added that, of course, there are many aspects of human individuality that are of considerable clinical and developmental interest but yet which can not be included in temperament. For example, it has been shown that names and physical appearance have psychological significance for people's own behaviour and other people's reaction to them (Rutter, 1977).

Personality

This point raises the broader issue of how personality differs from temperament. Hall & Lindzey (1978) defined personality as . . . "that which gives order and congruence to all the different kinds of behaviour in which the individual engages". Thus, like Sroufe (1979) they placed emphasis on integrative and organising functions and, by implication, on cognitive and motivational as well as dispositional components. It is these non-behavioural aspects that provide the clearest differentiation from temperament. Both agree in their focus on consistency in styles of behaviour, but personality differs in its inclusion of coherences that stem from attitudinal features, from patterns of thought, and from motivational factors as well as from simple dispositional attributes. Personality differs, also, in its concern with meaningful patterns of behaviour that may be evident in a *lack* of behavioural generalisation across situations. For example, Dunn & Kendrick (1982) found that girls who had an intense, warm playful relationship with their mothers were more likely to have a poor relationship with their siblings. Instead of a generalisation of warm relationships, jealousy led to an *inverse* association. Somewhat similarly, Sroufe *et al* (1985) argued that maternal seductiveness provided the organising function for an extreme form of markedly different reactions to sons and daughters. It is evident that there is much more to personality development than just the increasing stabilisation of generalised temperamental traits. However, this 'more' stems from the role of non-behavioural components and not simply from the force of environmental influences. There is no reason to suppose that temperament is immune from environmental shaping; equally, there is every reason to assume that heritable factors play some part in the shaping of attitudes and expectations as well as behavioural traits (Scarr & Kidd, 1983). Indeed, the various components of personality, at least as usually measured, differ surprisingly little in the extent to which they are heritable (Loehlin & Nichols, 1976; Plomin, 1986).

If we accept the need for a concept of personality that is much broader than a collection of temperamental traits, it is necessary to go on to ask how different aspects of personality are best dealt with. Allport (1937), one of the most influential personality theorists, referred to dynamic organisation, a phrase that emphasised both an active mental role and also the importance of developmental changes. Personality, in a sense, is the patterning that each person, as a thinking being, develops as a way of dealing with the traits with which he is endowed, the social contexts that he encounters, and the experiences he has received. For Allport, adult personality encompassed attitudes to the self (including elements such as acceptance, humour and insight), a style of relating to others, projections into the future (as evident in hoping and planning) and an implicit philosophy of life. His ideas have much in common with contemporary theorising on the development of self-concepts (Harter, 1983). Murray (Murray *et al*, 1938; Murray & Kluckhohn, 1953) similarly conceived of personality as an organising and integrating force, a force that exhibited both coherence and flux, and which grew out of life experiences as well as basic needs. As he put it, the history of the personality is the personality.

These general notions may be accepted as reasonable guiding principles but, in themselves, they lack specificity and predictive power. Various theoretical approaches have sought to provide such specificity. Psychoanalysis constitutes the key example, of course. It opened our eyes to the richness of the workings of the human mind, to the crucial importance of the meanings attached to experiences, and to the role of intrapsychic processes. Many of these ideas have become part of the currency of contemporary theories of personality functioning (Rutter, 1986*b*, 1987*b*). But, equally, many of the key tenets of psychoanalysis have had to be abandoned and others stand in need of radical overhaul (Eagle, 1984; Dare, 1985). It is doubtful whether, as a theory of personality, it is very helpful today. Some would argue for the utility of types of defence mechanisms as a means of classifying personality (Vaillant, 1977, 1983) but the evidence seems unconvicing. The maturity of defences are most strongly correlated with IQ and feelings of adequacy and there is major overlap with general measures of functioning. That people do indeed employ coping mechanisms (of which defences are a variety) in dealing with intrapsychic and interpersonal problems is not in doubt (Rutter, 1981; Moos, 1986); also it is likely that the coping process may play a part in both the onset and course of psychological disturbances following stress or adversity (Parker *et al*, 1986). What is much more uncertain is whether particular coping styles constitute a stable and pervasive aspect of personality functioning and whether they provide a useful means of classifying personality types or processes.

Probably three main approaches receive most attention now: 1) concepts of attachment and relationships; 2) cognitive processing; and 3) personality traits. Humans are social animals and it is clear that social relationships play a crucial role in most activities. The loss of a love relationship constitutes a powerful stressor throughout the lifespan and the presence of close confiding relationships provide important emotional support at all ages (Wolkind & Rutter, 1985). In recent years, stimulated especially by Hinde's (1979; Hinde & Stevenson-Hinde 1986) writings, and by the study of peer relationships in childhood (Hartup, 1983; Hartup & Rubin, 1986) there has been interest in frameworks to describe relationships. It has been argued that studying the origins of personality and the origins of social relations is one and the same task (Sroufe & Fleeson, 1986). Particular attention has been paid to the role of early parent-child attachments in the development of later relationships (Bretherton & Waters, 1985). It is not suggested, of course, that relationships should be viewed as a consistent personality trait. The same individual may have quite different relationships with different people; relationships show reciprocity and complementarity as well as generalisation. Nevertheless, it has been suggested that relationships are in some sense internalised and carried forward in ways that influence later relationships. Sroufe & Fleeson (1986) argued that this influence may arise through several routes, including: 1) the internal organisation of needs, attitudes, beliefs and styles of coping – that is the features that give rise to coherence of personality functioning; 2) expectations about relationships; and 3) understanding of relationships and skills in social encounters. Most attention has been paid to the categorisation of early attachment relationships according to types of security and insecurity. There is now a body of evidence showing that the security classification in infancy is associated with predictable patterns of peer relationships over the next few years and, less certainly, with consequences reaching very much further ahead (Bretherton & Waters, 1985). In part these continuities are likely to reflect environmental consistencies over the childhood years but, at least in the case of severely constrained attachment opportunities as in an institutional up-bringing, this does not constitute the whole explanation (Rutter, 1987*c*). Hodges & Tizard (1987*a,b*) found that late adopted children still maintained a distinctive social style at 16 years that seemed to be a function of institutional caretaking in the early years, in spite of a radical change in environment.

The mechanisms involved in these continuities in relationship are not fully understood. The concept of an 'internal working model' of relationships has been put forward as an explanatory variable (Bowlby, 1969/82; Bretherton & Waters, 1985). The general idea is that children derive a set of expectations about their own relationship capacities and about other people's response to their social overtures and interactions, these expectations being created on the basis of their early parent–child attachments. Thus, to some extent, children shape their later relationships as a result of cognitive concepts derived from earlier relationships. But, in turn, later relationships serve to modify the working model. That is, it is supposed that there is both coherence and fluidity; a substantial degree of consistency but not a rigidity or fixity of functioning. The ideas are important and intuitively plausible but, as yet, largely untested.

The hypotheses link closely with the second main approach to personality functioning, namely cognitive processing (Rutter, 1987*b*). The central concept is that personality involves, amongst other things, a set of cognitions about ourselves, our relationships, and our interactions with the environment. These cognitions constitute the self-system (Harter, 1983, 1986). We see ourselves as having a coherence of functioning that is related to our concept about what sort of people we are. The counterpart to our self-image is our tendency to respond to others on the basis of their reputations or our perceptions of what sort of person they are. There is evidence that we do indeed respond to such percepts and not just to people's immediate behaviours; similarly it has been shown that our allocated social roles influence our self concepts which in turn influence our behaviour (Rutter, 1987*b*).

Cognitive sets are thought to play a major role in how people respond to potentially negative life experiences but it is uncertain just which aspect of the self is most crucial in that connection. It may be self-esteem (itself a multi-faceted concept – Harter, 1986), or self-efficacy or social problem-solving skills. Probably in varying degrees all play a part. There are effects on people's appraisal of life experiences, on their mental reaction to them, and on their action to deal with the situation. These self concepts are not seen as static traits. Rather attention has focused on how feelings of self worth develop, how the constituent parts of the self concept become integrated and how changes occur.

Cognitive processes have been postulated as having a particularly important role in the genesis and perpetuation of depression. Seligman has emphasised the importance of feelings of learned helplessness (Abahamson *et al*, 1978; Peterson & Seligman, 1984); Beck (1967) the role of a negative triad of feelings about the self, the immediate life-situation and the future; and Brown & Harris (1978), too, have given the cognitive styles of helplessness and hopelessness a key place in their concepts of how vulnerability factors cause reactions to loss events to lead to depression. There are many unanswered questions regarding these theories (Bebbington, 1985; Brewin, 1985) but there is some evidence in support of the suggestion that low self-esteem may operate as the mediating variable linking early adverse experiences with a later vulnerability to depression (Brown *et al*, 1986; Ingham *et al*, 1986); also the ideas have been useful in formulating effective cognitive therapies for depression (Brewin, 1985; Mathews, 1986).

One of the key issues concerns the relationship between these cognitive sets and temperamental variables. To what extent, for example, does neuroticism predispose to learned helplessness? Insofar as the two are related, does helplessness constitute the mechanism by which the temperamental feature predisposes to emotional disorder, or are the cognitive features no more than epiphenomena for a trait that leads more directly to disorder? We do not know. The different approaches to personality have remained regrettably separate from each other.

The last approach to mention is that based on personality traits. There are many traits that have been considered over the years, covering virtually the whole range of adjectives that could be applied to people. Altruism, empathy and nurturance (Rushton *et al*, 1986) constitute examples of apparently positive attributes; suspiciousness, abasement and rebelliousness (Ahern *et al*, 1982) of those at the negative pole. Some traits, however, are more obviously derived from theoretical constructs. Sensation-seeking (Quay, 1977; Fulker *et al*, 1980), internal locus of control (Rotter, 1975; Miller & Rose, 1982), and femininity (Rowe, 1982; Huston, 1983) constitute examples of this kind. It is difficult to know how to evaluate the usefulness of this heterogeneous mixture of labels. Many of them have been found to have a substantial genetic component (see refs above) and many show moderate stability over time after early childhood (Moss & Susman, 1980). However, these qualities do not tell us much that is useful about personality functioning. In particular, traits reflect a static concept that does not provide for an understanding of developmental change or of dynamic processes (Pervin, 1985).

Nevertheless, three developmental findings warrant mention. First, several longitudinal studies have shown *heterotypic* continuity (Kagan, 1971; Moss & Susman, 1980; Campos *et al*, 1983), meaning that the overt character of behaviour changes over time while individual differences remain stable. For example, passivity in early childhood predicted non-competitiveness in adult life in the Fels study (Kagan & Moss, 1962). Similarly, indirect competitiveness in childhood was related to adult achievement orientation and physical adventurousness to adult sexual behaviour (Ryder, 1967). The notion of heterotypic continuity is an important one but unfortunately at present it lacks a coherent theoretical framework and it is difficult to know how to apply it to clinical issues. Second, it seems that the extent to which personality functioning remains stable over time may be itself a useful feature to consider. Block (1971) found that individuals who changed differed from those who did not, in being less relaxed, less effective people in greater conflict with societal values. Again, the notion seems potentially valuable but we lack the data upon which to build a set of expectations regarding the meaning and predictability of temporal instability in personality functioning. Third, it is evident that genetic contributions to personality development need to be viewed in fluid rather than fixed terms (Plomin, 1986). Not only is it likely that several different sorts of gene-environment interaction operate (Kendler & Eaves, 1986), but also it seems that genetic effects are amplified during development, that to an important extent people create their own environments, so that nature shapes nurture (Scarr & McCartney, 1983), and that the strongest environmental effects are those that impinge differently on different children in the same family (Plomin & Daniels, 1987). This is a field of growing interest and importance.

Finally, following Schneider (1958) there are those traits that derive from psychiatric concepts; such as obsessionality, paranoid qualities and hysterical features (Presley & Walton, 1973; Tyrer & Alexander, 1979; Mann *et al*, 1981). These overlap with the more general neuroticism characteristic and evidence on the relative contribution of genetic and environmental influences to the supposedly more specific traits is contradictory and inconclusive (Torgersen, 1980; Clifford *et al*, 1984). As these traits are central to traditional psychiatric concepts of personality disorder, they are more appropriately discussed under that rubric.

Personality disorder

The concept of personality disorder has had a very chequered history in psychiatry (Lewis, 1974; Millon 1981; Widiger & Frances, 1985; Frances & Widiger, 1986) and the literature is full of disputes on this or that aspect of the topic. Nevertheless, most psychiatrists have found it necessary to make use of this diagnostic category, even if they have done so in widely disparate ways. The three unifying features have been: 1) an onset in childhood or adolescence; 2) longstanding persistence over time without marked remission or relapses; and 3) abnormalities that seem to constitute a basic aspect of the individual's usual functioning. Perhaps four broad trends in the approach to personality disorder may be discerned. First, there is the traditional psychiatric approach in which particular personality types are linked with specific forms of mental illness. In their rather different ways, Kretschner and Schneider were both pioneers of this approach, which has given rise to terms such as schizoid, schizotypal and anankastic or obsessional personality disorders. Second, personality disorders have been defined in terms of some postulated psychological deficit. Prichard's (1835) diagnosis of 'moral insanity' based on a perversion of moral feelings was the forerunner here. Cleckley's (1964) focus on an inability to learn from experience or to exhibit remorse, and Bowlby's (1946) concentration on an inability to form close affectionate ties are in the same tradition. Third, the disorder may be seen solely in terms of social deviance or incompetence, as with Robins' (1966) use of 'sociopathic personality', which was defined as a generalised persistent failure to conform to societal norms. Fourthly, there are the psychodynamic approaches in which the concept is defined in terms of some hypothesised psychodynamic aetiological mechanism. Alexander's (1930) character neurosis, the psychoanalytical oral and anal character types (Fenichel, 1945) and, more recently, Kernberg's (1967) concept of 'borderline personality disorders' are all in that tradition.

The two main 'official' classification systems, the World Health Organisation's (1978) ICD-9 and the American Psychiatric Association's (1980) DSM-III do not follow any approach consistently; rather they contain a somewhat uneasy mixture of several different paradigms (Shepherd & Sartorius, 1974; Siever & Klar, 1986). Both ICD-9 and DSM-III are similar in their general requirement that there be deeply ingrained maladaptive patterns that have been present since adolescence and which have given rise to personal distress or social impairment. However, they differ somewhat in their approach to sub-classification. ICD-9 mostly follows the traditional psychiatric system of basing the types on extremes of particular traits such as paranoid or obsessional qualities. DSM-III employs three clusters (Vaillant & Perry, 1980; Millon, 1981; Siever & Klar, 1986). The first is based on odd eccentric behaviour (the paranoid, schizoid and schizotypal varieties); the second on dramatic, erratic or emotional behaviour (the histrionic, narcissistic, antisocial and borderline varieties); and the third on anxious or fearful behaviour (the avoidant, dependent, compulsive and passive-aggressive types).

Most British research measures have followed the WHO style in supposing that personality disorder derives from the individual's being extreme on particular traits, although the traits assessed have not been synonymous with those in ICD-9 (Walton & Presley, 1973; Tyrer & Alexander, 1979; Mann *et al*, 1981). North American measures are rather more varied and differ in their focus on broader aspects of social functioning and on specific psychological abnormalities, particularly those of the types required for the diagnosis of schizotypal or borderline conditions (Reich, 1985). Taken together in conjunction with the studies relying on less standardised clinical assessments of personality disorder, they provide a body of data that allows examination of some of the main postulates of the various approaches to personality disorder.

On the positive side, it is evident from the few available studies that the overall diagnosis of personality disorder has satisfactory reliability and reasonable predictive validity (Merikangas & Weissman, 1986). Thus, in our 4-year follow-up study of a representative sample of newly referred adults with children below the age of 15 years, we found that those with a personality disorder differed from the rest of the group in being much more likely to have continuing symptoms with social impairment (Rutter & Quinton, 1984*a*). This worse outcome was *not* just a function of duration of symptoms as duration did not relate to outcome in those without a personality disorder (Rutter, 1977; Rutter & Quinton, 1984*a*). Other investigations, too, have shown that personality disorders tend to be persistent over the course of years (Sund, 1973; Tyrer *et al*, 1983*a*). Drake & Vaillant (1985) and Robins (1986, 1987) have shown the childhood origins of personality disorder and the associated social impairment (but low rate of contact with psychiatric services) in adult life.

On the negative side, however, the findings fail to support the trait based approach to personality disorder. The reliability and temporal stability of most trait-defined subtypes has been found to be quite poor (Mann *et al*, 1981; Tyrer *et al*, 1983*b*) and it is common to find that individual patients meet the criteria for several supposedly distinct subtypes (Boyd *et al*, 1984; Siever & Klar, 1986). Follow-up studies of patients with neurotic disorders have similarly shown fluctuating patterns of symptomatology (Greer & Cawley, 1966) and there is only a relatively weak association between specific personality traits and the type of neurotic disorder (Snaith *et al*, 1971; Tyrer *et al*, 1983*b*). It may be useful to assess those personality traits that are associated with an increased predisposition to neurotic disorder (such as obsessionality and emotionality) but these are probably best dealt with in dimensional rather than categorical terms and they do not seem to provide the best approach to personality *disorder*. Evidence is lacking that personality disorder derives from the person being extreme on some particular trait and it seems unlikely that this is the case.

Undoubtedly, the best evidence on the coherence of a subtype is that for antisocial personality disorder. As Robins has shown in her own impeccable follow-up studies, as well as in her review of the literature (Robins, 1978; 1987), almost always it has its origins in childhood conduct disturbance, it is associated with widespread social impairment, and it shows substantial persistence over time. Even so, it is a questionable diagnostic entity. In the first place, it is partially defined in terms of social deviance. This is particularly unsatisfactory from a forensic point of view as Barbara Wootton (1959) pointed out so clearly years ago; because the same behaviours (i.e. antisocial acts) are being used to define the crime and the psychiatric condition (i.e. the personality disorder) said to account for it. However, more importantly, the psychiatric findings do not justify the use of antisocial behaviour as the defining criterion for the personality disorder in adult life. Conduct disturbance in childhood leads onto both antisocial *and* inadequate or dependent type personality disorders, more often the former in males and the latter in females (Quinton *et al*, 1984; Zeitlin, 1986). Indeed, in women emotional and somatisation problems are a common sequel to conduct disorders in childhood (Robins, 1986). Both antisocial *and* other types of personality disorders in parents predispose to conduct disorder in the children (Rutter & Quinton, 1984*a*). Furthermore, family history studies show that antisocial personality disorders and somatisation disorders are associated, again with male and female preponderance respectively (Crowe, 1983; Guze *et al*, 1986). It seems possible that the unifying feature may turn out to be a widespread and pervasive abnormality in social relationships rather than antisocial acts or dependent behaviour or somatic complaints.

ICD-9 has a category for affective personality disorder characterised by mood swings (WHO, 1978) and the category of 'asthenic' personality (meaning anxiety proneness and inability to cope with stress) has been widely employed in European studies in the past (Goldberg, 1983). DSM-III has a somewhat similar category 'dysthymic disorder' but, in contrast, it is classified with affective, rather than personality, disorders (APA, 1980). Longitudinal data have confirmed that such personality features are indeed associated with a substantially increased risk of psychiatric disorder (Hagnell, 1966; Tolle, 1968). However, what is much less clear is how to differentiate these supposed personality disorders from remitting and relapsing affective conditions. Follow-up studies of psychiatric patients show that the pattern of fluctuating recurrance of symptoms is common (Greer & Cawley, 1966; Rutter & Quinton, 1984*a*; Bronisch *et al*, 1985). Sometimes the recurrence amounts to a clinically handicapping disorder and sometimes it does not. The presence of mild depressive symptoms at one point predicts the development of a major affective disorder at some later date (Weissman *et al*, 1986); this appears to be so in childhood as well as in adult life (Kovacs *et al*, 1984*a,b*). Also it is evident that people's responses to personality questionnaires fluctuate markedly according to whether or not they are currently suffering from an affective disorder (Coppen & Metcalfe, 1965; Liebowitz *et al*, 1979; Hirschfeld *et al*, 1983). The same difficulty in differentiating personality features from chronic or recurrent disorders applies in lesser degree to interview measures (Tyrer *et al*, 1983*a,b*). The conclusion seems to be that these subclinical manifestations of dysthymia or cyclothymia are real enough but that they are better dealt with in terms of mild affective disorders rather than separated into a different personality disorder category. That is indeed what has happened to the diagnosis in the draft guidelines for ICD-10.

The category of borderline personality disorder has given rise to a substantial literature in recent years but without resolution of its nosological status (Siever & Klar, 1986; Docherty *et al*, 1986). It is defined primarily in terms of an instability in both mood and interpersonal relationships but it overlaps considerably with the DSM-III categories of narcissistic and histrionic personality disorders, and investigators have differed considerably in the criteria upon which they have placed emphasis. It seems clear enough that there are individuals who have unstable intense personal relationships; who tend to be exploitative and impulsive; and who show marked affective instability, often with recurrent suicidal attempts. Moreover, such characteristics tend to persist over time. Despite its rather misleading title, there seems to be little or no genetic or predispositional association with schizophrenia. Two main questions arise. First, are there good reasons for making this a distinct category that differs from other personality disorder characterised by chronic and widespread relationship difficulties? The data to provide a definitive answer do not exist but perhaps there is more to be said for 'lumping' that 'splitting' at this point. Second, what is the nature of the association with affective conditions? There is no doubt that individuals with borderline disorders have an increased rate of affective disturbance but, on the whole, family data suggest that the two sets of conditions are likely to be genetically distinct (Loranger *et al*, 1982; Pope *et al*, 1983).

Schizotypal personality disorder differs most strikingly from the other subtypes in its emphasis on both odd eccentric behaviour and abnormalities of cognition and perception, with suspiciousness, ideas of reference and paranoid feelings, as well as poor social relationships. Although twin data have been equivocal (Torgerson, 1984) family data have been consistent in showing a relatively strong association with schizophrenia (Kendler *et al*, 1981, 1984; Baron *et al*, 1983). Clinical follow-up also shows that there is substantial overlap with schizophrenia (McGlashan, 1986) and it seems preferable to group schizotypal conditions with schizophrenia rather than with personality disorder. That move is proposed in the draft guidelines for ICD-10.

The situation is comparable, but less clear, with respect to paranoid personality disorders. They, too, show a familial association with schizophrenia (Stephens *et al*, 1975; Kendler & Gruenberg, 1984). However, recent data from Kendler *et al* (1981) suggest a stronger association with delusional states than with schizophrenia. It remains uncertain quite where paranoid personality disorders should be placed in any psychiatric classification scheme.

Finally, there is the issue of how to define and where to place schizoid personality disorder. In spite of its name, no association with schizophrenia was found in the Danish Adoption studies (Kety *et al*, 1975). However, it was defined rather broadly in that investigation. Wolff's (Chick *et al*, 1979; Wolff & Barlow, 1979; Wolff & Chick, 1980) criteria for schizoid disorder of childhood overlap greatly with those for the adult personality disorder in their emphasis on specific abnormalities of empathy and social relatedness. Her findings show many similarities between schizoid disorder and childhood autism, as well as with Asperger's syndrome (Wing, 1981); her follow-up also demonstrated that strong persistence of the clinical pattern. On the whole, the connections with autism seem much more striking than those with schizophrenia (although in both Wolff's and Wing's series a few individuals developed schizophrenia-like disorders). The problem of possible links with both autism and schizophrenia probably stems from the inherent difficulties in differentiating schizoid from schizotypal disorders; in reality it is likely that the former links with autism and the latter with schizophrenia but that the two sometimes get confused. The best means of differentiation have yet to be established but perhaps the schizoid pattern (better termed Asperger's syndrome) should place weight on the early appearance of the pattern, the stereotyped interests, and the lack of empathy; in contrast, the schizotypal pattern may be best characterised by the paranoid, suspicious qualities and the eccentric incongruous behaviour.

The literature on personality disorder is far from satisfactory in numerous respects, most strikingly in the divergent definitions, concepts, and measures. Accordingly, it is not possible to draw any firm conclusions that are likely to receive general assent. Indeed, it is striking that different groups are moving in disparate, often opposite, directions. Thus, the second draft of DSM-III R retains the 11 personality disorder categories in DSM-III and adds two more, sadistic and masochistic or self-defeating. As already noted, the draft for ICD-10 has removed two categories (cyclothymia and schizotypal) from the personality disorder section to other sections. On the other hand, it has created a whole new set of codes for 'personality trait accentuation', which differ from personality disorders only in terms of severity and pervasiveness.

As I have tried to indicate, research findings emphasise both the heterogeneity of the overall personality disorder category and the substantial overlap between the supposedly separate subcategories. It seems desirable to remove those that fit more easily into other sections of the classification, as the ICD-10 draft has done with cyclothymia and schizothymia. The draft guidelines for ICD-10 also groups Asperger's syndrome with autistic conditions, which seems equally justifiable. On the other hand, it retains a separate category for schizoid personality disorders in adult life that are not schizotypal. Whether that is necessary remains to be seen. It is unclear what to do with paranoid personality disorders.

I see little justification for the retention of trait-defined personality disorders. The traits may well be useful descriptors but it seems that they are better dealt with in dimensional not categorical terms.

Rather than split the remaining personality disorder categories, for reasons already discussed, I would group together those that DSM-III termed the 'dramatic' cluster – namely the borderline, antisocial, histrionic and narcissistic varieties, perhaps with the addition of somatisation disorder (although that is less certain). The characteristic that seems to define them all is a pervasive persistent abnormality in maintaining social relationships. The abnormality differs from that in the schizoid and schizotypal varieties in that there is no lack of interest in relationships; moreover it is likely that the early childhood pattern is also different. Of course, it is highly desirable to provide a more precise description of the nature of the relationship difficulty but that requires data not available at present. There is evidence for both genetic (Crowe, 1983) and environmental (Rutter & Quinton, 1984*b*) determinants and, at present, it is quite uncertain whether those with a strong heritable component differ from the remainder, or whether some other subdivision is required. But, for the moment, one overall category defined in terms of relationship abnormalities seems preferable to multiple subcategories without clear boundaries between them.

Conclusions

The terms temperament, personality and personality disorder have often been used interchangeably or, alternatively, they have been viewed in sequential fashion with one leading to the next, so that personality disorders represent the extremes of genetically determined temperamental traits together with the added effect of environmental shaping of personality during the years of development. Although hallowed by time, the concept is mistaken in my view. There is, indeed, a need for all three terms but they refer to rather different phenomena and rather different processes. It is suggested that temperament is best viewed in terms of a relatively small number of simple, non-motivational, non-cognitive, stylistic features of which emotionality, activity and sociability are the best validated. It is possible that these characteristics have fairly direct neurobiological correlates but this has yet to be demonstrated. By contrast, personality refers to the coherence of functioning that derives from how people react to their given attributes, how they think about themselves, and how they put these together into some form of conceptual whole. There is no one way of measuring personality. Some aspects may be dealt with in terms of dimensional traits such as obsessionality but others are best thought of in terms of processes involving cognitive, emotional and social mechanisms. Personality disorders, as traditionally conceived, include several categories that are most appropriately considered as variants of conditions such as affective disorder, autism and schizophrenia. However, there remains a substantial group of disorders that are characterised by a persistent, pervasive abnormality in social relationships and social functioning generally. These personality disorders are clinically important in terms of both their incidence and their chronicity. They deserve much greater attention than they have received in the past.

References

Abrahamson, L. Y., Seligman, M. E. P. & Teasdale, J. D. (1978) Learned helplessness in humans. Critique and reformulation. *Journal of Abnormal Psychology*, **87**, 49–74.

Ahern, F. M., Johnson, R. C., Wilson, J. R., McClearn, G. E. & Vandenberg, S. G. (1982) Family resemblances and personality. *Behaviour Genetics*, **12**, 261–280.

Alexander, F. (1930) The neurotic character. *International Journal of Psychoanalysis*, **11**, 292–313.

Allport, G. W. (1937) *Personality: A Psychological Interpretation*. New York: Holt.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)*. Washington DC: American Psychiatric Association.

Asher, S. R. (1983) Social competence and peer status: Recent advances and future directions. *Child Development*, **54**, 1427–1434.

Barkley, R. A. (1981) The use of psychopharmacology to study reciprocal influences in parent–child interaction. *Journal of Abnormal Child Psychology*, **9**, 303–310.

Baron, M., Gruen, R., Asnis, L. & Kane, J. (1983) Age-of-onset in schizophrenia and schizotypal disorders: Clinical and genetic implications. *Neuropsychobiology*, **10**, 199–204.

Bates, J. E. (1980) The concept of difficult temperament. *Merrill Palmer Quarterly*, **26**, 299–319.

—— (1986) The measurement of temperament. In *The Study of Temperament: Changes, Continuities and Challenges*, (eds R. Plomin & J. Dunn). Hillsdale NJ: Lawrence Erlbaum.

—— & Bayles, K. (1984) Objective and subjective components in mothers' perceptions of their children from age 6 months to 3 years. *Merrill-Palmer Quarterly*, **30**, 111–130.

——, Maslin, C. A. & Frankel, K. A. (1985) Attachment security, mother–child interaction and temperament as predictors of behaviour-problem ratings at age three years. In *Growing Points of Attachment and Research, Monographs of the Society of Research in Child Development*, (eds I. Bretherton & E. Waters), Series 209, **50**, Nos 1-2.

Bebbington, P. (1985) Three cognitive theories of depression. *Psychological Medicine*, **15**, 759–770.

Beck, A. T. (1967) *Depression: Clinical, Experimental and Theoretical Aspects*, New York: Harper & Row.

Berger M. (1985) Temperament and individual differences. In *Child and Adolescent Psychiatry: Modern Approaches (2nd Edition)* (eds M. Rutter & L. Hersov). Oxford: Blackwell Scientific.

Block, J. (1971) *Lives through Time*. Berkeley, CA. Bancroft Books.

Bowlby, J. (1946) *Fourty-Four Juvenile Thieves: Their Characters and Home Life*. London: Bailliere, Tindall & Cox.

—— (1969/82) *Attachment and Loss I: Attachment*. London: Hogarth Press.

Boyd, J. H., Burke, J. D., Gruenberg, E., Holzer, C. E., Rae, D.S., George, L. K., Karno, M., Stolzman, R., McEvoy, L. & Nestadt, G. (1984) Exclusion criteria of DSM-III. *Archives of General Psychiatry*, **41**, 983–989.

Bretherton, I. & Waters, E. (eds) (1985) Growing points of attachment and research. *Monographs for the Society for Research in Child Development* (eds I. Bretherton & E. Waters). Series 209, **50**, Nos 1–2.

Brewin, C. (1985) Depression and causal attributions: What is their relation? *Psychological Bulletin*, **9**, 297–309.

Bronisch, T., Wittchen, H. U., Krieg, C. Rupp, H. U. & Von Zerssen, D. (1985) Depressive neurosis: A long-term prospective and retrospective follow-up study of former patients. *Acta Psychiatrica Scandinavia*, **71**, 237–248.

Brown, G. W. & Harris, T. O. (1978) *Social Origins of Depression: A Study of Psychiatric Disorders in Women*. London: Tavistock Publications.

——, —— & Bifulco, A. (1986) The long-term effects of early loss of parent. In *Depression in Young People: Clinical and Developmental Perspectives*, (eds M. Rutter, C. E. Izard & P. B. Read). New York: Guilford Press.

Brunk, M. A. & Henggeler, S. W. (1984) Child influence on adult controls. An experimental investigation. *Developmental Psychology*, **20**, 1074–1081.

Buss, D. M. (1981) Predicting parent–child interactions from children's activity level. *Developmental Psychology*, **17**, 59–65.

Buss, A. H. & Plomin, R. (1975) *A Temperament Theory of Personality Development*. New York: John Wiley.

—— & —— (1986) The EAS approach to temperament. *The Study of Temperament: Changes, Continuities and Challenges*, (eds R. Plomin & J. Dunn), Hillsdale NJ: Lawrence Erlbaum.

Campos, J. J., Barrett, K. C., Lamb, M. E., Goldsmith, H. H. & Stenberg, C. (1983) Socioemotional development. In *Infancy and Developmental Psychobiology (Volume 2) Mussen's Handbook of Child Psychology*, (eds M. M. Haith & J. J. Campos), 4th ed. New York: Wiley.

Carey, W. B. (1986) Interactions of temperament and clinical conditions. In *Advances in Developmental and Behavioral Pediatrics*, (eds M. Wolraich & D. Routh), Vol. 6. Greenwich CT: JAI Press.

Chess, S. & Thomas, A. (1984) *Origins and Evolution of Behavior Disorders*. New York: Brunner/Mazel.

Chick, J., Waterhouse, L. & Wolff, S. (1979) Psychological construing in schizoid children grown up. *British Journal of Psychiatry*, **135**, 435–440.

Cleckley, H. (1964) *The Mask of Sanity*. St Louis: Mosby.

Clifford, C. A., Murray, R. M. & Fulker, D. W. (1984) Genetic and environmental influences on obsessional traits and symptoms. *Psychological Medicine*, **14**, 791–800.

Cloninger, C. R. (1987) A unified biosocial theory of personality and its role in the development of anxiety states. *Psychiatric Developments* (in press).

Condry, J. & Condry, S. (1976) Sex differences: A study of the eye of the beholder. *Child Development*, **47**, 812–819.

Coppen, A. & Metcalfe, M. (1965) Effects of a depressive illness on MPI scores. *British Journal of Psychiatry*, **111**, 236–239.

Cross, T. G., Nienhuys, T. G. & Kirkman, M. (1985) Parent–child interaction with receptively disabled children: Some determinants of maternal speech style. In *Children's Language*, (ed K. E. Nelson). Hillsdale, NY: Lawrence Erlbaum.

Crowe, R. R. (1983) Antisocial personality disorders. In *The Child at Psychiatric Risk*, (ed. R. E. Tarter). New York: Oxford University Press.

Dare, C. (1985) Psychoanalytic theories of development. In *Child and Adolescent Psychiatry: Modern Approaches*, (eds M. Rutter & L. Hersov). Oxford: Blackwell Scientific.

De Vries, M. W. (1984) Temperament and infant mortality among the Masai of East Africa. *American Journal of Psychiatry*, **141**, 1189–1194.

Dodge, K. A. (1980) Social cognition and children's aggressive behavior. *Child Development*, **51**, 162–172.

Drake, R. E. & Vaillant, G. E. (1985) A validity study of Axis II of DSM-III. *American Journal of Psychiatry*, **142**, 553–558.

Docherty, J. P., Fiester, S. J. & Shea, T. (1986) Syndrome diagnosis and personality disorder. In *American Psychiatric Association Annual Review*, Vol. 5. (eds A. J. Frances & R. E. Hales). Washington DC: American Psychiatric Press.

Duncan-Jones, P., Grayson, D. A. & Moran, P. A. P. (1986) The utility of latent trait models in psychiatric epidemiology. *Psychological Medicine*, **16**, 391–406.

Dunn, J. (1980) Individual differences in temperament. In *Scientific Foundations of Developmental Psychiatry*, (ed. M. Rutter). London: Heineman Medical.

—— & Kendrick, C. (1980) Studying temperament and parent–child interaction: Comparison, interview and direct observation. *Developmental Medicine and Child Neurology*, **22**, 494–496.

—— & —— (1982) *Siblings: Love, Envy and Understanding*. London: Grant McIntyre.

Eagle, M. N. (1984) *Recent developments in psychoanalysis: A critical Evaluation*. New York: McGraw Hill.

Epstein, S. (1979) The stability of behavior: I: On predicting most of the people much of the time. *Journal of Personality and Social Psychology*, **37**, 1097–1126.

—— (1983) Aggregation and beyond: Some basic issues of the prediction of behavior. *Journal of Personality*, **51**, 360–392.

Eysenck, H. J. (1967) *The Biological Basis of Personality*. Springfield Ill: CC Thomas.

—— (1982) *Personality, Genetics and Behavior*. New York: Prager.

Fenichel, O. (1945) *The Psychoanalytic Theory of the Neurosis*. New York: Nortons.

Frances, A. J. & Widiger, T. (1986) The classification of personality disorders: An overview of problems and solutions. In *American Psychiatric Association Annual Review*, Vol. 5, (eds A. J. Frances & R. E. Hales). Washington DC: American Psychiatric Press.

Fulker, D. W., Eysenck, S. B. G. & Zuckerman, M. (1980) A genetic and environmental analysis of sensation seeking. *Journal of Research in Personality*, **14**, 261–281.

Gesell, A. (1937) Early evidence of individuality in human infants. *Scientific Monthly*, **45**, 217–255.

Goldberg, D. (1983) Personality disorders in adults predisposing to minor depressive reactions. In *Handbook of Psychiatry*, Vol. 4, *The Neurosis and Personality Disorders*, (eds G. F. M. Russell & L. Hersov). Cambridge: Cambridge University Press.

Graham, P. & Stevenson, J. (1986) Temperament and psychiatric disorder—The genetic contribution to behaviour in childhood. Paper given at the Child and Adolescent Psychiatry Section of the Royal Australian and New Zealand College of Psychiatrists, 5–7 September, 1986.

——, Rutter, M. & George, S. (1973) Temperamental characteristics as predictors of behavior disorders in children. *American Journal of Orthopsychiatry*, **43**, 328–339.

Gray, J. A. (1983) Anxiety, personality and the brain. In *Physiological Correlates of Human Behaviour, III: Individual Differences and Psychopathology*, (eds A. Gale & J. A. Edwards). New York: Academic Press.

Greer, H. S. & Cawley, R. H. (1966) *Some Observations on the Natural History of Neurotic Illness*. Australian Medical Association Mervyn Archdall Medical Monograph No 3. Glebe NSW; Australian Medical Publishing Company.

Guze, S. B., Cloninger, C. R., Martin, R. L. & Clayton, P. J. (1986) A follow-up and family study of Briquet's syndrome. *British Journal of Psychiatry*, **149**, 17–23.

Hagnell, O. (1966) *A Prospective Study of the Incidence of Mental Disorder*. Lund: Svenska Bokforlaget.

Hall, C. S. & Lindzey, G. (1978) *Theories of Personality*, 3rd ed. New York: Wiley.

Harter, S. (1983) Developmental perspectives on self-system. In *Socialization, Personality and Social Development*, Vol. 4. *Handbook of Child Psychology*, (ed. E. Hetherington), 4th ed. New York: Wiley.

—— (1986) Processes underlying the construction, maintenance and enhancement of the self concept in children. In *Psychological Perspectives on the Self*, Vol. 3. (eds J. Suls & A. Greenwald). Hillsdale NJ: Lawrence Erlbaum.

Hartup, W. W. (1983) Peer relations. In *Socialization, Personality and Social Development*, Vol. 4, *Handbook of Child Psychology*, (ed. E. M. Hetherington), 4th ed. New York: Wiley.

—— & Rubin, Z. (eds) (1986) *Relationships and Development*. Hillsdale NJ: Lawrence Erlbaum.

Haviland, J. J. & Malatesta, C. Z. (1981) The development of sex differences in non-verbal signals: Fallacies, facts and fantasies. In *Gender and Non-Verbal Behavior*, (eds C. Mayo & M. M. Henley). New York: Springer-Verlag.

Hinde, R. A. (1979) *Towards Understanding Relationships*. London: Academic Press.

—— & Stevenson-Hinde, J. (1986) Relating childhood relationships to individual characteristics. In *Relationships and Development*, (eds W. W. Hartup & Z. Rubin). Hillsdale, HJ: Lawrence Erlbaum.

Hirschfeld, R. M. A., Klerman, G. L., Clayton, P. J. *et al* (1983) Assessing personality: Effects of the depressive state on trait measurement. *American Journal of Psychiatry*, **140**, 695–699.

Hodges, J. & Tizard, B. (1987*a*) IQ and behavioural adjustment of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry*. (in press).

—— & —— (1987*b*) Social and family relationships of ex-institutional adolescents. *Journal of Child Psychology and Psychiatry*. (in press).

Huttenden, M. O. & Nyman, G. (1982) On the continuity, change and clinical value of infant temperament in a prospective epidemiological study. In *Temperamental Differences in Infants and Young Children*, (eds R. Porter & G. M. Collins). London: Pitman.

Huston, A. C. (1983) Sex-typing. In *Socialization, Personality and Social Development*, Vol. 4, *Mussen's Handbook of Child Psychology*, (ed. E. M. Hetherington), 4th ed. New York: Wiley.

Ingham, J. G., Kreitman, N. B., Miller, P., Sashidharan, S. P. & Surtess, P. G. (1986) Self-esteem, vulnerability and psychiatric disorder in the community. *British Journal of Psychiatry*, 148, 375–385.

Kagan, J. (1971) *Change and Continuity in Infancy*. New York: Wiley.

—— & Moss, H. A. (1962) *Birth to Maturity*. New York: Wiley.

——, Reznick, J. S. & Snidman, N. (1986) Temperamental inhibition in early childhood. *The Study of Temperament: Changes, Continuities and Challenges* (eds R. Plomin & J. Dunn). Hillsdale NJ: Lawrence Erlbaum.

——, —— & —— (1987) The physiology and psychology of behavioral inhibition in children. *Child Development*, (in press).

Kendler, K. S. & Eaves, L. J. (1986) Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry*, 143, 279–289.

—— & Gruenberg, A. M. (1984) An independent analysis of the Danish adoption study of schizophrenia. VI. The relationship between psychiatric disorders as defined by DSM-III in the relatives and adoptees. *Archives of General Psychiatry*, 41, 555–564.

——, —— & Strauss, J. S. (1981) An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia. II. *Archives of General Psychiatry*, 38, 982–984.

——, Masterson, C. C., Ungaro, R. *et al* (1984) A family history study of schizophrenia related personality disorders. *American Journal of Psychiatry*, 141, 424–427.

Kernberg, O. F. (1967) Borderline personality organization. *Journal of the American Psychoanalytic Association*, 15, 641–685.

Kety, S. S., Rosenthal, D., Wender, P. H., Schulsinger, F., & Jacobsen, B. (1975) Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic. A preliminary report based on psychiatric interviews. In *Genetic Research in Psychiatry* (eds R. Fieve, D. Rosenthal & H. Brill), Baltimore: Johns Hopkins Press.

Korn, S. J. (1984) Continuities and discontinuities in difficult/easy temperament: Infancy to young adulthood. *Merrill Palmer Quarterly*, 30, 189–199.

Kovacs, M., Feinberg, J. L., Crouse-Novak, H. A., Paulauskas, S. L. & Finkelstein, R. (1984*a*) Depressive disorders in childhood I. A longitudinal perspective study of characteristics and recovery. *Archives of General Psychiatry*, 41, 229–237.

——, ——, ——, Pollack, M. & Finkelstein, R. (1984*b*) Depressive disorders in childhood. II. A longitudinal prospective study of risk of a subsequent major depression. *Archives of General Psychiatry*, 41, 643–649.

Lacey, J. & Lacey, B. (1958*a*) The relationship of resting autonomic activity to motor impulsivity. *Research in Nervous and Mental Disease*, 36, 144–209.

—— & —— (1985*b*) Verification and extension of the principle of autonomic response specificity. *American Journal of Psychology*, 71, 50–73.

Lee, C. L. & Bates, J. E. (1985) Mother–child interaction at age two years and perceived difficult temperament. *Child Development*, 56, 1314–1325.

Lewis, A. (1974) Psychopathic personality: A most elusive category. *Psychological Medicine*, 4, 133–140.

Liebowitz, M. R., Stallone, F., Dunner, D. L. *et al* (1979) Personality features of patients with primary affective disorders. *Acta Psychiatrica Scandinavica*, 60, 214–244.

Loehlin, J. C. & Nichols, R. C. (1976) *Heredity, Environment and Personality: A study of 850 twins*. Austin: University of Texas Press.

Loranger, A. W., Oldham, J. M. & Tulis, E. H. (1982) Familial transmission of DSM-III borderline personality disorder. *Archives of General Psychiatry*, 39, 795–799.

Magnusson, D. (1986) *Individual Development in an Interactional Perspective* Vol. 1. Series: *Paths through Life*, (ed. D. Magnusson). Hillsdale NJ: Lawrence Erlbaum.

Mann, A. H., Jenkins, R., Cutting, J. C. & Cowen, P. J. (1981) The development and use of a standardised assessment of abnormal personality. *Psychological Medicine*, 11, 839–847.

Martin, N. G., Eaves, L. J. & Fulker, D. W. (1979) The genetical relationship of impulsiveness and sensation seeking to Eysenck's personality dimensions. *Acta Geneticae Medicae et Gemellologiae*, 28, 197–210.

Mathews, A. (1986) Cognitive processes in anxiety and depression: A discussion paper. *Journal of the Royal Society of Medicine*, 79, 158–161.

Maziade, M., Caperaca, P., Laplante, D., Boudreault, M., Thivierge, J., Cote, R. & Boutin, P. (1985) Value of difficult temperament among 7-year-olds in the general population for predicting psychiatric diagnosis at age 12. *American Journal of Psychiatry*, 142, 943–946.

McCall, R. B. (1986) Issues of stability and continuity in temperament research. *The Study of Temperament: Changes, Continuities and Challenges*, (eds R. Plomin & J. Dunn), Hillsdale NJ: Lawrence Erlbaum.

McGlashan, T. H. (1986) Schizotypal personality disorder. Chestnut Lodge follow-up study. VI. Long-term follow-up perspectives. *Archives of General Psychiatry*, 43, 329–334.

Merikangas, K. & Weissman, M. M. (1986) Epidemiology of DSM-III axis II personality disorders. In *American Psychiatric Association Annual Review*, Vol. 5, (eds A. J. Frances & R. E. Hales), Washington DC: American Psychiatric Press.

Miller, J. Z. & Rose, R. J. (1982) Familial resemblance in locus of control: A twin family study of the internal–external scale. *Journal of Personality and Psychology*, 42, 535–540.

Millon, T. (1981) *Disorders of Personality: DSM III Axis II*. New York: Wiley.

Mischel, W. (1968) *Personality and Assessment*. New York: Wiley.

—— (1979) On the interface of cognition and personality: Beyond the person–situation debate. *American Psychologist*, 34, 740–754.

Moos, R. H. (ed.) (1986) *Coping with Life Crises: An Integrated Approach*. New York: Plenum Press.

Moss, H. A. & Susman, E. J. (1980) Longitudinal study of personality development. In *Constancy and Change in Human Development*, (eds O. G. Brim & J. Kagan). Cambridge Mass: Harvard University Press.

Mora, G. (1980) Historical and theoretical trends in psychiatry. In *Comprehensive Textbook of Psychiatry*, Vol. 1. (eds H. I. Kaplan, A. M. Freedman & B. J. Sadock), 3rd ed. Baltimore: Williams and Wilkins.

Murray, H. A. *et al* (1938) *Explorations of Personality*, New York: Oxford.

—— & Kluckhohn, C. (1953) Outline of a conception of personality. In *Society and Culture* (eds C. Kluckhohn, H. A. Murray & D. Schneider), 2nd ed. New York: Knopf.

Olweus, D. (1979) Stability of aggressive reaction patterns in males: A review. *Psychological Bulletin*, 86, 852–875.

Parker, G., Brown, L. & Blignault, I. (1986) Coping behaviors as predictors of the course of clinical depression. *Archives of General Psychiatry*, **43**, 561–565.

Pavolov, I. P. (1927) *Conditioned Reflexes* (trans. by G. V. Anrep). London: Oxford University Press.

Pervin, L. A. (1985) Personality: Current controversies, issues and directions. *Annual Review in Psychology*, **36**, 83–114.

Peterson, C. & Seligman, M. E. P. (1984) Causal explanation as a risk factor for depression: Theory and evidence. *Psychological Review*, **91**, 347–374.

Plomin, R. (1983) Childhood temperament. In *Advances in Clinical Child Psychology*, Vol. 6 (eds B. B. Lahey & A. E. Kazdin), New York: Plenum Press.

—— (1986) *Development, Genetics and Psychology*. Hillsdale, NJ: Lawrence Erlbaum.

—— & Daniels, D. (1987) Why are children in the same family so different from one another? *The Behavioral and Brain Sciences*, (in press).

Pope, H., Jonas, J., Hudson, J. *et al* (1983) The validity of DSM-III borderline personality disorders. *Archives of General Psychiatry*, **40**, 23–30.

Presley, A. S. & Walton, H. J. (1973) Dimensions of abnormal personality. *British Journal of Psychiatry*, **122**, 269–276.

Prichard, J. C. (1835) *A treatise on insanity and other disorders affecting the mind*. Philadelphia: Haswell, Barrington and Haswell.

Quay, H. C. (1977) Psychopathic behavior; reflections on its nature, origins and treatment. In *The Structuring of Experiences* (eds I. C. Uzgiris & F. Weizmann), New York: Plenum Press.

Quinton, D., Rutter, M. & Liddle, C. (1984) Institutional rearing, parenting difficulties and marital support. *Psychological Medicine*, **14**, 107–124.

Reich, J. (1985) Measurement of DSM-III axis II. *Comprehensive Psychiatry*, **26**, 352–363.

Reznick, J. S., Kagan, J., Snidman, N., Gersten, M., Baak, K. & Rosenberg, A. (1986) Inhibited and uninhibited children: A follow up study. *Child Development*, **57**, 660–680.

Robins, L. N. (1966) *Deviant Children Grown Up*. Baltimore: Williams and Wilkins.

—— (1978) Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. *Psychological Medicine*, **8**, 611–622.

—— (1986) The consequence of conduct disorder in girls. In *Development of Antisocial and Prosocial Behavior*. (eds D. Olweus, J. Black & M. Radke-Yarrow). New York: Academic Press.

—— (1987) The epidemiology of antisocial personality. In *Psychiatry* (ed. J. O. Cavenar), Philadelphia: J B Lippincott.

Rotter, J. B. (1975) Some problems and misconceptions related to the construct of internal versus external control of reinforcement. *Journal of Consulting and Clinical Psychology*, **43**, 56–67.

Rowe, D. C. (1982) Sources of variability in sex-linked personality attributes. *Developmental Psychology*, **18**, 431–434.

Rushton, J. P., Fulker, D. W., Neale, M. C., Nias, D. K. B. & Eysenck, H. J. (1986) Altruism and aggression: The heritability of individual differences. *Journal of Personality and Social Psychology*, **50**, 1192–1198.

Rutter, M. (1977) Surveys to answer questions. In *The Epidemiological Approaches in Child Psychiatry*, (ed. P. Graham). London and New York: Academic Press.

—— (1978) Family area and school influences on the genesis of conduct disorders. In *Aggression and Antisocial Behaviour in Childhood and Adolescence, Journal of Child Psychology and Psychiatry Book Supplement No 1*, (eds L. A. Hersov & M. Berger with D. Shaffer). Oxford: Pergamon Press.

—— (1981) Stress, coping and development: Some issues and some questions. *Journal of Child Psychology and Psychiatry*, **22**, 323–356.

—— (1986*a*) Child psychiatry: The interface between clinical and developmental research. *Psychological Medicine*, **16**, 151–160.

—— (1986*b*) Meyerian psychobiology: Personality development and the role of life experiences. *American Journal of Psychiatry*, **143**, 1077–1087.

—— (1987*a*) Epidemiological approaches to developmental psychopathology. (Submitted for publication).

—— (1987*b*) The role of cognition in child development and disorder. *British Journal of Medical Psychology*, (in press).

—— (1987*c*) Continuities and discontinuities from infancy. In *Handbook of Infant Development*, (ed J. Osofsky), 2nd ed. New York: Wiley (in press).

—— & Giller, H. (1983) *Juvenile Delinquency: Trends and Perspectives*. Harmondsworth, Middx: Penguin Books.

—— & Quinton, D. (1984*a*) Parental psychiatric disorder: Effects on children. *Psychological Medicine*, **14**, 853–880.

—— & —— (1984*b*) Long-term follow-up of women institutionalised in childhood: factors promoting good functioning in adult life. *British Journal of Developmental Psychology*, **2**, 191–204.

——, Birch, H., Thomas, A. & Chess, S. (1964) Temperamental characteristics in infancy and later development of behavioural disorders. *British Journal of Psychiatry*, **110**, 651–661.

Ryder, R. G. (1967) Birth to maturity revised: A canonical reanalysis. *Journal of Personality and Social Psychology*, **7**, 168–172.

Scarr, S. & Kidd, K. K. (1983) Developmental behavior genetics. In *Handbook of Child Psychology*, Vol. 2 (ed. P. H. Mussen), New York: Wiley.

—— & McCartney, K. (1983) How people make their own environments: A theory of genotype-environmental effects. *Child Development*, **54**, 424–435.

Schaffer, H. R. (1966) Activity level as a constitutional determinant of infantile reaction to deprivation. *Child Development*, **37**, 595–602.

Schneider, K. (1958) *Psychopathic Personalities*. London: Cassel and Co.

Sheldon, W. (1942) *The varieties of temperament: A psychology of constitutional differences*. New York: Harper.

Shepherd, M. & Sartorius, N. (1974) Personality disorder and the international classification of diseases. *Psychological Medicine*, **4**, 141–146.

Siever, L. J. & Klar, H. (1986) A review of DSM-III criteria for the personality disorders. In *American Psychiatric Association Annual Review*, Vol. 5 (eds A. J. Frances & R. E. Hales), Washington DC: American Psychiatric Press.

Snaith, R. P., McGuire, R. J. & Fox, K. (1971) Aspects of personality in depression. *Psychological Medicine*, **1**, 239–246.

Sroufe, L. A. (1979) The coherence of individual development. *American Psychologist*, **34**, 834–841.

—— & Fleeson, J. (1986) Attachment and the construction of relationships. In *Relationships and Development*, (eds W. W. Hartup & Z. Rubin), Hillsdale NJ: Lawrence Erlbaum.

——, Jacobvitz, D., Mangelsdorf, S., DeAngelo, E. & Ward, M. J. (1985) Generational boundary dissolution between mothers and their preschool children: A relationship systems approach. *Child Development*, **56**, 317–325.

Steinberg, L. (1986) Stability (and instability) of type A behavior from childhood to young adulthood. *Developmental Psychology*, **22**, 393–402.

Stephens, D. A., Atkinson, M. W., Kay, D. W. K. *et al* (1975) Psychiatric morbidity in parents and siblings of schizophrenics and non-schizophrenics. *British Journal of Psychiatry*, **127**, 97–108.

Stevenson, J. & Graham, P. (1982) Temperament: A consideration of concepts and methods. In *Temperamental Differences in Infants and Young Children* Ciba Foundation Symposium 89 (eds R. Porter & G. M. Collins). London: Pitman.

Stevenson-Hinde, J. & Hinde, R. A. (1986) Changes in associations between characteristics. *The Study of Temperament: Changes, Continuities and Challenges*, (eds R. Plomin & J. Dunn). Hillsdale NJ: Lawrence Erlbaum.

SUND, A. C. (1973) The prognosis of psychiatric disorders in young Norwegian men. *British Journal of Psychiatry*, **122**, 125–139.

SUOMI, S. J. (1983) Social development in rhesus monkey: Considerations of individual differences. In *The Behavior of Human Infants*, (eds A. Oliverio & M. Zappella). New York and London: Plenum Press.

TELLEGEN, A. (1985) Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In *Anxiety and the Anxiety Disorders*, (eds A. H. Tuma & J. Maser), Hillsdale NJ: Lawrence Erlbaum.

THOMAS, A. & CHESS, S. (1977) *Temperament and Development*, New York: Brunner/Mazel.

——, ——, BIRCH, H., HERTZIG, M. & KORN, S. (1963) *Behavioral Individuality in Early Childhood*. New York: New York University Press.

TIZARD, J. (1975) Race and IQ: the limits of probability. *New Behaviour*, **1**, 6–9.

TOLLE, R. (1968) The mastery of life by psychopathic personalities. *Psychiatric Clinic*, **1**, 1968.

TORGERSEN, S. (1980) The oral, obsessive and hysterical personality syndromes. A study of hereditary and environmental factors by means of the twin method. *Archives of General Psychiatry*, **37**, 1272–1277.

—— (1984) Genetic and nosological aspects of schizotypal and borderline personality disorders. A twin study. *Archives of General Psychiatry*, **41**, 546–554.

TYRER, P. & ALEXANDER, J. (1979) Classification of personality disorder. *British Journal of Psychiatry*, **135**, 163–167.

——, CASEY, P. & GOLL, J. (1983*a*) Relationships between neurosis and personality disorder, *British Journal of Psychiatry*, **142**, 404–408.

——, STRAUSS, J. & CICCHETTI, D. (1983*b*) Temporal reliability of personality in psychiatric patients. *Psychological Medicine*, **13**, 393–398.

VAILLANT, G. E. (1977) *Adaptation to Life*. Boston: Little, Brown & Co.

—— (1983) Childhood environment and maturity of defense mechanisms. In *Human Development: An Interactional Perspective*, (eds D. Magnusson & V. L. Allen), New York: Academic Press.

—— & PERRY, J. C. (1980) Personality disorders. In *Comprehensive Textbook of Psychiatry*, Vol. 2, (eds H. Kaplan, A. M. Freedman & B. J. Sadock), 3rd ed. Baltimore: Williams and Wilkins.

WALTON, H. J. & PRESLEY, A. S. (1973) Use of a category system in the diagnosis of adult personality. *British Journal of Psychiatry*, **122**, 259–269.

WEISSMAN, M. M., MYERS, J. K., TOMPSON, W. D. & BELAGER, A. (1986) Depressive symptoms as a risk factor for mortality and for major depression. In *Life Span Research on the Prediction of Psychopathology*, (eds L. Earlenmenyer-Kimling & N. E. Muller). Hillsdale NJ: Lawrence Erlbaum.

WIDIGER, T. A. & FRANCES, A. (1985) The DSM-III personality disorders. *Archives of General Psychiatry*, **42**, 615–623.

WING, L. (1981) Asperger's syndrome: A clinical account. *Psychological Medicine*, **11**, 115–130.

WOLFF, S. & BARLOW, A. (1979) Schizoid personality in childhood: A comparative study of schizoid, autistic and normal children. *Journal of Child Psychology and Psychiatry*, **20**, 29–46.

—— & CHICK, J. (1980) Schizoid personality in childhood: A controlled follow-up study. *Psychological Medicine*, **10**, 85–100.

WOLKIND, S. N. & DE SALIS, W. (1982) Infant temperament, maternal mental stage and child behavioural problems. In *Temperamental Differences in Infants and Young Children*, CIBA Foundation Symposium 69 (eds R. Porter & G. M. Collins). London: Pitman.

—— & RUTTER, M. (1985) Separation, loss and family relationships. In *Child and Adolescent Psychiatry: Modern Approaches* (eds M. Rutter & L. Hersov), 2nd ed. Oxford: Blackwell Scientific.

WOOTTON, B. (1959) *Social Science and Social Pathology*. London: Allen and Unwin.

WORLD HEALTH ORGANISATION (1978) International classification of diseases, 9th Revision, Geneva: World Health Authority.

ZEITLIN, H. (1986) *The Natural History of Psychiatric Disorder in Childhood*. Institute of Psychiatry/Maudsley Monograph, No 29, Oxford: Oxford University Press.

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The Dexamethasone Suppression Test in Depression

A WORLD HEALTH ORGANIZATION COLLABORATIVE STUDY

The response to the dexamethasone suppression test (DST) was examined in 543 patients suffering from major depressive illness and 246 healthy controls, from 13 research centres, representing 12 different countries, in a World Health Organization Collaborative Study. In almost all the centres, the post-dexamethasone plasma cortisol concentration was significantly higher in the patients than in the controls. Although there is variation between centres, this abnormal response to the DST was shown to be frequent in patients from widely different geographical areas.

The dexamethasone suppression test (DST) has been reported to be abnormal in many but not all investigations in depressive patients. The proportion of patients showing an abnormal response has varied greatly between different centres (Carroll *et al*, 1982).

In order to study the incidence of this abnormal response, the World Health Organization initiated in 1981 a multicentre prospective investigation of DST response in patients from 13 research centres. Since it was unlikely that 13 research centres would use the same technique to measure plasma cortisol concentration, it was decided that all samples would be sent to one laboratory (Epsom) for the estimation of cortisol and for the collation of the clinical details of the patients.

Method

Thirteen research centres from Europe, Africa, Asia and North America took part in the study. These centres were located in Basle, Brussels, Budapest, Casablanca, Copenhagen, Epsom, Irvine, Lucknow, Moscow, Munich, Nagasaki, Sapporo and Utrecht.

Dexamethasone suppression test

One mg of dexamethasone (Oradexon–Organon®) was administered to all patients at 23h00 (except for the patients from the Epsom centre who received the dexamethasone at 20h00). Blood samples were collected into heparin tubes from the patients between 15h00 and 16h00 the following day; the plasma was obtained by centrifugation; it was stored at −20°C until a sufficient number of samples had been acquired for transport in dry ice by air to the laboratory at the Epsom Centre. Cortisol was estimated using a radioimmunoassay technique (Amerlex®). The samples from Epsom were assayed using a radioimmunoassay kit produced by CIS International. It has already been reported that there is an excellent agreement between these two kits (Wood *et al*, 1983).

The results of the cortisol assays are expressed for each centre in two ways (a) as the mean post-dexamethasone plasma cortisol concentration and (b) as the percentage of abnormal responses. A post-dexamethasone cortisol level of <50 ng/ml was taken as a normal response; an abnormal response was taken as $\geqslant 50$ ng/ml of cortisol. The scientific officer who estimated the cortisol levels was unaware of the clinical details of the patients.

Classification and rating of patients

All patients from each centre fulfilled the Research Diagnostic Criteria (RDC) for major depressive disorder (Spitzer *et al*, 1978) and these were taken as the criteria for entry into the study. They were consecutively admitted to the study without any other pre-selection. The patients were classified according to the International Classification of Diseases - 9th Revision (ICD-9) (World Health Organization, 1978) and the Newcastle Diagnostic Scale (Carney *et al*, 1965). The severity of the patients' depressive symptoms was assessed using the Hamilton Rating Scale for Depression (HRS-D) (Hamilton, 1967).

Before the start of the study, a researcher responsible for the implementation of the study from each centre attended a training session in Basle to become familiar with the procedures of the research protocol and assessment instruments. This training was followed up by a reliability rating exercise to obtain the levels of inter-rater agreement on the clinical rating of patients on the Newcastle Scale.

Information was provided as to whether the patient was an in-patient or out-patient, his age, sex, any recent or chronic physical illness and any medication received in the 2 weeks prior to the administration of dexamethasone.

Control groups

During the early stages of the investigation it became apparent that the small number of abnormal responses previously reported in a healthy population might also vary from country to country and all investigators were therefore asked, if possible, to supply post-dexamethasone plasma from normal controls.

Means were compared using a *t*-test, with a Satterthwaite's modification where appropriate.

Results

Plasma samples for cortisol analysis were received from 562 patients. All except 31 were in-patients at the time of the test. Nineteen patients had to be omitted from the study: two because their concomitant medication was thought to have affected the DST, three because of a severe physical illness, three because of incomplete clinical data and 11 because their diagnosis did not conform to the RDC criteria for major depressive disorder. Plasma was also received and assayed for cortisol from 246 controls.

Table I indicates the number of patients in each of the research centres who participated in the study, the sex distribution, the mean age, HRS-D and Newcastle scores.

Most centres (all except Basle, Irvine, Moscow and Utrecht) collected and sent post-dexamethasone plasma samples from healthy controls, 246, altogether.

Table II indicates the mean post-dexamethasone plasma cortisol concentration of patients and controls. When the centre included controls in the study, the statistical comparison has been made between the patient and the control groups within the centre. Since some centres did not provide controls, a statistical comparison has also been made between the patients and the total ($n=246$) control group. In seven out of nine centres in which comparison

TABLE I

Clinical characteristics of patients

	n		*Age*		*Newcastle Score*	
	Male	Female	(years)	HRS-D	≤*5*	≤*6*
Basle	13	26	57.1±2.3	24.2±1.0	14	25
Brussels	20	16	47.7±2.2	24.8±1.1	14	22
Budapest	4	44	53.6±1.9	24.3±0.8	1	47
Casablanca	8	4	38.3±2.7	28.5±1.3	2	10
Copenhagen	4	10	49.4±3.6	15.0±1.0	9	5
Epsom	28	91	59.4±1.3	22.2±0.4	41	78
Irvine	12	14	38.2±2.9	21.6±1.4	4	22
Lucknow	16	9	37.9±1.5	25.3±1.0	5	20
Moscow	9	45	48.2±2.1	21.4±0.7	5	49
Munich	11	50	48.6±1.8	21.4±0.6	25	36
Nagasaki	17	11	43.7±2.4	17.1±2.0	3	25
Sapporo	31	21	46.8±1.8	19.6±0.8	22	30
Utrecht	9	20	45.0±2.1	22.7±1.1	10	19

Results expressed as means ± s.e.m.

TABLE II

Post-dexamethasone plasma cortisol concentration (ng/ml) and percentage of abnormal responses of patients and controls

	Patients		*Controls*	
	Plasma concentration	*% Abnormal response*	*Plasma concentration*	*% Abnormal response*
Basle	82.2±10.9 †	59	—	—
Brussels	93.6±12.3*†	69	13.4±2.4	0
Budapest	65.8± 7.2*†	54	21.3±3.3	7
Casablanca	37.4±12.1	25	26.2±8.0	14
Copenhagen	116.5±23.2*†	71	16.5±1.8	0
Epsom	101.0± 6.7*†	70	26.2±2.6	11
Irvine	52.6± 9.0 †	42	—	—
Lucknow	46.8± 8.2 †	48	48.9±9.4	40
Moscow	22.5± 4.2	15	—	—
Munich	91.1±10.1*†	62	25.0±3.5	8
Nagasaki	42.0±10.9*	21	13.5±1.0	0
Sapporo	39.3± 6.8*	23	16.3±2.0	3
Utrecht	66.8±11.7 †	41	—	—
All centres ($n=543$)	70.6± 2.9 †		($n=246$) 24.9±1.7	

Results expressed as means ± s.e.m.

*Patients significantly different ($P<0.02$) from controls from same centre

†Patients significantly different ($P<0.02$) from all controls

was possible, the mean post-dexamethasone plasma cortisol concentration of the patients was significantly higher than the mean of the control group. In 12 out of 13 centres, the mean post-dexamethasone plasma cortisol concentration of the patients was higher than the mean of the total control group.

Table II also indicates the percentage of abnormal responses to dexamethasone administration in patients and controls, using the 50 ng/ml cut-off point.

Since previous reports indicated a relationship between the endogenous nature of the illness and an abnormal DST response (Coppen *et al*, 1983) it was decided to check whether this relationship was still evident in such a large group of depressed patients. The result of the inter-rater reliability of Newcastle Scale scoring, prior to the onset of this study, was considered satisfactory. However, only five centres provided a large enough number of patients in each of the two categories of endogenous (Newcastle score ⩾6) and non-endogenous (Newcastle score ⩽5) illness. Four of these five centres had a higher mean post-dexamethasone plasma cortisol concentration in the endogenous patients than in the non-endogenous patients, but the difference was only statistically significant in the Epsom and Brussels centres. In these two centres the mean post DST plasma concentrations were respectively 66 and 62 for the non-endogenous patients and 120 and 114 for the endogenous.

All patients had been classified according to ICD-9. The depressive patients in category 296 (who come nearest to the definition 'endogenous') were compared to patients in all other categories of depression. Means for the ICD 296 groups were not significantly different from the means of the other ICD categories of depression except in the case of the Epsom centre where ICD 296 had a significantly higher mean (109.8 ng/ml) than the others (66.0 ng/ml).

There was no significant difference in the mean post-dexamethasone cortisol concentration or in the proportion of abnormal responders between groups of in-patients and out-patients in those centres which contained both.

Discussion

These results support the view that post-dexamethasone plasma cortisol concentration is higher among depressive patients than among controls. There is, however, variation between the centres with the highest mean plasma cortisol concentration (116.5 ng/ml) and the lowest mean (22.5 ng/ml). This variation is also evident in the percentage of abnormal responses (71% to 15%) among the patients.

Two Japanese centres had a low mean post-dexamethasone plasma concentration. However, the means for their control groups are also very low. This indicates that it is important for any centre undertaking a DST comparison to seek controls locally. It also points to the inappropriate nature of a universal cut-off point to define an abnormal DST response.

In general, the controls were younger than the patients; this does not account for the difference in DST response. When subsamples of younger patients were matched for age with controls from the same centre, the difference in DST response remained as marked as for the older group.

The results of this collaborative study point to the wide incidence of abnormal DST responses among depressive patients. They do not contribute to the question of the specificity of that response. They also do not indicate whether normal or abnormal DST responses are related to particular subgroups of depressive patients. The association between an abnormal DST response and an endogenous type of depression (as defined by the Newcastle classification) finds support in a similar study reported by the Copenhagen Centre (Dam *et al*, 1985) of 91 depressive patients tested in a similar way but not included in the present investigation: 50% of the endogenous and only 30% of the non-endogenous patients gave an abnormal response to the DST.

Sex distribution, age and severity of the illness were examined but did not account for the differences in post-dexamethasone cortisol concentration between the centres. Other factors such as ethnic features, psychopathological characteristics, geographical location, climate, etc. probably influenced the results of the DST. A more detailed analysis of the data is already under way.

Conclusion

Most of the healthy control population samples from various countries responded normally to the DST. A significantly larger proportion of patients suffering from major depressive illness failed to respond normally and the present findings confirm that an abnormal DST response is one of the more robust biological characteristics of that illness.

Whether this subgroup of patients with an abnormal DST response represents a homogeneous category with regard to treatment or to other clinical and biological characteristics remains to be demonstrated by further research.

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References

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

CARROLL, B. J., MARTIN, F. I. R. & DAVIES, B. M. (1982) The dexamethasone suppression test for melancholia. *British Journal of Psychiatry*, **140**, 292–304.

COPPEN, A., ABOU-SALEH, M., MILLN, P., METCALFE, M., HARWOOD, J. & BAILEY, J. (1983) Dexamethasone suppression test in depression and other psychiatric illness. *British Journal of Psychiatry*, **142**, 498–504.

DAM, H., MELLERUP, E. T. & RAFAELSEN, O. J. (1985) The dexamethasone suppression test in depression. *Journal of Affective Disorders*, **8**, 95–103.

HAMILTON, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Clinical and Social Psychology*, **6**, 278–296.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1978) Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.

WOOD, K., HARWOOD, J. & COPPEN, A. (1983) Technique and accuracy of the dexamethasone suppression test. *Archives of General Psychiatry*, **40**, 585.

WORLD HEALTH ORGANIZATION (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases*. Geneva: WHO.

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The Structure of Depressive Symptoms in the Elderly

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In a structured sample of 100 male and 100 female minimally impaired patients, aged 60 years and over, females were more depressed. Varimax factor analysis demonstrated four factor groupings which have clinical relevance – Depression, Anxiety, Cognitive impairment, and Psychosomatic disorder; their relative importance is different in males and females. Analysis of variance of the scores of clusters generated by cluster analysis demonstrated four groups of subjects – normal, mildly depressed, moderately depressed with borderline dementia and disability, and severely depressed with moderate dementia and frank disability. In the mildly and moderately depressed, symptoms of anxiety predominated.

Depression may refer to mood, symptoms, syndromes or disease (Calloway, 1982) and patients may be labelled 'depressed' as a result of changes in any of these categories. The classification of depressive illness is confused (Kendall, 1976), but there is general agreement on a basic division into Types A and B. Type A ('psychotic'/'endogenous') denotes severe depression with diurnal variation of mood, guilt, retardation, severe insomnia and loss of insight. Type B ('neurotic'/'reactive') denotes milder illnesses, prone to fluctuation and lacking the characteristic features of Type A. In describing the symptoms of a community, it is not necessary to make the *a priori* assumption that Type A/B classification is either hierarchical or dichotomous.

The prevalence of clinical diagnosis of depression is increased in the elderly, but the increase of prevalence of depressive symptoms is disproportionately greater. Of 'normal' (functionally normal for age) people aged over 65 years, 44% had a Self-rating Depression Scale (SDS) score above the SDS criterion for depression. It was concluded that "the normal elderly patient may feel depressed . . . but not enough to show measurable changes" in functional performance (Zung, 1967, 1972). This suggests that depressive symptoms may be more common in the elderly, even in the absence of clinical depression, but no symptomatic structure has been described in the normal elderly, comparable to that seen in the depressed.

If the difference between the normal and the depressed is one of intrinsic type, i.e. if the division is dichotomous, mutually comparable symptomatic structures could not be expected. However, if this difference is merely one of degree, i.e. if the division is hierarchical, then it should be possible to identify a symptomatic structure common to both.

Some community studies of 'depression' have described heterogenous samples which include patients with depressive illness, those with depressive syndrome in the absence of disease, those with depressive symptoms only, and those with simple altered mood. It has been concluded (Finestone & Blazer, 1982) that, while some elderly patients experience late-life dysphoria (dejection, despondency or unhappiness) in the absence of frank disease, their symptoms do not render them unable to maintain their usual activities and they do not require acute medical intervention.

Factor analysis of depressive symptoms describes patterns of association between them. On the basis of factor scores, it may be possible to allocate to diagnostic groups those patients who show highly polarised factor scores, but the majority of mildly or moderately impaired patients cannot easily be allocated on this basis. However, factor analysis is inappropriate for eliciting clinical syndromes which may be more appropriately described by discriminant function analysis (Hamilton, 1960).

Factor analysis has since been used widely to characterise groups of associated symptoms (Kiloh & Garside, 1963; Carney *et al*, 1965; Mowbray, 1972; Gurland *et al*, 1976; Giambra, 1977; Pull *et al*, 1979; Overall, 1980), which may or may not correspond to clinical entities. Most of the studies have demonstrated a Factor One, which corresponds to Hamilton's General Factor of Depressive Illness, and which is commensurate with overall severity of symptoms. Some have further demonstrated a second factor, which may describe the dichotomy between Type A and Type B depression, but allocation of patients to diagnostic groups outside this simple dichotomy is equivocal.

Mowbray (1972) based his analysis on a consideration of principal components and pointed out that Factor One is unipolar, in contrast to all further factors, which are bipolar. It is inherent to the technique of principal component analysis (PCA) that Factor One is unipolar and others bipolar, and that all variables have a complexity greater than 1 (Kim, 1975). In other words, every variable loads onto more than one factor, and further comment on the nature of underlying phenomena cannot be based on this differentiation. In PCA, differences in the form of a factor do not necessarily imply differences in form in the underlying dimension. (The concept of polarity of factors seen in factor analytical studies should not be confused with unipolar and bipolar clinical concepts of depressive illness.)

Hamilton (1960) provided a rotated factor matrix "For the interest of those factorists who have a taste for . . . simple structures". In Varimax rotation, "The clustering or patterning of . . . variables into . . . groups is more obvious after the rotation than before, even though the relative position or configuration of the variables remains unchanged. In the unrotated solution, every variable is accounted for by two (*or more*) significant common factors, while in the rotated solution each variable is accounted for by a single significant common factor. Therefore, the rotated factor loadings are conceptually simpler than the unrotated ones" (Kim, 1975). It is conceptually simpler to adopt a method of analysis which ascribes each variable to only one factor.

Hamilton presents an orthogonal rotation which presupposes that all factors, and therefore dimensions, are independent of each other. When multiple dimensions are orthogonal, "variation along each is independent. Description of position on one such dimension says nothing as to position on any of the others and has very different implications from membership of one of a set of mutually exclusive groups" (Paykel, 1971).

While factor analysis can distinguish between hierarchical and dichotomous models of depressive symptoms, it cannot differentiate dimensional (quantitative) and categorical (qualitative) mechanisms within these models (Eysenck, 1970). Factor analysis describes the inter-relationship of symptoms in a sample; it does not demonstrate interrelationships of subjects or allow their division into groups.

Cluster analysis provides methods of allocating subjects, on the basis of their factor scores, to groups which are independent of previous classification. The similarity of individuals within groups, and the difference between groups, is maximised, and this provides optimal categorisation of subjects. Cluster analysis has been used for the classification of depressed patients (Paykel, 1971) and of the mentally ill in general (Everitt *et al*, 1971); detailed reviews of clustering techniques may be found in Cormack (1971) and Everitt (1974).

Method

A study was made in 1979 of 200 elderly patients in the community (Griffiths *et al*, 1987); 100 males and 100 females were selected to provide 20 men and 20 women in each quinquennium from 60 to 79 years and a similar group aged 80 years and over. All these patients were assessed for depression and dementia using a modification of the Hamilton Depression Rating Scale (HDRS) (Hamilton, 1960) and the Mental Test Score (MTS) (Hodkinson, 1973). The HDRS was modified to produce a semi-structured interviewer-assisted questionnaire (Griffiths *et al*, 1987) to standardise the assessment (available on application to authors).

The 200 'good' subjects included 51 whose HDRS was 14 or more, (our criterion for 'depression') and/or who had an MTS of 28 or less, (our criterion for 'dementia').

Factor analysis was performed on all 200 subjects and also separately on males and females.

Subjects were allocated to groups by cluster analysis of their rotated factor scores; differences in factor score profiles between groups were tested by analyses of variance.

Statistical techniques

The grouping of HDRS elements was assessed initially by PCA, with iteration, of the Pearson correlation matrix. Those factors having an eigenvalue greater than 1 were subjected to Varimax rotation; variable loadings were selected to maximise individual eigenvalues.

The significant critical value of each item loading in the Varimax-rotated factor matrices was calculated according to the method of Burt and Banks (Child, 1970). To guard against over-optimistic inclusion of marginal loadings, intentionally strict critical values were selected, corresponding to a probability $P<0.001$.

Subjects were allocated severally to clusters, on the basis of their HDRS item scores, by three different methods of cluster analysis: Ward's (1963), *k*-means (MacQueen, 1966) and the Normix method (Wolfe, 1965, 1967, 1970). The first two methods require similarity coefficients, and as such the Euclidean distances were specified; they make different initial assumptions of the structure of the data. The third method assumes a mixture of multivariate normal distributions with unequal covariance matrices, and uses the method of maximum likelihood to estimate the parameters. The subjects are then assigned to the cluster for which the probability of belonging is highest. These three methods are sufficiently dissimilar to test the validity of the resulting clusters.

F ratios were calculated by one-way analysis of variance (ANOVA) with each factor in turn as the dependent variable, and the cluster as independent. Multiple analysis of variance (MANOVA) was performed between the cluster as the independent variable, and all factors simultaneously as dependent variables. The association between sex and cluster was tested using χ^2. A two-way ANOVA was performed for each factor as the dependent variable, with sex and cluster as independent variables.

A one-way ANOVA was performed for each of the variables HDRS, MTS, usual activities score (UA) (Griffiths *et al*, 1987) and age as dependent variables, and the cluster as independent. Where a significant difference was observed, the relationships between the clusters for these variables was determined by Newman–Keuls test (Armitage, 1971).

Computing techniques

Data were analysed on an ICL 2988 computer. Factor analysis and analyses of variance were derived from the Statistical Package for the Social Sciences (SPSS II [Nie *et al*, 1975]) and the SPSS Update 7–9 (Nie & Hull, 1981), and were performed by subprograms 'Factor' and 'ANOVA'. Cluster analyses were derived from the statistical package CLUSTAN (Wishart, 1978) and were performed by subprogram 'Ward', '*k*-means' and 'Normix'. The χ^2 tests were performed manually. The Newman–Keuls tests were performed by the SNK routine of the SPSS subprogram 'Oneway'.

Results

Depression and dementia here refer to categories defined according to criteria established for rating scales (Griffiths *et al*, 1987) and not to clinical diagnoses. The criterion for depression was HDRS>13, and that for dementia, MTS<29. The numbers of subjects in each group are shown in Table I.

The severity of depression depends on the number of depressive symptoms and on the severity of each of those symptoms; the HDRS total score depends on the number of items answered positively, and on the degree of impairment revealed by each positive item.

PCA of the elements of the HDRS revealed three items which produced unrelated factors, i.e. they produced factors which were unique to themselves and derived no loading from other items; these items occurred on 1% or less of responses and were items 19, 20, and 21 (Griffiths *et al*, 1987) which are 'Depersonalisation and derealisation', 'Ideas of reference and persecution' and 'Obsessional ideas and behaviour'. Therefore, these items were excluded by us from repeat PCA and subsequent analysis, as they were by Hamilton (1960). In addition, item 14, 'Disturbance of libido', had been eliminated before data collection. Further individual deletion of items of the HDRS did not alter the number or pattern of principal components with an eigenvalue greater than 1; therefore, this 17-item scale was subjected to further analysis. The 17-item scale, so derived, corresponds to that on which Hamilton (1960) based his analysis, except that item 18, 'Diurnal variation', was not excluded by us.

Factor analysis

Separate PCA with iteration of the correlation matrices of the HDRS for all 200 'good' patients and for females only demonstrates four factors, which account for 60.7 and 62.6% of total variance respectively. In males only, six factors are seen, which account for 68.7% of the variance; but the last two factors here account for only 9.6% of the variance and have very low factor loadings. Therefore, it was decided to retain only the first four factors: their interpretation is equivocal and bears little relationship to clinical syndromes, although they are not dissimilar to those of Hamilton (1960), Mowbray (1972) and others. All these principal components have a complexity of greater than 1, and thus describe elements of more than one underlying dimension.

Varimax rotation produces an equally scattered distribution, although minimising the bipolarity seen in the unrotated matrix, and demonstrates factor groupings which resemble clinical syndromes (Table II). Variables having a less than critical loading (i.e. less than 0.23, which corresponds to a probability $P<0.001$) by the method of Burt and Banks (Child, 1970) are excluded from consideration in Table II. Primary loadings are shown in normal type; secondary loadings are in italics.

The four factors thus produced for the whole group, for females and for males, are similar in composition, although their order of emergence is different, and they differ in importance between the sexes. We have designated these four factors Anxiety, Depression, Cognitive impairment, and Psychosomatic disorder.

Factor A is a well-defined anxiety factor, common in all aspects to both sexes. Factor B, the depression factor, is first in order for the females, with high loadings, whereas

TABLE I

Prevalence of depression and dementia in 100 male and 100 female elderly people

	Depressed only	*Demented only*	*Depressed and demented*	*Normal*	*Total*
Male	1	16	2	81	100
Female	15	10	7	68	100
Total	16	26	9	149	200

TABLE II
Item allocation to factor grouping, Varimax analysis of HDRS scores of 200 subjects

		All 200		*100 Males*		*100 Females*	
Factor	*HDRS item*	*Loading*	*Variance %*	*Loading*	*Variance %*	*Loading*	*Variance %*
A Anxiety	05 Middle insomnia	0.601	69.0	0.425	21.4	0.733	12.6
	09 Agitation	0.576		0.860		*0.387*	
	10 Psychic anxiety	0.551		0.750		*0.406*	
	04 Early insomnia	0.536		0.392		0.429	
	11 Somatic anxiety	0.525		0.461		0.469	
	06 Late insomnia	0.522		0.303		0.650	
	18 Diurnal variation	0.405		*0.270*		0.307	
	01 Depression	*0.403*		*(0.200)*		*0.393*	
	13 General somatic	*0.352*		*(0.217)*		*(0.203)*	
	15 Hypochondriasis	*0.314*		*(0.118)*		*(0.213)*	
B Depression	02 Guilt	0.763	13.1	0.475	16.5	0.873	61.8
	03 Suicide	0.751		0.326		0.756	
	01 Depression	0.570		0.833		0.571	
	07 Work and interests	0.479		*0.282*		0.513	
	15 Hypochondriasis	*0.412*		*0.357*		*0.447*	
	09 Agitation	*0.377*		*(0.119)*		0.419	
	10 Psychic anxiety	*0.374*		*(0.129)*		0.461	
C Cognitive impairment	17 Loss of insight	0.774	10.7	0.817	47.7	0.783	9.9
	08 Retardation	0.709		0.672		0.748	
	07 Work and interests	*0.462*		0.539		*0.380*	
	13 General somatic	*0.356*		0.586		*0.231*	
	15 Hypochondriasis	*0.331*		0.539		*0.259*	
D Psychosomatic disorder	12 Gastrointestinal	0.676	7.3	0.846	14.4	0.592	15.7
	15 Hypochondriasis	0.587		*0.323*		0.695	
	13 General somatic	0.508		*0.257*		0.649	
	16 Weight loss	0.412		0.394		0.402	
	17 Loss of insight	*0.368*		*0.307*		*0.458*	
	18 Psychic anxiety	*0.260*		*0.261*		*0.368*	

Primary loadings are shown in normal type; secondary loadings are shown in italics. Non-significant loadings are shown in brackets.

TABLE III
Mean values of factors A, B, C and D by sex

Factor	*Sex*	*N*	*Mean*	*s.e.*	*t-value*	*d.f.*	*Significant difference between males and females*
A	Males	100	−0.242	0.077	−4.18	198	<0.01
	Females	100	0.242	0.087			
B	Males	100	−0.170	0.045	−2.79	198	<0.01
	Females	100	0.170	0.113			
C	Males	100	−0.075	0.076	−1.22	198	NS
	Females	100	0.075	0.097			
D	Males	100	−0.123	0.059	−2.17	198	<0.05
	Females	100	0.123	0.097			

for the males it is slower to emerge, and with low loadings on Agitation and Psychic anxiety. Factor C, Cognitive impairment, is first to emerge for the males, but is only fourth for the females, having low loadings on Guilt and Diurnal variation. Factor D, Psychosomatic disorder, is more apparent in females than in males, in whom low loadings are seen for Work and interests and Somatic anxiety.

Females scored more highly on every factor than did males (Table III). This is significant for all factors except factor C, Cognitive impairment.

Cluster analysis

The three methods used each produced a clear four-cluster solution. The clusters generated by Ward's method and by the *k*-means method were identical; those generated by the Normix method were slightly different, in that a total of 30 subjects were allocated differently among the first two clusters of the first two solutions. Therefore the solution produced by the first two methods was adopted for subsequent analysis, and the mean values for each factor, by cluster, are shown in Table IV.

TABLE IV

Mean values of factors A, B, C and D by cluster (Ward's and k-means solutions) (standard deviations in brackets)

Cluster	*n*	*Factor A*	*Factor B*	*Factor C*	*Factor D*
1	110	−0.59	−0.24	−0.10	−0.19
		(0.38)	(0.28)	(0.60)	(0.46)
2	55	0.75	−0.01	−0.49	−0.12
		(0.59)	(0.65)	(0.34)	(0.64)
3	27	0.64	0.25	0.87	0.46
		(0.81)	(1.44)	(1.25)	(1.24)
4	8	0.82	2.49	1.79	1.89
		(0.90)	(1.27)	(0.91)	(0.90)
F-value		97.843	39.072	42.370	27.928
d.f.		3,196	3,196	3,196	3,196
P		<0.01	<0.01	<0.01	<0.01

The four cluster mean values were significantly different for every factor. Examining all four factors simultaneously, i.e. forming a vector of dependent variables, MANOVA produced a Wilks' lambda of 0.0522 (d.f. 4, 3, 196) and *F* value of 87.43 (d.f. 12, 510); this was highly significant ($P<0.00001$), and the factor structure thus shows highly significant clustering.

Cluster 1, the largest, has negative mean values for every factor, an indication that it is composed mainly of unimpaired subjects. Cluster 2 has a high positive mean for factor A, Anxiety, and negative values for the other factors. Cluster 3 has high positive means for factors A and C, Anxiety and Cognitive impairment. Cluster 4 has high positive means for all four factors, and comprises eight subjects, all females.

TABLE V

Sex composition of the four clusters (Ward's and k-means solutions)

	Cluster 1	*Cluster 2*	*Cluster 3*	*Cluster 4*	*Total*
Males	68	23	9	0	100
Females	42	32	18	8	100
Total	110	55	27	8	200

The sex composition of the four clusters is seen in Table V. There is a significant association between sex and cluster ($\chi^2_{(3)}=18.6$, $P<0.001$); female subjects are found in higher proportion than males in clusters 2, 3 and 4, but in lower proportion in cluster 1.

The results of two-way ANOVA for each factor as the dependent variable, and sex and cluster as independent variables, are presented. The cluster effect on every factor is highly significant, but the sex effect is not significant for factors A and B.

For factors B and C, there is significant cluster by sex interaction. Cell means for these factors are shown in Table VI. Clusters 3 and 4 (composed of females only) are the only clusters in which there is a significant difference between the sexes. The clusters differ in respect to HDRS, MTS and UA score (Griffiths *et al*, 1987) (Table VII). The MTS is expressed as dMTS, the value obtained by subtracting the MTS value from the maximum obtainable (34), so that the direction of increasing impairment is maintained in all three test variables. Increased values of UA indicate increased disability.

TABLE VI

Mean values of factors B and C by sex and cluster

	Cluster 1	*Cluster 2*	*Cluster 3*	*Cluster 4*
Factor B[1]				
Males	−0.21	−0.00	−0.26	0.00
Females	−0.28	−0.01	0.50	2.49
Factor C[2]				
Males	−0.17	−0.49	1.68	0.00
Females	0.01	−0.49	0.46	1.79

1. $s^2=0.479$, based on 193 d.f.
2. $s^2=0.429$, based on 193 d.f.

Each cluster is significantly different from all others with respect to HDRS score; this might have been expected, since the clusters were generated using the HDRS scores. The clusters do not differ significantly in respect of age. However, for both UA and dMTS, clusters 3 and 4 differ significantly from each other and from clusters 1 and 2 combined.

TABLE VII
Analysis of variance of HDRS, MTS, UA and age in four clusters

	Cluster				
	1 *n=110* *(68m, 42f)*	*2* *n=55* *(23m, 32f)*	*3* *n=27* *(9m, 18f)*	*4* *n=8* *(0m, 8f)*	*ANOVA F-value Significance (P)*
HDRS±s.d.	2.9±1.8	9.1±2.5	14.2±3.4	27.6±3.0	436.0,
(range)	(0–8)	(6–16)	(8–22)	(24–33)	<0.001
dMTS±s.d.	3.0±2.5	2.6±2.2	5.4±4.7	9.9±6.9	17.3,
(range)	(0–11)	(0–8)	(0–16)	(3–17)	<0.001
UA±s.d.	3.5±4.1	3.8±4.3	7.2±4.9	12.3±2.9	15.3,
(range)	(0–18)	(0–21)	(0–20)	(9–16)	<0.001
Age±s.d.	72.2±7.2	70.0±7.2	73.8±5.9	73.1±6.0	2.1,
(range)	(60–87)	(60–88)	(65–85)	(68–83)	>0.1
					d.f. 3196

All clusters are significantly different for each variable (Newman–Keuls test, $P<0.01$), except where they are bracketed together (NS)

Discussion

In the elderly population, depressive symptoms are disproportionately more prevalent than clinical diagnoses (Zung, 1967), and clinical diagnosis is subject to considerable error (Cooper *et al*, 1972). The clinical diagnosis of depression depends on the presence and severity of the symptoms of a definitive syndrome plus the physical, psychological and social disabilities manifest.

In their attempts to characterise depression by PCA, previous authors have identified a 'General factor of depressive illness' which correlates well with total score and resembles Type A ('psychotic'/'endogenous') depression. Subsequent factors are less amenable to interpretation; PCA trivialises all factors except Factor 1, by maximising the variance loaded on to that factor. Subsequent factors have a complexity greater than unity; factors overlap, and individual factors do not, in practice, represent unique underlying syndromes. In factors subsequent to Factor 1, there is a duality, with high positive and high negative saturation, which misleadingly suggests bipolarity of the syndromes they attempt to describe.

Varimax rotation distributes the accountable variance and the item allocation to a factor more evenly across the factor field. The duality engendered by the presence of significant negative saturation in PCA is largely eliminated by the Varimax method; the negative loadings are trivial, implying the absence, in these results, of a true bipolarity in factor groupings. PCA factor groupings in this study approximate to those of previous authors, despite the complexity of the grouping matrix.

Our Varimax technique produces a simpler structure, and the factor groupings derived by this technique have obvious clinical interpretation. The four factors A (Anxiety), B (Depression), C (Cognitive impairment), and D (Psychosomatic disorder) are relevant to the classification of affective disorders in the elderly. However, the Anxiety factor contains a significant loading for depression. The Depression factor contains a significant loading for hypochondriasis – particularly relevant in the elderly. Although the Cognitive impairment factor is derived from the HDRS scores, all subjects with high scores for this factor also had impaired mentation, as measured by the MTS. The Psychosomatic disorder factor is relevant to depression in the elderly (Post, 1965), and we observed that high scores on this factor were frequently associated with high scores on the Depression factor.

The females in our sample account for 65% of the depressive symptoms reported and 88% of the defined Depressed patients. In females, the Depression factor accounts for the greatest part of the explained variance (61.8%), followed by Psychosomatic disorder (15.7%), Anxiety (12.6%) and Cognitive impairment (9.9%). In males, Cognitive impairment is most important (47.7%), followed by Anxiety (21.4%), Depression (16.5%) and Psychosomatic disorder (14.4%).

It has been suggested (Oltman *et al*, 1980) that the types of depression in the elderly may differ by sex; in a sample in which depressive symptoms are apparently twice as prevalent in females as in males (Griffiths *et al*, 1987), it is surprising that the pattern and structure of factors is not radically different, although their relative importance is changed.

Although diurnal variation has been accepted as a characteristic of some types of depression, it does not assume a fixed position in this factor field, and was excluded by Hamilton (1960). The HDRS does not distinguish between morning and evening dysphoria in the item score.

Work and interests in both males and females is associated with the factor with highest correlation with total score. In females, this is the Depression factor, while in males the association is with Cognitive impairment. This may be related to different changes in patterns of life events in males and females on retirement.

Although we do not present separate results for our subset of 149 'normal' subjects, (MTS > 28, HDRS < 14), the factor structure produced by separate analysis of this subset is essentially similar to that for the whole sample.

Community studies of depression, using factor analytical techniques, identify both patients with depressive illness and other patients with less severe syndromes of late-life dysphoria (Blazer & Williams, 1980; Finestone & Blazer, 1982). Our study demonstrates clinically relevant factors which are identifiable components of depression in the elderly community.

In our data, the patterns of association of depressive symptoms into factors are similar in the 'normal' and 'depressed' groups, which suggests that the threshold of depression as a clinical syndrome is not an intrinsic dichotomy. However, to test this, it would be necessary to perform factor analysis on larger groups for each HDRS score. Having established the association between the items of the HDRS, we attempted by cluster analysis to identify coherent groups of subjects, on the basis of their factor scores. The various techniques of cluster analysis (Cormack, 1971; Everitt, 1974) attempt to construct clusters whose members are as similar as possible and differ from the members of other clusters. The most serious criticism of cluster analysis lies in the difficulty of establishing the validity or significance of derived clusters, especially in the absence of objective external criteria. However, by adopting three different clustering techniques, which make different assumptions of the structure of the data, we hoped to provide some measure of validation.

All three clustering methods produced a clear four-cluster solution; the results of Ward's method and the *k*-means method were identical, while the Normix results showed some small differences, but largely agreed with the other two methods. The four clusters which emerged were characterised as Normal, Anxious, Anxious and cognitively impaired and Impaired on all factors. However, a closer examination of factor A (Anxiety) shows that it contains a significant secondary loading for depression; thus, the cluster we have here characterised as Anxious might more properly be described as anxious and mildly depressed. Consideration of the mean rating scale scores of each cluster (Table VII) confirms their classification as normal, mildly depressed, moderately depressed with borderline dementia and disability, and severely depressed with moderate dementia and frank disability.

Thus, the clusters may be retrospectively justified as having direct relevance to the clinical classification of psychiatric disorder in the elderly community. This technique provides clear differentiation of the severely impaired, while indicating two borderline groups of subjects—those with dysphoric symptoms only, and those with depression and disability, warranting clinical diagnosis, but in whom the primary disorder was not clear.

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References

ARMITAGE, P. (1971) *Statistical Methods in Medical Research*. Oxford: Blackwell.

BLAZER, D. & WILLIAMS, C. D. (1980) Epidemiology of dysphoria and depression in an elderly population. *American Journal of Psychiatry*, **137**, 439–444.

CALLOWAY, S. P. (1982) Endocrine changes in depression. *Hospital Update*, **8**, 1343–1350.

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of E.C.T. response. *British Journal of Psychiatry*, **111**, 659–674.

CHILD, D. (1970) *The Essentials of Factor Analysis*. London: Holt, Rinehart & Winston.

COOPER, J. E., KENDELL, R. E., GURLAND, B. J., SHARPE, L., COPELAND, J. R. M. & SIMON, R. (1972) *Psychiatric Diagnosis in New York and London*. London: Oxford University Press.

CORMACK, R. M. (1971) A review of classifications. *Journal of the Royal Statistical Society, Series A*, **134**, 321–367.

EVERITT, B. S. (1974) *Cluster Analysis*. London: Heinemann.

——, GOURLAY, A. J. & KENDELL, R. E. (1971) An attempt at validation of traditional psychiatric syndromes by cluster analysis. *British Journal of Psychiatry*, **119**, 399–412.

EYSENCK, H. J. (1970) The classification of depressive illness. *British Journal of Psychiatry*, **117**, 241–250.

FINESTONE, D. H. & BLAZER, D. (1982) Clinical clues to depression in the elderly patient. *Geriatric Medicine Today*, **1**, 87–94.

GIAMBRA, L. M. (1977) Independent dimensions of depression: a factor analysis of three self-report depression measures. *Journal of Clinical Psychology*, **33**, 928–935.

GRIFFITHS, R. A., GOOD, W. R., WATSON, N. P., O'DONNELL, H. F., FELL, P. J. & SHAKESPEARE, J. M. (1987) Depression, dementia and disability in the elderly. *British Journal of Psychiatry*, **150**, 482–493.

GURLAND, B. J., FLEISS, J. L., GOLDBERG, K., SHARPE, L., COPELAND, J. R. M., KELLEHER, M. J. & KELLETT, J. M. (1976) A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly: I. The geriatric mental state schedule. II. A factor analysis. *Psychological Medicine*, **6**, 451–459.

HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

HODKINSON, H. M. (1973) Mental impairment in the elderly. *Journal of the Royal College of Physicians, London*, **7**, 305–317.

KENDELL, R. E. (1976) The classification of depressions: A review of contemporary confusion. *British Journal of Psychiatry*, **129**, 15–28.

KILOH, L. G. & GARSIDE, R. F. (1963) The independence of neurotic depression and endogenous depression. *British Journal of Psychiatry*, **109**, 451–463.

KIM, J-O. (1975) Factor analysis. In *Statistical Package for the Social Sciences* (eds N. H. Nie, C. H. Hull, J. G. Jenkins, K. Steinbrenner & D. H. Bent) 2nd ed. New York: McGraw Hill.

MACQUEEN, J. (1966) Some methods of classification and analysis of multivariate observations. *Proceedings of the 5th Berkley Symposium*, **1**, 281–297.

MOWBRAY, R. M. (1972) The Hamilton rating scale for depression: a factor analysis. *Psychological Medicine*, **2**, 272–280.

NIE, N. H. & HULL, C. H. (1981) *SPSS Update 7–9: New Procedures and Facilities for Releases 7–9*. New York: McGraw Hill.

——, ——, JENKINS, J. G., STEINBRENNER, K. & BENT, D. H. (1975) *Statistical Package for the Social Sciences* 2nd ed. New York: McGraw Hill.

OLTMAN, A. M., MICHALS, T. J. & STEER, R. A. (1980) Structure of depression in older men and women. *Journal of Clinical Psychology*, **36**, 672–674.

OVERALL, J. E. (1980) Correlational structure of a new self-rating scale for psychiatric patients. *Journal of Clinical Psychology*, **36**, 616–624.

PAYKEL, E. S. (1971) Classification of depressed patients: A cluster analysis derived grouping. *British Journal of Psychiatry*, **118**, 275–288.

POST, F. (1965) *The Clinical Psychiatry of Late Life*. Oxford: Pergamon Press.

PULL, C. B., PICHOT, P., PULL, M.-C. & VON FRENCKELL, R. (1979) The principal dimensions of manifest depression: a factor analysis of manifest depressive symptomatology. *Neuropsychobiology*, 5, 207–212.

WARD, J. H. (1963) Hierarchical grouping to optimize an objective function. *Journal of the American Statistical Association*, **58**, 236–244.

WISHART, D. (1978) *Clustan 2.1* 3rd edition. Program Library Unit, Edinburgh University.

WOLFE, J. H. (1965) A computer program for the maximum likelihood analysis of types. Technical Bulletin, 65-15, US Naval Personnel Research Activity, San Diego.

—— (1967) Normix: computational methods for estimating the parameters of multi-variate normal mixtures of distributions. Research Memorandum, SRM 68-2, US Naval Personnel Research Activity, San Diego.

—— (1970) Pattern clustering by multi-variate mixture analysis. *Multivariate Behavioural Research*, 5, 329–350.

ZUNG, W. W. K. (1967) Depression in the normal aged. *Psychosomatics*, **8**, 287–292.

—— (1972) How normal is depression? *Psychosomatics*, **13**, 174–178.

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DSM-III Personality Disorder Clusters in Three Populations

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DSM-III personality disorder clusters had not previously been tested for their ability to distinguish between different population groups. A major obstacle – different demographic characteristics of different populations – was overcome by using logistic regression. Three out-patient psychiatric referral groups (chronic pain, disability applicants and competency to stand trial) provided subjects; adjustments were made for age, race, sex and education. The groups were significantly different in the adjusted prevalence of DSM-III personality disorder clusters. Clinical association between personality disorder and drug or alcohol abuse was again confirmed.

The third edition of the *Diagnostic and Statistical Manual for Mental Disorders* (DSM-III) (American Psychiatric Association, 1980) represents a major leap forward in the methodology of American psychiatry. Its major innovation is in standardising and specifying the inclusion and exclusion criteria for each psychiatric disorder, which were field-tested for reliability. This helps rectify the problem that arises when psychiatrists use the same diagnostic label to refer to different patients, or different labels to refer to similar patients. Another innovation was the use of several axes to diagnose different types of disorders or information. The first axis is used to diagnose clinical syndromes such as affective disorders, schizophrenia, anxiety disorders and life circumstance problems; the second to diagnose personality and some specific developmental disorders; the third, physical disorders; the fourth, severity of psychosocial stressors; and the fifth, highest level of adaptive functioning for the past year.

One of the great values of DSM-III is that, by defining specifically the inclusion and exclusion criteria of the various disorders, the association between them can often be determined accurately. This allows empirical evidence to demonstrate important clinical associations, and perhaps guide the way for future revisions of diagnoses. Frances *et al* (1984) demonstrated considerable overlap of the various DSM-III personality disorders, which raises the possibility of developing a clustering scheme of these disorders in order to classify them more economically.

A potential clustering which has much apparent validity is described in DSM-III. The first group consists of the paranoid, schizoid and schizotypal personality disorders – the asocial personality type. The second group consists of the histrionic, narcissistic, antisocial and borderline disorders – the flamboyant personality type. The third group consists of the avoidant, dependent, compulsive and passive-aggressive disorders – the anxious personality type. However, in spite of their apparent validity, little empirical work has been performed to validate these clusters.

This report examines the association of the presence or absence of a personality disorder with seven Axis I psychiatric diagnostic categories (mental retardation, organic brain syndrome, affective disorder, schizophrenia, alcohol abuse, drug abuse and a combined drug or alcohol abuse category). It also provides a test of validity for the DSM-III personality types by determining whether they can differentiate three different groups of patients.

Method

Subjects were from three population groups: one concerning competence to stand trial ($n = 390$), one with chronic pain ($n = 43$) and one of applicants for disability benefits ($n = 55$). All analyses were adjusted for age, race, sex and level of education and have been reported in detail elsewhere (Reich *et al*, 1983; Reich, 1985*b*; Reich & Wells, 1985). These populations are representative of previously described groups of a similar nature (Devine & Merskey, 1965; Merskey, 1965; Cooke, 1968; Henn *et al*, 1977; Sikorski & Benedek, 1977); Bluestone & Mellela, 1978; Bluestone & Baskin, 1981).

The forensic group consisted of 390 defendants, referred for evaluation of competency to stand trial at the Yale–New Haven Psychiatric Court Clinic in 1980–1982. Each person was included only once for data collection; if any was referred more than once, only the most recent information was used, but such cases were very few. All were given a semistructured, standardised interview, to ascertain psychiatric diagnosis and other forensic variables. Data were gathered from those previously precoded from the case records. This population was 83.3%

male, 53.8% white and 36.2% black; the average age was 30.8 years, and the average years of education 10.2. To establish reliability, a blind review of the records was performed by one of the authors (J. R.) on 27 cases, with which he was not familiar, for the diagnosis of organic brain syndrome, affective disorder, schizophrenia, mental retardation, presence or absence of a personality disorder, alcohol abuse and drug abuse. An overall kappa of 0.69 was achieved.

The pain group consisted of 43 patients seen by J. R. in 1980–1981 as psychiatric consultant to the University of California, Davis Pain Board. All had an extensive review of records and were given a semistructured interview to elicit a DSM-III diagnosis and information relating to pain. Every patient seen by the board during that period was included. This population was 35% male, 88% white and 7% black; the average age was 43.2 years and the average length of education 12.9 years. Reliability for both Axes I and II was achieved by case review by a senior clinician (Reich *et al*, 1983), with 96% agreement on Axis I and II disorders.

The disability group consists of 55 patients referred to J. R. in 1983, for psychiatric evaluation for receiving social security disability (SSI). The medical records of many were available prior to the interview. All received the same semistructured interview to determine a DSM-III diagnosis and information relating to the disability. All were from the New Haven (Connecticut) metropolitan area. The population was 53% male, 63% white and 21.8% black; the average age was 44.6 years, and the average length of education 10.5 years. Reliability of psychiatric diagnoses was tested by review of medical records and 90% agreement was reached on the primary diagnosis for seven categories (organic brain syndrome, mental retardation, schizophrenia, affective disorder, specific personality disorders, alcohol abuse and drug abuse). As the diagnoses in the disability group were checked by a psychiatrist with a large data base at his disposal, it was felt that bias resulting from secondary gain was controlled adequately.

The diagnoses were all made by DSM-III criteria, using semistructured interview instruments of similar design, which collected information about the diagnosis, and relevant to the special interest (pain, disability or competency to stand trial). The two instruments for the pain and disability groups were designed by J. R., who also performed all the interviews for these groups; he also participated in some forensic interviews and, by studying the instrument and records review, was able to conclude that its results were equivalent to the other instruments, in terms of DSM-III diagnosis. The interview instrument was designed to diagnose both Axis I and II syndromes by DSM-III criteria. Questions relevant to major psychiatric diagnostic groups (e.g. schizophrenia, affective disorder, anxiety, organic brain syndrome, mental retardation, drug or alcohol abuse) were asked, and from these answers specific Axis I diagnoses were arrived at, using DSM-III criteria. After the Axis I disorders were diagnosed, questions were asked about occupational, interpersonal and social functioning and legal difficulty; if necessary, additional questions were asked to diagnose a DSM-III-defined personality disorder definitively. All diagnoses (Axes I and II) were by specific DSM-III diagnoses, and only later grouped together in categories for ease of analysis. The diagnoses for both Axes I and II were reviewed either by a senior clinician (pain group) or by review of records (disability group), with agreement greater than 90%. As there were no comprehensive standardised instruments for measuring Axis II at the time, this represented the state of the art for DSM-III personality disorder diagnoses by clinicians. The forensic group was diagnosed by a different instrument, which asked specific questions about certain symptoms such as depression and psychoticism to aid an Axis I diagnosis, and also questions that would assist an Axis II diagnosis (legal problems, drug usage, impulsivity, ability to relate appropriately to people etc.). All forensic diagnoses were by DSM-III criteria and were also reviewed by a senior clinician. Material from case records was also used to aid in the diagnoses of all groups.

The two semistructured interviews differed in the following ways. The J. R. instrument was designed to help diagnose specific DSM-III diagnostic categories, while the forensic instrument elicited extensive symptom information, which the clinician could then use in his diagnosis of DSM-III disorders. Both provided extensive information on symptoms, with the J. R. instrument organising it into diagnostic categories and the forensic instrument organising it into symptom groups, but as both resulted in DSM-III diagnoses, the end result was the same.

At the time of collection of the data, there was no plan for a personality cluster analysis (which was decided later by the nature of the clinical material); thus, in this respect, all raters were blind. The patients were placed in one of the three personality clusters if they had any diagnosis DSM-III personality disorder; those with such a disorder were placed in only one group. In the few cases where there was more than one personality disorder, patients were placed in the personality cluster which best reflected their most severe symptoms.

Using logistic regression (similar to linear regression), two sets of odds ratios and their associated significance levels were calculated. The first was the association between having any personality disorder and seven Axis I psychiatric diagnostic categories (mental retardation, organic brain syndrome, affective disorder, schizophrenia, drug abuse, alcohol abuse and a combined drug or alcohol abuse category). The second was the ability of a personality disorder, or presence or absence of a personality type (asocial, flamboyant or anxious), to differentiate between the three source groups (forensic, pain and disability). In all analyses, age, race, sex and education were adjusted for, in order to control for confounding. Unfortunately, the sample size was too small to allow an analysis of association between personality disorder types and Axis I diagnosis. The logistic regression program used for these analyses was PROC LOGIST (SAS Institute, 1980).

To reduce the type I (false positive) error that occurs when running multiple tests, the odds ratios for personality disorders and Axis I category were run adjusting for other Axis I disorders, in essence making it one test. It is reported as individual odds ratios for ease of presentation. Similarly only four (instead of 12) analyses were run for the association of personality disorder types and different populations. As the pairwise comparisons gave identical results, they are reported in pairs, again for ease of presentation.

An odds ratio is the proportion of an event in one population, compared with the occurrence of the same event in a second population (for example, if a borderline personality disorder occurs in 10% of population A and 5% of population B, then the A:B odds ratio for borderline personality would be 2. If A should be less than B (say 5 *vs* 10%) then the odds ratio would be less than 1 (in this case 0.5). In general, the higher the odds ratio, or the closer to 0 it is, the greater the chance that it will be significant. However, when the sample size is very small, the logistic regression program may generate a large odds ratio which does not achieve significance.

Results

The unadjusted percentages of seven major Axis I diagnostic groups, where a personality disorder diagnosis was also made, were: mental retardation 10.5, organic brain syndrome 5.3, affective disorder 19.6, schizophrenia 4.1, drug abuse 28.2, alcohol abuse 15.2 and drug or alcohol abuse 22.2. The odds ratios for presence of a personality disorder for each of these categories were: mental retardation 2.1, organic brain syndrome 0.0, affective disorder 0.9, schizophrenia 0.2 ($P<0.001$), drug abuse 2.1, alcohol abuse 1.0 and drug or alcohol abuse 2.1 ($P<0.05$). These odds ratios were adjusted for age, race, sex, group of origin, education and other Axis I disorders. Only two associations were significant: schizophrenia diagnoses had a decreased risk of having an associated diagnosed personality disorder diagnosis, with an odds ratio of 0.18 ($P<0.001$), while the combined drug or alcohol abuse category had a significantly increased chance of having an additional personality disorder diagnosis with an odds ratio of 2.06 ($P<0.05$). Table I gives the unadjusted percentages of total personality disorders and personality disorder types that occurred in each of the three study groups. Table II shows the odds ratios for the various pairwise comparisons of the three study groups for total personality disorders and the personality disorder types. These odds ratios (ORs) were adjusted for age, race, sex and education. Total personality disorders discriminated successfully the disability from the forensic group (OR = 3.19, $P<0.05$) and the pain from the forensic group (OR = 4.25, $P<0.001$). The asocial personality type distinguished successfully the disability from the forensic group (OR = 6.58, $P<0.05$) and the flamboyant personality type distinguished successfully the pain from the forensic group (OR = 2.86, $P<0.05$). Finally, the anxious personality type distinguished successfully both the pain from the forensic group (OR = 12.2, $P<0.001$) and the disability from the pain group (OR = 0.13, $P<0.05$).

TABLE I

Percentages of personality disorders and personality disorder subtypes by group (n = 488)

Diagnostic variable	*Forensic group n = 390*	*Pain group n = 43*	*Disability group n = 55*
Total personality disorders	11.8	37.2	22.2
Personality disorder subgroup A (asocial)	0.8	2.3	5.5
Personality disorder subgroup B (flamboyant)	8.0	20.9	10.9
Personality disorder subgroup C (anxious)	3.1	14.0	5.5

TABLE II

Odds ratios for group comparisons by total number of personality disorders and personality subtypes (odds ratios adjusted for age, race, sex and education) (n = 488)

Diagnostic variable	*Disability vs forensic*	*Pain vs forensic*	*Disability vs pain*
Total personality disorders	3.2*	4.3**	0.5
Personality subgroup A (asocial)	6.6*	1.2	2.6
Personality subgroup B (flamboyant)	2.5	2.9*	0.7
Personality subgroup C (anxious)	2.2	12.2**	0.1*

*$P<0.05$; **$P<0.001$.

Discussion

The finding of a significant association between drug or alcohol abuse and the presence of a concurrent personality disorder is consistent with clinical observation and other recent findings (Koenigsberg *et al*, 1985). Several research workers, using different techniques, have noted either an excess of personality disorders or specific personality traits in alcohol or drug abusers (Henn *et al*, 1977; Craig, 1979*a*,*b*). This finding supports clinical judgement that detoxifying a drug abuser often reveals a second diagnosis of personality disorder. The questions as to which personality disorders are more likely to be present in drug abusers and whether there is a difference in the personality disorders that are related to drug and alcohol abuse constitute important areas of future research.

The low frequency of personality disorders among schizophrenics has several possible interpretations. There may be simply less personality disorders to diagnose, but it is more likely that the diagnosis of schizophrenia subsumes many symptoms which might otherwise be used to make a diagnosis of personality disorder. The DSM-III in these cases often excludes such a diagnosis. This explanation would also be consistent with the hypothesised close relationship between schizophrenia and certain personality disorders (Kendler & Gruenberg, 1982; Baron *et al*, 1983; Lowing *et al*, 1983; Kendler *et al*, 1984).

The finding that total personality disorders can distinguish between the forensic and the pain and disability groups (the former having less personality disorders) is interesting in two respects. It demonstrates that the frequency of personality disorders differs across patient subgroups as well as across Axis I categories, and how differences across Axis I diagnoses can interact with personality disorder differences across groups. For example, since schizophrenics are diagnosed as having personality disorders less often, and since the forensic group contains a relative excess of schizophrenics, one would expect group differences among the forensic group and the other groups on the frequency of personality disorders. Both the research question as to why these groups differ and the clinical question as to how this difference would affect treatment approaches require further work.

To some extent, the significant findings between the personality subgroups are consistent with what we know about these patient populations. As those in the disability group are to some extent looking for a way to opt out of the competitive demands of daily life, we would expect to see more asocial personality types in this group than in the forensic group, whose members are interactive enough to get arrested. For the same reason, it is not unexpected that there are less anxious personality types in the disability than in the pain group. Those in the disability group are withdrawing from contact, in contrast with the pain group who still seek it, but it is not immediately clear why the latter should have an excess of flamboyant and anxious personality types, relative to the forensic group.

What is of some interest is that the personality subtypes were able to distinguish all three patient populations, even when adjusted for age, sex, race and education. This gives evidence for the validity of the grouping system.

There is evidence to support the DSM-III personality disorder types. In the asocial type, Kendler *et al* (1984) used a family history method and were able to show a relationship between a family history of schizophrenia and the schizophrenic spectrum (schizoid and schizotypal) personality disorders. This was replicated by Lowing *et al* (1983) using DSM-III criteria, while Kendler *et al* (1982), using similar methods were also able to demonstrate a relationship between paranoid personality disorder and schizophrenic spectrum disorder. Baron *et al* (1983) further demonstrated a possible genetic-familial link between schizotypal personality disorder and a family history of schizophrenia.

There is also evidence supporting the creation of a flamboyant type. Akiskal *et al* (1977) reported that some patients with a diagnosis of cyclothymic disorder are often diagnosed as personality disorders of the flamboyant type, while McNamara *et al* (1984) and Akiskal *et al* (1985) have documented similarities between REM latencies in the borderline group and in those with affective disorders. It appears that a subgroup of antisocial personalities are related to the affective disorders (Reich, 1985*a*), while the anxious personality disorders may well be related to Akiskal's (1983) concept of subaffective dysthymic group, in addition to having apparent validity and clinical overlap.

Work by Tyrer (Tyrer & Alexander, 1979; Tyrer *et al*, 1979, 1983), who approached the measurement of personality from a different perspective (measuring personality traits and then factor analysing for categories of personality disorders), yields categories which are not inconsistent with the DSM-III groupings. Their sociopathic type has similarities to the DSM-III flamboyant type, their schizoid type to the asocial type and their passive-dependence to the anxious type, while their final grouping of anakastic has similarities to the DSM-III compulsive personality disorder. These similarities also lend evidence to the validity of the DSM-III groupings of the personality disorder.

In summary, there is evidence that the frequency of occurrence of DSM-III personality disorders may differ among the groups meeting the criteria for various Axis I categories. There is also evidence supporting the validity of the DSM-III method of subtyping the personality disorders. The clinical implications of these findings are largely in the necessity of treating symptoms of personality disorders in many drug and alcohol abusers. Further research investigation into DSM-III personality clusters appears warranted.

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References

Akiskal, H. S. (1983) Dysthymic disorder: psychopathology of proposed chronic depressive subtypes. *American Journal of Psychiatry*, 140, 11–20.

Akiskal, H. S., Djenderdjuan, A. & Rosenthal, R. (1977) Cyclothymic disorder, validating criteria for inclusion in the bipolar affective group. *American Journal of Psychiatry*, **134**, 1227–1233.

——, Yervanian, B. I., Davis, G. C., *et al* (1985) The nosologic status of borderline personality: clinical and polysomnographic. *American Journal of Psychiatry*, **142**, 192–198.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual for Mental Disorders* 3rd ed. (DSM-III). Washington, DC: APA.

Baron, M., Gruen, R., Asnis, L. *et al* (1983) Familial relatedness of schizophrenia and schizotypal states. *American Journal of Psychiatry*, **140**, 1437–1442.

Bluestone, H. & Mellela, J. (1978) A study of criminal defendants referred for competency to stand trial in New York City. *Bulletin of the American Academy of Psychiatry and Law*, **7**, 166–178.

—— & Baskin, D. (1981) A study of criminal defendants referred for multiple psychiatric examinations regarding their competency to stand trial. *Journal of Forensic Science*, **26**, 416–423.

Cooke, G. (1968) The court study unit: patient characteristics and differences between patients judged competent and incompetent. *Journal of Clinical Psychology*, **25**, 140–143.

Craig, R. J. (1979*a*) Personality characteristics of heroin addicts: A review of the empirical literature with critique—Part I. *International Journal of Addictions*, **14**, 513–531.

—— (1979*b*) Personality characteristics of heroin addicts: A review of the empirical literature with critique—Part II. *International Journal of Addictions*, **14**, 607–626.

Devine, R. & Merskey, H. (1965) The description of pain in psychiatric and general medical patients. *Journal of Psychosomatic Research*, **9**, 311–316.

Frances, A., Clarkin, J. F., Gilmore, M., *et al* (1984) Reliability of criteria for borderline personality disorder: A comparison of DSM III and the diagnostic interview for borderline patients. *American Journal of Psychiatry*, **141**, 1080–1083.

Henn, F. A., Herjanic, M. & Vanderpearl, R. H. (1977) Forensic psychiatry: anatomy of a service. *Comprehensive Psychiatry*, **18**, 337–345.

Kendler, K. S. & Gruenberg, A. M. (1982) Genetic relationship between paranoid personality disorder and the 'schizophrenic spectrum' disorders. *American Journal of Psychiatry*, **139**, 1185–1186.

——, Masterson, C. C., Ungaro, R., *et al* (1984) A family history study of schizophrenia-related personality disorders. *American Journal of Psychiatry*, **141**, 424–427.

Koenigsberg, H. W., Kaplan, R. D., Gilmore, M. M., *et al* (1985) The relationship between syndrome and personality disorder in DSM III. *American Journal of Psychiatry*, **142**, 207–212.

Lowing, P. A., Mirsky, A. F. & Pereira, M. A. (1983) The inheritance of schizophrenia spectrum disorders: A reanalysis of the Danish adoptee study data. *American Journal of Psychiatry*, **140**, 1167–1171.

McNamara, E., Reynolds, C. F., Soloff, P. H., *et al* (1984) EEG sleep evaluation of depression in borderline patients. *American Journal of Psychiatry*, **141**, 182–186.

Merskey, H. (1965) The characteristics of persistent pain in psychological illness. *Journal of Psychosomatic Research*, **9**, 291–298.

Reich, J. (1985*a*) The relationship between antisocial behavior and affective illness. *Comprehensive Psychiatry*, **26**, 296–303.

—— (1985*b*) DSM III diagnosis in SSI applicants referred for psychiatric evaluation. *Journal of Clinical Psychiatry*, **47**, 81–82.

—— & Wells, J. (1985) Psychiatric diagnosis and competency to stand trial. *Comprehensive Psychiatry*, **26**, 421–432.

——, Tupin, J. P. & Abramowitz, S. I. (1983) Psychiatric diagnosis of chronic pain patients. *American Journal of Psychiatry*, **140**, 1495–1498.

SAS Institute (1980) SAS Supplement Library Users Guide. SAS Institute, Inc., Box 8000, Cary, North Carolina 27511.

Sikorski, C. R. & Benedek, E. P. (1977) Competency to stand trial—the female offender. *Journal of the American Medical Women's Association*, **32**, 150–154.

Tyrer, P. & Alexander, J. (1979) Classification of personality disorder. *British Journal of Psychiatry*, **135**, 163–167.

——, Cicchetti, D., *et al* (1979) Reliability of a schedule for rating personality disorders. *British Journal of Psychiatry*, **135**, 168–174.

——, Strauss, J. & Cicchetti, D. (1983) Temporal reliability of personality in psychiatric patients. *Psychological Medicine*, **13**, 393–398.

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Improving the Quality of Care on Psychiatric Hospital Rehabilitation Wards
A Controlled Evaluation

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This study attempted to use feedback from the Model Standards Questionnaires (a series designed to measure the quality of care) to improve care on a psychiatric hospital's rehabilitation wards. Both a controlled and quasi-experimental design were used. The controlled design provided evidence that feedback from the Questionnaire concerned with the Individual Programmes of Treatment and Care led to improvements in these practices but not for the remaining three questionnaires. The quasi-experimental design indicated that the significant generalised improvement in care, over all questionnaires, was most likely due to the feedback.

In the field of psychiatric rehabilitation much has been written about the need to develop methods of ensuring a high quality of care for clients in need of long-term care (DHSS, 1979; Shepherd, 1984). However, as Lavender (1985) points out there is no published scale which evaluates the broad range of conditions and practices that are important in the rehabilitation process (Watts & Bennett, 1983).

A number of studies have measured some of these conditions and practices. Morris (1969) developed measures of the physical environment and community contact, King *et al* (1972) of the physical environment and management practices and Wing & Brown (1970) of a number of aspects of the social environment (i.e. level of restrictiveness, number of personal possessions and amount of time doing nothing). More recently, Wykes *et al* (1982) devised measures of the management practices (Hostel Hospital Practices Profile) and level of community involvement and Garety & Morris (1984) [developing Richardson's (1977) work] of hostel management practices. The difficulty with these scales is that they only assess selected areas of practice and/or were devised for use in non-psychiatric settings, such as institutions for the mentally handicapped.

Given these difficulties, it proved necessary to devise a more comprehensive scale. Lavender (1985) describes the development of such a measure, the Model Standards Questionnaires. These questionnaires measured five aspects of the environment – (1) Staff Resources, (2) Physical Resources, (3) Management Practices, (4) Contact with the Community Practices, and (5) Treatment Practices. The present study was an attempt to use the information gathered from these questionnaires to improve the quality of care on the rehabilitation wards of a psychiatric hospital.

It is evident there are different ways of providing feedback about the performance of ward staff. Herold & Greller (1977) distinguish between five forms of feedback – (1) Negative, (2) Positive, (3) Negative from Hierarchy, (4) Positive from hierarchy, (5) From self. Andrask *et al* (1981) reviewed the behavioural methods that have been used to improve work performance and suggest that although instructions appear to be an ineffective method of feedback, if the instructions are clear and specific their effectiveness is increased (Iwata *et al*, 1977; Burg *et al*, 1979).

Further, a number of studies have shown social praise to be an effective method of improving staff level of interaction with patients (Montegar *et al*, 1977). Additionally, the work of Prue *et al* (1980) suggests that the effectiveness of feedback, whether from an individual or a ward can be enhanced if it is made public. Thus for this study a number of methods of providing feedback were used including social praise for high quality practices, specific instructions about how to improve low quality practice and finally a combination of private (to the ward team) and public (to the ward's senior nurses and rehabilitation committee) feedback.

Much of the literature concerned with interventions within institutions has involved descriptions about how and why such interventions succeed or fail (Georgiades & Phillimore, 1975; Towell & Dartington, 1976). This work, although somewhat anecdotal, is useful in providing practical advice to the researcher/change agent. Georgiades

& Phillimore (1975) record some of the ways institutions resist change and note how hospitals "like dragons eat hero innovators for breakfast". Similarly, Towell & Dartington (1976) point out that the publication and availability of guidance provided by Government Reports and research literature "should be a valuable stimulus for change but past experience suggests that such documents by themselves are likely to have only a diffuse and limited impact in many settings where improvement might be desirable". Given the inherent difficulties it was apparent that the manner in which the intervention was undertaken required careful consideration.

Thus from this anecdotal body of knowledge, a number of practical pieces of advice were drawn. Firstly, in order to maximise the impact of the intervention it was necessary to gain influence on the basis of 'expertise and ability' rather than formal power. Secondly, in order for the change agents to manage the institution's resistance and maintain high morale, it was important to establish a cohesive 'change agent team'. Thirdly, to maximise the impact of the intervention it was necessary to utilise the limited channels of authority to which the Rehabilitation Team (change agents) had access, an approach described as 'top down' by Towell & Dartington (1976). Finally, in order to harness the commitment of ward staff (essential if ward practices were to be changed), it was important to involve them actively in each stage of the project - an approach described by Towell & Dartington (1976) as 'bottom up'.

This paper describes an evaluation of an intervention designed to improve the quality of care in a group of 12 psychiatric hospital rehabilitation wards. This intervention was based on the Model Standards Questionnaires (Lavender, 1985) and consisted of specific recommendations (feedback) to the ward team about how the quality of care provided could be improved. The intervention was carried out by members of the hospital's Rehabilitation Committee following a request from management to investigate the quality of care provided on the hospital's rehabilitation wards.

The evaluation used two methodologies - the first, a controlled design to investigate whether the wards changed on questionnaires about which they received feedback but not on the other questionnaires. Thus in this part of the evaluation half the wards received feedback and half the wards did not receive feedback about each questionnaire. The second was a quasi experimental design (Cook & Campbell, 1979) to investigate whether the feedback lead to a more generalised change in ward practice. This was achieved by (a) comparing pre- and post-feedback scores on all wards, and (b) investigating whether other important ward variables (e.g. staff/patient ratios) changed in a way likely to account for any generalised changes in practice. If not it is a reasonable supposition that changes in practices can be attributed to the provision of feedback.

Method

Setting

The settings for both studies were 12 rehabilitation (medium- to long-stay) wards in a large psychiatric hospital (900 beds). For each ward the number of patients ranged between 21 and 30, the percentage of patients with a length of stay of 1 year or more ranged between 10 and 100 and the average nurse/patient ratio ranged from 1:5.6 to 1:10.8.

Measures and procedures

For the purposes of this study three of the five Model Standards Questionnaires were used. The data concerned with the reliability and validity of these questionnaires have been reported elsewhere (Lavender, 1985) but indicated that the measures were both reliable and valid. The three questionnaires used in this study are described in more detail below.

Individual programmes of treatment and care

This questionnaire consisted of 10 items assessing the extent to which patients on the ward/unit were engaged in particular therapeutic/training activities and the frequency with which these programmes were reviewed. The therapeutic/training activities included those designed to improve patients personal hygiene, domestic, social, occupational and leisure skills. Scoring involved a 1-10 rating based on the percentage of patients engaged in each area of training. An example of one of the items is given below.

3.(i) How many patients can do all the following: (a) cook two different main meals, (b) use a launderette, (c) use a Hoover or carpet sweeper, (d) buy at least three items from a shop and know how much change they should have.

Number ....

3.(ii) Could you please list the training activities that exist on and off the ward for teaching home management skills (cooking, cleaning, washing, laundry, shopping, budgeting) and the names of patients who have been actively engaged in such training in the last four weeks.

Ward management practices

This questionnaire included 25 items and assessed the extent to which management practices allowed patients to act autonomously, had their own personal possessions and minimised the social distance between patients and staff. Two items from the Questionnaire are given below to illustrate the format used.

6.(i) How many patients are allowed to operate the T.V. controls?

Number ......

24. How many patients have leisure possessions of their own? For example, books, records, musical instruments, radios, games etc.

Men *Women*

Number ...... Number ......

Scoring involved a 1.5 rating based on a unique set of criteria for each item but with clear markers at each point. For example, from item 9 (Visiting Hours) the average daily visiting hours is computed and scored using the following rating criterion: 1. 1-3 hours per day; 2. 4-6 hours; 3. 7-9 hours; 4. 10-12 hours; 5. 13 hours or more.

Community contact practices

This questionnaire included 13 items and assessed the extent to which patients had contacted with community-based facilities in the previous 4 weeks. No attempt was made to assess the extent of contact with family or friends from the community as this would have been less a measure of the quality of care provided and more a measure of a residents' community support. Scoring involved a 1-10 rating based on the percentage of patients engaged in each particular type of community contact.

Interviewer training procedure

The interviewer training procedure included the following steps: (1) reading through the questionnaires; (2) reading through the Scoring Manual; (3) reading through the Training Manual (the Training Manual was developed to clarify the items and provide advice to the interviewer for dealing with difficult or ambiguous responses), and (4) a discussion of each item with a trained interviewer before and after completing a training interview.

Interview procedure

The interview involved two interviewers attending the multidisciplinary meeting where the questionnaires were administered. The interviewers were two members of the Rehabilitation Committee which included a psychiatrist, psychologist, occupational therapist, nursing officer, sister and social worker. The interviewers included members of the ward's multiprofessional team which minimally included a consultant psychiatrist, social worker, two charge nurses/sisters, a staff nurse, student nurse and psychologist (if attached to the ward team). The interview was conducted by only one of the interviewers whilst the other remained silent and merely completed the questionnaire. The main interviewer took care to explain the purpose of the Questionnaires and the reason for the second silent interviewer (i.e. "to check that both interviewers recorded the same and therefore reliable information"). When completing the Physical Environment Questionnaire the main interviewer carried out the assessment by direct observation and measurement with the assistance and/or accompaniment of a nurse. The reliability interviewer recorded observations independently. Following the administration of the questionnaires, the interviewers rated the questionnaires independently using the Scoring Manual. The data presented in this paper are from the main interviewer.

Design

The 12 wards included all the medium- to long-stay rehabilitation wards in the hospital, thus they were not a group of specially selected rehabilitation wards. The basic design of the study was that following the first administration of the questionnaires the individual wards were given feedback about their current performance. For the purposes of the design the Ward Management Practices Questionnaire was divided into two - Part I (11 items) and Part II (11 items). Feedback was not given about three items in this scale because it was considered beyond the power of the ward team to immediately introduce the appropriate change.

The feedback consisted of a series of specific recommendations about whether the ward should continue or change the specific practices referred to by the individual items in the questionnaire. The feedback was given in two forms, firstly, in an interview between selected members of the Rehabilitation Committee and the ward's multidisciplinary team where the recommendations were discussed in detail. Secondly, in the form of a typed Model Standards Feedback Report which will be described in detail later. Nine months after the receipt of this first Feedback Report the Questionnaires were administered for a second time. The questionnaires were scored and the pre- and post-intervention scores compared.

The evaluation used two methodologies:

(a) *A controlled design* in which control was achieved by arranging that half the wards received feedback and half the wards did not receive feedback about each questionnaire. Thus each ward received feedback about two questionnaires and had feedback withheld for two questionnaires. The decision about which wards received feedback about which questionnaires was made using a random number assignment procedure.

(b) *A quasi experimental design* to investigate whether the feedback led to a generalised change in ward practice. This was achieved by (i) comparing pre- and post-intervention scores and (ii) measuring other important ward variables to investigate whether they could account for any generalised changes in practice. The other ward variables included nurse/patient ratio, involvement of other professional staff, number of patients on each ward, percentage of patients over 60 and the physical environment.

Results

Following a detailed analysis of the properties of the data (normality of distribution, equality of variance and level of measurement) it was considered appropriate to use parametric statistics. The results from the controlled and quasi-experimental design are presented separately.

Controlled design

Two methods of analysis were used, firstly, analysis of variance of gain scores and secondly, t-tests. The analysis of variance of gain scores assessed the significance of the difference or change in performance from pre- to post-intervention. These results proved insignificant for all questionnaires indicating that there was not a significantly greater improvement in the experimental (i.e. wards receiving feedback) than the control group. This method of analysis clearly provides the most conservative statistical estimate of the intervention effect.

The second form of statistical analysis was used to test whether there was a significant change when pre- and post-intervention scores were compared in the experimental and control group separately. This analysis (Table I) revealed that in the experimental group there was a significant increase in scores on the Individual Programmes of Treatment and Care Questionnaire ($t = 3.76$, $P < 0.01$) but not on the remaining questionnaires.

TABLE I

Experimental group analysis of pre- vs post-intervention data

Questionnaire	*t-Value*	*Probability*	*Significance*
1	3.706	<0.01	Significant
2	1.691	>0.05	Not significant
3	1.871	>0.05	Not significant
4	1.476	>0.05	Not significant

Quasi experimental design

In order to test whether there had been a generalised change in practice following the feedback intervention the pre- and post-intervention scores for the whole group were compared. These analyses confirmed that for all questionnaires there was a significant increase between pre- and post-intervention (Table II).

TABLE II

Experimental and control groups combined data comparison of pre- and post-intervention scores

Questionnaire	*t-Value*	*Probability*	*Significance*
1	2.296	<0.05	Significant
2	2.094	<0.05	Significant
3	2.551	<0.05	Significant
4	2.070	<0.05	Significant

The second part of the design involved an attempt to estimate whether this generalised change was due to changes in other important ward variables. Data were collected and analysed about five variables:

(i) *Nurse/patient ratios* were calculated from the data collected on the Policies, Planning, Monitoring of Progress and Staffing Levels Questionnaire (PPMSQ). In all cases although data were gathered only for the week prior to the interview, the nursing figures given were said to be the same as usual. These data were used to test whether there was a change in the nurse/patient ratio. The results of the analysis are presented in Table III and indicate that no significant change occurred and therefore it appears unlikely that this variable caused the generalised change in the questionnaire scores.

TABLE III

Statistical evaluation of changes in ward demographic variables between pre- and post-interventions

Variable	*Related t-Value*	*d.f.*	*Significance*
Nurse/patient ratio	0.8693	11	NS
Other professional staff	0.4528	11	NS
Patient numbers	0.3652	11	NS
Percentage patients over 60	0.3405	11	NS

(ii) *Other professional staff involvement.* Data concerning the time spent by other professional staff (doctors, psychologists, social workers, occupational therapists, etc.) on the ward or directly involved in work connected with the ward were collected on the Policies, Planning, Management and Staffing Levels Questionnaire (see Lavender, 1985). These data were used to test whether there was any change in the level of involvement of other professional staff between pre- and post-intervention. However, once again the statistical analysis presented in Table III revealed no significant change in the amount of professional staff involvement between pre- and post-intervention. This mitigates against this variable being an important cause of the observed changes in practice.

(iii) *Number of patients* on each ward was recorded on the Ward Information Sheet. Comparison of the number of patients on each ward between pre- and post-intervention was undertaken and as can be seen from Table III no significant changes were recorded.

(iv) *Percentage of patients over 60* was calculated from the data collected on the Ward Information Sheet. Again as Table III indicates there was no significant change in the percentage of patients over 60 between pre- and post-intervention.

(v) *The physical environment.* The fifth Model Standards Questionnaire measured the quality of the physical environment. Comparative analysis of this data revealed no significant difference between pre- and post-intervention. Inspection of the data

TABLE IV

Physical environment questionnaire pre- and post-intervention scores

	Ward											
Intervention	1	2	3	4	5	6	7	8	9	10	11	12
Pre	81	86	86	83	78	87	79	83	77	82	87	84
Post	81	86	86	83	78	87	79	84	77	82	82	84

presented in Table IV will reveal that there were very few changes of any kind in the wards' physical environments.

Discussion

Given the limited number of wards available for study, the controlled design provided the most stringent test of whether feedback affected ward practice. The results provided some statistical evidence that feedback about treatment and care practices (Individual Programmes of Treatment and Care Questionnaire) led to improvements in those practices. However, although there was a similar tendency to improve in wards receiving feedback about ward management and community contact practices this was not significant. The hypothesis that feedback about these latter two categories of practice led to wards changing those specific practices was not supported.

The study's quasi-experimental design was used to investigate whether the feedback had led to a generalised improvement in practices. The first part of this analysis revealed that there had been a significant improvement in practices on all four Questionnaires (Table II). The second part of the analysis attempted to establish whether this improvement had coincided with changes in other important ward variables which equally could have led to change in practice. A number of ward variables including nurse patient ratios, level of other professional staff involvement, number of patients on the wards, percentage of patients over 60 and the physical environment, all proved not to have changed significantly between pre- and post-feedback assessments (Table III). Clearly these variables could not account for the observed improvements in practice.

There is, however, a possibility that the changes were due to maturation (Cook & Campbell, 1979). Maturation refers to the fact that the practices could have been developing/maturing for some time and the results merely reflected these ongoing developments. Whilst this explanation is possible it is clearly unlikely for two main reasons. Firstly, large psychiatric institutions are not noted for their changes in practice and indeed many organisational writers have described with considerable emotion the difficulties and resistance to change in such establishments (Fairweather *et al*, 1974; Georgiades & Phillimore, 1975; Mealiea, 1978; Broome, 1979). Secondly, as with the last point, large psychiatric institutions are not noted for their speed of change and the recorded changes occurred within a relatively short period. Given these two points it is considered unlikely that a straightforward maturation explanation is sufficient to account for the changes.

Clearly a number of variables that might have caused the changes in practice were not recorded. It is indeed possible that the reasons for change varied between wards, so that in some cases it was due to increases in nurse/patient ratios and in others an increase in the percentage of patients over 60. Included in the unrecorded variables would be changes in the quality of staff (although there were very few new staff acquired by the wards during the 9 months of the study) and changes brought in by old staff, unrelated to feedback (for example, if staff introduce a change in practice as a result of a nurse teaching lecture).

Obviously, the simple pre- and post-intervention design makes it impossible to come to firm conclusions because of the important unrecorded and unconsidered variables. However, from the attempts made to record important variables and to estimate their significance it would seem unlikely that they would have led to such generalised changes in practice as were recorded between pre- and post-assessments.

One major issue raised by the study concerns the general applicability of the Model Standards Questionnaires and feedback as a means of improving the quality of care. Within psychiatric hospitals there has been a tendency when attempting to develop rehabilitation services to set up a special unit or develop a particular part of the rehabilitation service (Fairweather *et al* 1974; Paul & Lentz, 1977; Cope & Cox, 1980). This has resulted in the setting up of particular units such as the 'token economy' (Hall *et al*, 1977), the Lodge group (Fairweather *et al*, 1974), and even "the therapeutic community", (Jones, 1952). The problem with setting up such units in terms of the rest of the hospital (i.e. the whole system) is that many wards become neglected and receive little input. A secondary effect is that staff in the remaining wards become resentful about the 'new units' that receive all the 'good things' and the resentments result in the isolation of these 'new units'. Such special rehabilitation units seldom seem to lead to a generalised improvement in the hospital's rehabilitation service as a whole.

The type of intervention described in this study attempted to avoid these difficulties by designing an intervention likely to improve the practices on the 'back wards' in addition to the units already functioning well. When looking at the hospital's ward-based rehabilitation service as a whole the failure to attempt to change the wards receiving little input (back wards) simply represents an avoidance of the most difficult areas rather than attempting to overcome the difficulties. It should be pointed out that this runs counter to the suggestions of Georgiades

& Phillimore (1975) that the line of least resistance should be followed when attempting organisational change. They argue that only the parts of the system that are keen and ready for change should be worked with. In a hospital this suggestion can lead to unfortunate misunderstandings such that certain wards become designated 'bad' without attempting to investigate why this is so and without identifying and encouraging the development of the healthy parts of that ward system. For example, within the present study a particular ward was noted hospital-wide for providing a custodial form of care and yet in response to the recommendations to improve its level of community contact, it made quite marked changes in this area.

It is not being argued here either that the development of special units within large hospitals is better than attempting to improve a whole section of the hospital service or vice versa. The point is that these different types of intervention will have differential effects. The present work is one of the few systematic research projects to have attempted to evaluate an intervention designed to have a generalised effect on a hospital's service.

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References

ANDRASK, F., HEIMBERG, J. S. & MCNAMARA, J. R. (1981) Behaviour modification of work and work related problems. In *Progress in Behaviour Modification* (Vol. 2) (ed. M. Hersen & P. M. Miller) New York: Academic Press.

BROOME, A. K. (1979) Setting targets for institutional care. Paper given at *1979 Annual Conference of the British Psychological Society*, Nottingham University.

BURG, M. M. & REID, D. H. & LATTIMORE, J. (1979) Use of self recording and supervision programme to change institutional staff behaviour. *Journal of Applied Behavioural Analysis*, 12, 363–375.

COOK, T. D. & CAMPBELL, D. T. (1979) *Quasi Experimentation Design and Analysis Issues for Field Settings*. Boston: Houghton-Mifflen.

COPE, D. & COX, S. (1980) Organisational development in a psychiatric hospital, creating desirable changes. *Journal of Advanced Nursing*, 5, 371–380.

DEPARTMENT OF HEALTH & SOCIAL SECURITY (1979) *Organisation and Management Problems of Mental Illness Hospitals: Report of a Working Group*. London: DHSS.

FAIRWEATHER, G. W., SANDERS, D. H. & TORNATSKY, L. G. (1974) *Creating Change in Mental Health Organisations*. New York: Pergammon Press.

GARETY, P. A. & MORRIS, I. (1984) A new unit for long stay psychiatric patients: organisation attitudes and quality of care. *Psychological Medicine*, 14, 183–192.

GEORGIADES, N. J. & PHILLIMORE, L. (1975) The myth of the hero innovator and alternative strategies for organisational change. In *Behaviour Modification with the Severely Retarded* (eds C. C. Keirnan & F. D. Woodford). Amsterdam: Associated Scientific Publishers.

HALL, J. N., BAKER, R. D. & HUTCHINSON, K. (1977) A controlled evaluation of token economy procedures with chronic schizophrenic patients. *Behaviour Research and Therapy*, 15, 261–283.

HEROLD, D. M. & GRELLER, M. (1977) Feedback: definition of a construct. *Academy of Management Journal*, 20, 147–157.

IWATA, B. A., BAILEY, J. S., BROWN, K. M., FOSHEE, T. J. & ALPERN, M. A. (1977) A performance based lottery to improve residential care and training by institutional staff. *Journal of Applied Behaviour Analysis*, 9, 417–431.

JONES, M. (1952) *Social Psychiatry: A Study of Therapeutic Communities*. London: Tavistock.

KING, R. D., RAYNES, N. V. & TIZARD, J. (1972) *Patterns of Residential Care*. London: Routledge & Kegan Paul.

LAVENDER, A. (1985) *Evaluation and Change in Settings for the Long Term Psychologically Handicapped*. Ph.D Thesis, University of London, Kings College Hospital Medical School.

MEALIA, L. N. (1978). Learned Behaviour: the key to understanding and preventing employee resistance to change. *Group and Organisational Studies*. 3, 211–223.

MONTEGAR, C. A., REID, D. H., MADSEN, C. H., JR. & EWELL, M. D. (1977). Increasing institutional staff to resident interactions through inservice training and supervisor approval. *Behaviour Therapy*, 8, 533–540.

MORRIS, P. (1969) *Put Away: Sociological Study of Institutions for the Mentally Retarded*. London: Routledge & Kegan Paul.

PAUL, G. L. & LENTZ, R. (1977) *Psychosocial Treatment of Chronic Mental Patients*. Cambridge, Mass: Harvard University Press.

PRUE, D. M., KRAPFL, J. E., NOAH, J. C., CANON, S. & MALEY, R. F. (1980) Managing the treatment activities of state mental hospital staff. *Journal of Organisational Behavioural Management*, 2, 165–181.

RICHARDSON, A. (1977) *Organisation and Interaction in Psychiatric Day Centres*. MPhil. Thesis, University of London.

SHEPHERD, G. W. (1984) *Institutional Care and Rehabilitation*. London: Longman.

TOWELL, D. & DARTINGTON, T. (1976). Encouraging innovations in hospital care. *Journal of Advanced Nursing*, 1, 391–398.

WATTS, F. N. & BENNETT, D. H. (1983). Introduction: the concept of rehabilitation. In F. N. Watts & D. H. Bennett. *Handbook of Psychiatric Rehabilitation Practice*. Chichester: Wiley.

WING, J. K. & BROWN, G. W. (1970) *Institutionalism and Schizophrenia*. London: Cambridge University Press.

WYKES, T., STURT, E. & CREER, C. (1982) Practices of day and residential units in relation to the social behaviour of residents. In *Long Term Community Care Experience in a London Borough* (ed J. K. Wing) *Psychological Medicine*, Monograph Supplement 2, London: Cambridge University Press.

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Depression, Dementia and Disability in the Elderly

R. A. GRIFFITHS, W. R. GOOD, N. P. WATSON, H. F. O'DONNELL, P. J. FELL and J. M. SHAKESPEARE

A structured sample of mobile elderly patients in a rural community practice was assessed on validated rating scales for depression, dementia and disability. A total of 62% of the sample was abnormal on at least one variable. The overall prevalence of depression was 13%; the overall prevalence of dementia was either 10 or 18% depending on the criterion of Mental Test Score (MTS). Depression and dementia were related, depression being more common in females. In depressed and demented patients, MTS was age-related in those over 60 years; in depression alone, MTS was not age-related. Dementia was age-related, particularly over the age of 75. Disability increased with age and was more common in females. Disability was associated with both depression and dementia.

Depression and dementia affect approximately 10–15% and 5–10% of the elderly population respectively, and both increase with age. Differences in diagnostic criteria for dementia and depression produce the greatest variation in reported prevalence rates (Mortimer *et al*, 1981; Blazer, 1982). Depression is more common in elderly females (Freedman *et al*, 1982); dementia is more common in elderly females than in elderly males, particularly in those aged 75 and over (Gurland *et al*, 1980; Kay & Bergmann, 1980). Chronic physical illness, and impaired vision and hearing loss, have been implicated in the aetiology of affective disorders in elderly patients (Roth & Kay, 1956; Herbst & Humphrey, 1980). Patients with senile psychosis (senile dementia) have less physical illness than have patients with affective disorders, but patients with arteriosclerotic psychosis (arteriosclerotic dementia) have more (Kay & Roth, 1955). Disability and dependence increase with age, major factors being dementia and functional psychiatric disorders, particularly depression (Akhtar *et al*, 1973).

There is considerable difficulty in obtaining a random sample of elderly patients in the community, non-response to letters being a particular problem. Up to one-third of initial contacts may not respond or may be excluded (Williamson *et al*, 1964; Milne *et al*, 1971; Herbst & Humphrey, 1980; Gurland *et al*, 1983).

Rating scales for depression and dementia have limitations in diagnosis; abbreviated scales may have more validity for measuring severity and extended scales for measuring response to treatment (Hodkinson, 1972; Bech *et al*, 1975).

We assessed depression, dementia and disability by validated rating scales to determine their association and to identify patients at risk in the community.

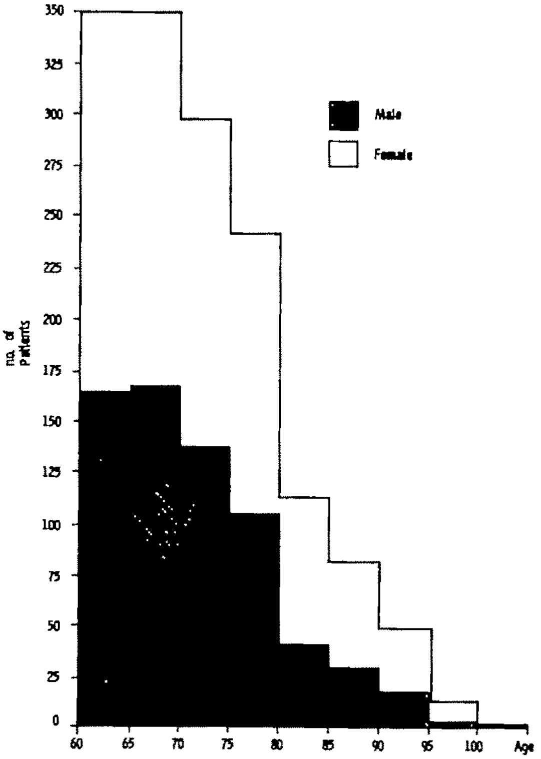

FIG. 1 Age distribution in quinquennia of 1498 patients aged 60 years and over enrolled at Deddington Health Centre on 1 October 1980.

Subjects

The study was undertaken in 1979, in a rural group practice in Oxfordshire, which had 1500 patients aged 60 years and over. The age distribution of these patients, in quinquennia, is shown (Fig. 1). The practice population was fairly stable, with a turnover of 8% per annum. The study was made on 200 patients to provide 20 men and 20 women in each 5-year group from 60 to 79 years and a similar group aged 80 and over. All patients aged 60 and over attending the health centre for any reason were referred by the principals or research nurse; elderly relatives and friends accompanying these patients were also asked to participate. To complete the quinquennia, patients were identified randomly from the practice list and contacted by letter or telephone. The patients included were those who were willing and able to attend the health centre; apart from minor current medical problems, they were not ill. These patients were a 'good' group, in that they were able to travel by car or practice bus or walk from the village. All surviving patients were called for review in 1980, by telephone or letter.

Assessment

Subjects willing to participate received an appointment for assessment at the health centre. All subjects were assessed at single interview by one observer on three standard rating instruments, for depression, dementia and disability. No subject failed to attend, and all were rated on all three instruments.

Depression score

Hamilton (1960) designed a Depression Rating Scale (HDRS) for the assessment of patients clinically diagnosed as depressed, and considered its greatest value to be in assessing the response to treatment. But scoring helps to differentiate endogenous and neurotic depression (Carney *et al*, 1965). Rating scales for depression are widely used (Hamilton, 1960, 1976; Beck *et al*, 1961; Zung, 1965; Beck, 1967; Weissman *et al*, 1977; Blazer, 1980, 1982). The prevalence of reported depression differs from study to study, which may be attributable to the different criteria of various rating scales (Blazer, 1982).

Comparisons of scales indicate that the long-established HDRS is most appropriate in community studies in the elderly (Kearns *et al*, 1982). Interview length may be a determinant of maintained interest (Hamilton, 1976). Some scales are too long (Wing *et al*, 1974; Copeland *et al*, 1976; Åsberg *et al*, 1978). The HDRS is a more precise discriminator of diagnostic groups than self-rating scales (Miller, 1980) and, as might be expected, fulfils the scale criteria of validity, reliability and sensitivity (Hamilton, 1976). Therefore we considered the HDRS most appropriate for use in the elderly.

The HDRS presupposes that all scores are the consequence of depression. We adapted the Hamilton Scale to form a semi-structured, interviewer-assisted questionnaire (Fig. 2), considering that the more formalised, structured approach would provide a more uniform data set. Taking into account the multiple disabilities of the elderly, it was considered that a score of 0–13 would include all normals but would not exclude all depressed patients.

Mental Test Score

The Information–Memory–Concentration Test of Blessed *et al* (1968) as modified by Hodkinson (1973) – the Mental Test Score (MTS) – has been used to evaluate medical factors in dementia and confusional states in the elderly. The maximum score on this test is 34; Lloyd (1970) and Hodkinson (1972) assumed a normal range of 25–34. It has been shown that the maximal improvement of scores on serial testing of 'normals' was 4.75, and that only 10% of all patients had a variation of 5 or more on serial testing (Stonier, 1973). Normality for this test can therefore be taken as 29–34, which would include all normals but would not exclude all demented patients.

To rationalise the direction of impairment in the three instruments used, the values for dementia are expressed as decrement of MTS (dMTS) – the score deficit below maximum (34). Our normal range is 0–5.

Usual activities (disability rating)

Garrad & Bennett (1971) discussed the measurement of disability by clinical assessment, by standard test of performance, and by questioning the patient on his current level of performance. They considered the third method to be most suitable for large-scale surveys conducted in patients' homes. They developed a 'Usual Activities' rating scale (UA), and a modification was used.

Statistical and computing techniques

The distributions of HDRS, dMTS and UA were highly skewed, and it would have been inappropriate to have used statistical techniques dependent on normal distribution. The skewness and kurtosis of the distributions of HDRS, dMTS and UA were calculated, using untransformed, square-root and logarithmically transformed data. Normality was assessed with the Y_S^2 test of Bowman & Shenton (1975). Both the untransformed and the logarithmically transformed distribution of data were not acceptably normal, but the distributions of the square-root transformed data were acceptably normal at 90, 95 and 99% levels. Therefore subsequent statistical analyses were performed using the square-root transformed data.

The associations of HDRS with dMTS, UA and dMTS and UA with HDRS were examined by linear and polynomial regression analyses. The residual sums of squares (RSS) and associated *F*-statistic of the linear and polynomial regression lines were calculated for comparison of goodness of fit. The regression lines, calculated from the transformed data, were plotted on axes labelled with untransformed values.

The relationships between HDRS and dMTS, UA and dMTS, and UA and HDRS were assessed using one-way analysis of variance (ANOVA) between each variable and sex, with age and the two remaining variables as covariates; the technique was constrained to include factors and then covariates serially, and all but two-way interaction terms were ignored. The classical experimental approach was adopted; each factor or covariate effect was adjusted for all preceding effects entering the analysis.

The relations of HDRS, dMTS and UA with age and sex were tested, using multiple linear regression analysis. These tests were repeated for the upper quartile of each data set – the 'best' 25%. The relation of dMTS with age in the subgroups 'depressed' and 'depressed and demented' was

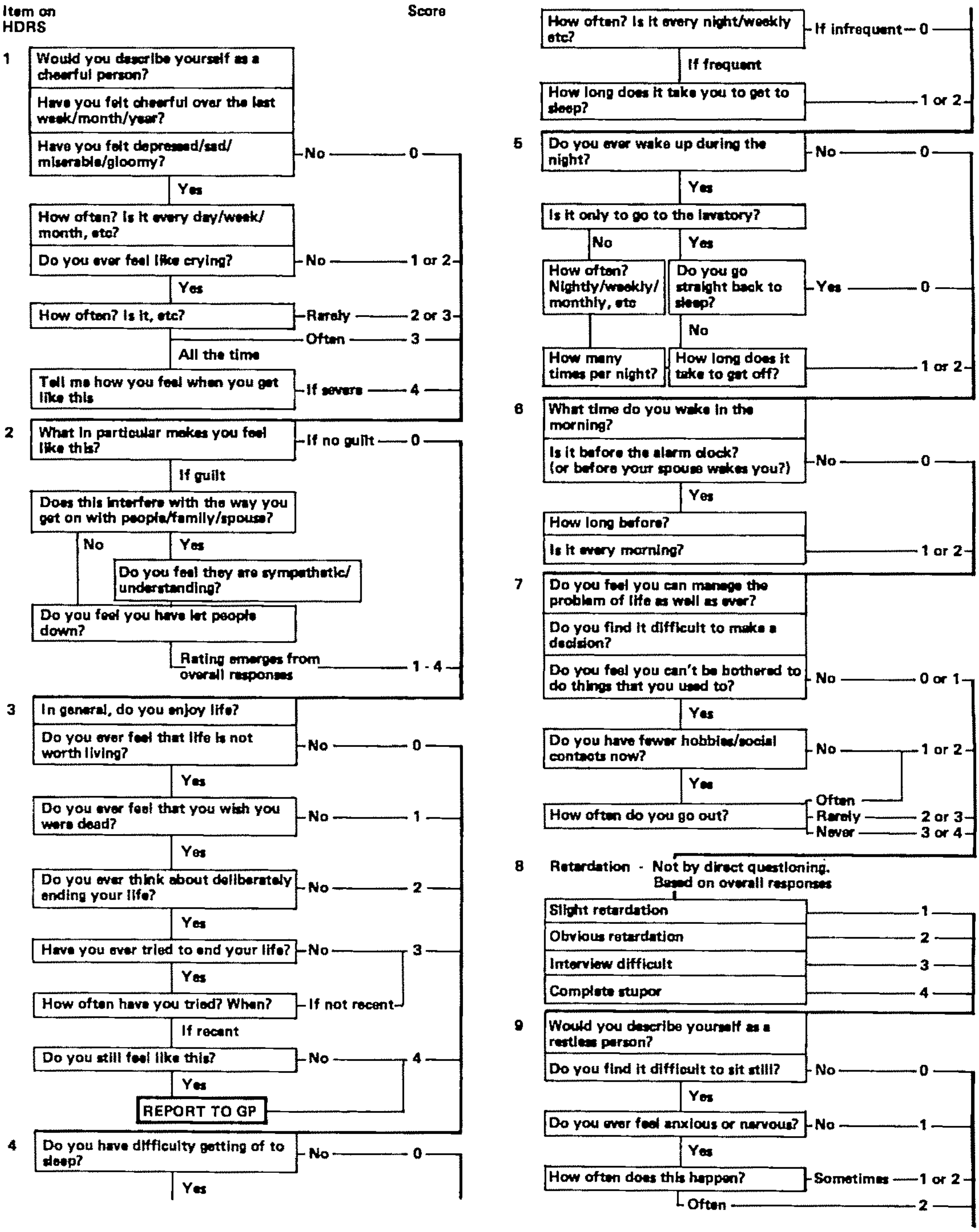
Item on HDRS
Score
1
Would you describe yourself as a cheerful person?
Have you felt cheerful over the last week/month/year?
Have you felt depressed/sad/miserable/gloomy?
No — 0
Yes
How often? Is it every day/week/month, etc?
Do you ever feel like crying?
No — 1 or 2
Yes
How often? Is it, etc?
Rarely — 2 or 3
Often — 3
All the time
Tell me how you feel when you get like this
If severe — 4
2
What in particular makes you feel like this?
If no guilt — 0
If guilt
Does this interfere with the way you get on with people/family/spouse?
No
Yes
Do you feel they are sympathetic/understanding?
Do you feel you have let people down?
Rating emerges from overall responses — 1 - 4
3
In general, do you enjoy life?
Do you ever feel that life is not worth living?
No — 0
Yes
Do you ever feel that you wish you were dead?
No — 1
Yes
Do you ever think about deliberately ending your life?
No — 2
Yes
Have you ever tried to end your life?
No — 3
Yes
How often have you tried? When?
If not recent
If recent
Do you still feel like this?
No — 4
Yes
REPORT TO GP
4
Do you have difficulty getting of to sleep?
No — 0
Yes
How often? Is it every night/weekly etc?
If infrequent — 0
If frequent
How long does it take you to get to sleep?
1 or 2
5
Do you ever wake up during the night?
No — 0
Yes
Is it only to go to the lavatory?
No
Yes
How often? Nightly/weekly/monthly, etc
Do you go straight back to sleep?
Yes — 0
No
How many times per night?
How long does it take to get off?
1 or 2
6
What time do you wake in the morning?
Is it before the alarm clock? (or before your spouse wakes you?)
No — 0
Yes
How long before?
Is it every morning?
1 or 2
7
Do you feel you can manage the problem of life as well as ever?
Do you find it difficult to make a decision?
Do you feel you can't be bothered to do things that you used to?
No — 0 or 1
Yes
Do you have fewer hobbies/social contacts now?
No — 1 or 2
Yes
How often do you go out?
Often
Rarely — 2 or 3
Never — 3 or 4
8
Retardation - Not by direct questioning. Based on overall responses
Slight retardation — 1
Obvious retardation — 2
Interview difficult — 3
Complete stupor — 4
9
Would you describe yourself as a restless person?
Do you find it difficult to sit still?
No — 0
Yes
Do you ever feel anxious or nervous?
No — 1
Yes
How often does this happen?
Sometimes — 1 or 2
Often — 2

FIG. 2 Hamilton Depression Rating Scale, as amended by Griffiths *et al* (present study).

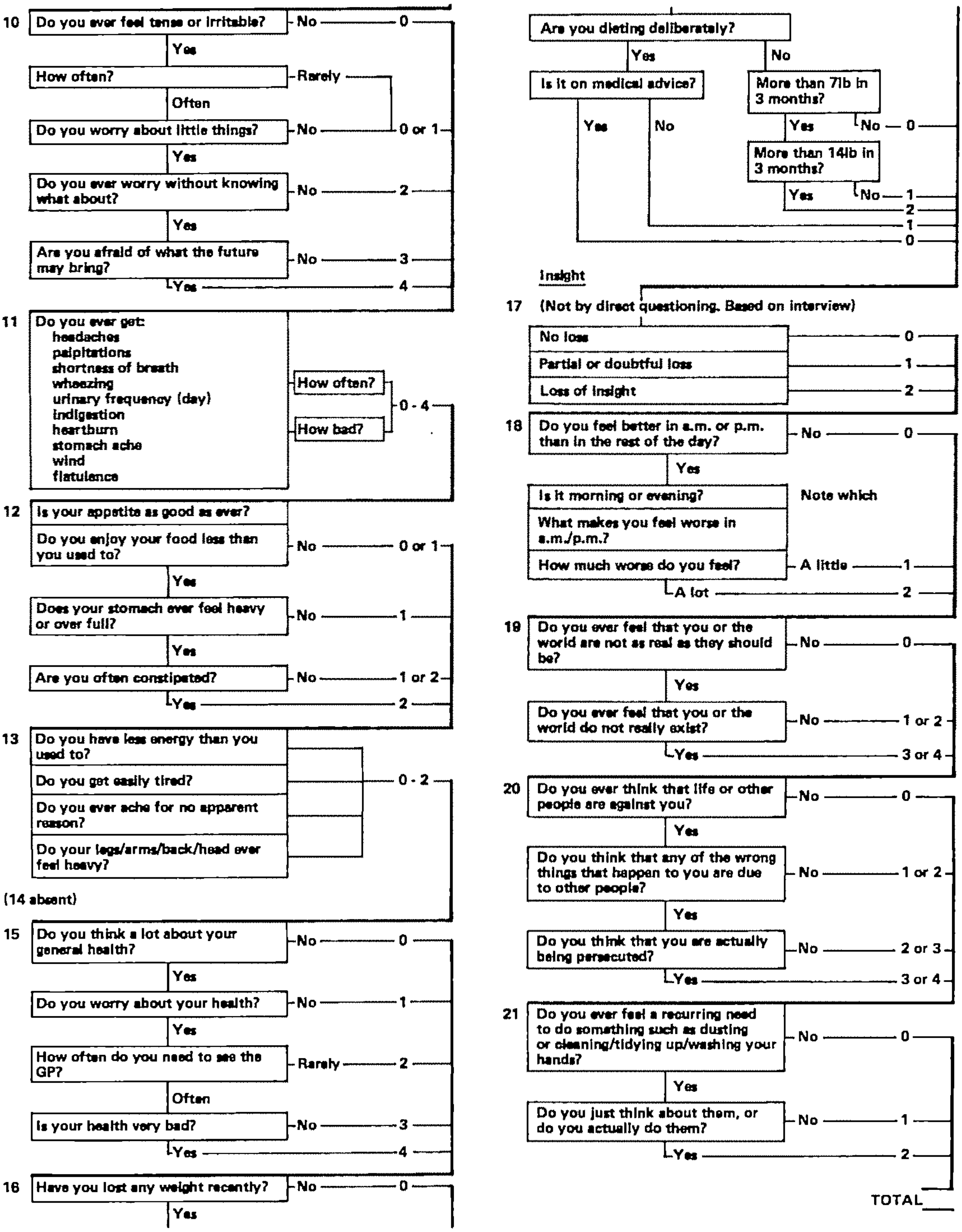
10 Do you ever feel tense or irritable? No 0
Yes
How often? Rarely
Often
Do you worry about little things? No 0 or 1
Yes
Do you ever worry without knowing what about? No 2
Yes
Are you afraid of what the future may bring? No 3
Yes 4
11 Do you ever get:
headaches
palpitations
shortness of breath
wheezing
urinary frequency (day)
indigestion
heartburn
stomach ache
wind
flatulence
How often?
How bad?
0 - 4
12 Is your appetite as good as ever?
Do you enjoy your food less than you used to? No 0 or 1
Yes
Does your stomach ever feel heavy or over full? No 1
Yes
Are you often constipated? No 1 or 2
Yes 2
13 Do you have less energy than you used to?
Do you get easily tired?
Do you ever ache for no apparent reason?
Do your legs/arms/back/head ever feel heavy?
0 - 2
(14 absent)
15 Do you think a lot about your general health? No 0
Yes
Do you worry about your health? No 1
Yes
How often do you need to see the GP? Rarely 2
Often
Is your health very bad? No 3
Yes 4
16 Have you lost any weight recently? No 0
Yes
Are you dieting deliberately?
Yes
No
Is it on medical advice?
More than 7lb in 3 months?
Yes
No
No 0
Yes
More than 14lb in 3 months?
Yes
No 1
2
1
0
Insight
17 (Not by direct questioning. Based on interview)
No loss 0
Partial or doubtful loss 1
Loss of insight 2
18 Do you feel better in a.m. or p.m. than in the rest of the day? No 0
Yes
Is it morning or evening? Note which
What makes you feel worse in a.m./p.m.?
How much worse do you feel? A little 1
A lot 2
19 Do you ever feel that you or the world are not as real as they should be? No 0
Yes
Do you ever feel that you or the world do not really exist? No 1 or 2
Yes 3 or 4
20 Do you ever think that life or other people are against you? No 0
Yes
Do you think that any of the wrong things that happen to you are due to other people? No 1 or 2
Yes
Do you think that you are actually being persecuted? No 2 or 3
Yes 3 or 4
21 Do you ever feel a recurring need to do something such as dusting or cleaning/tidying up/washing your hands? No 0
Yes
Do you just think about them, or do you actually do them? No 1
Yes 2
TOTAL

FIG. 2 continued

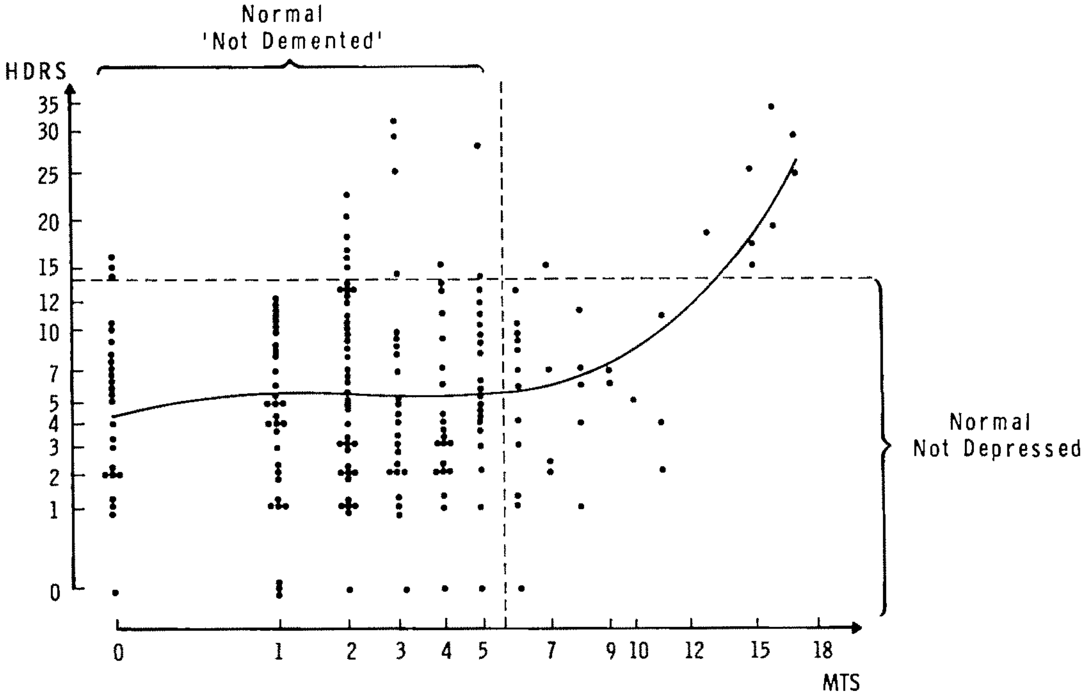

FIG. 3 Relationship between Hamilton Depression Rating Scale (HDRS) score and Mental Test Score (MTS) in 200 elderly people. Square-root transforms of the data are plotted; the axes are labelled with the untransformed values.

tested using linear regression analysis, and one-way ANOVA of dMTS by sex, with UA, HDRS and age as covariates.

In the analysis of variables with age, each of these variables is expressed graphically as the square of the mean of the square roots of the raw data - the square mean root (SMR). The analyses were performed on the individual square roots. Differences in each variable, associated with response patterns at annual review, were tested using χ^2, and two-way ANOVA by sex and response patterns, with the remaining variables and age as covariates. The technique was constrained to include covariates, then the factor 'sex', then the factor 'response', serially; all but two-way interactions were ignored, and the classical experimental approach was adopted.

These tests were performed on the University of Oxford's ICL 2988 computer, using the Statistical Package for the Social Sciences (SPSS [Nie *et al*, 1975]) and the SPSS Update 7-9 (Nie & Hull, 1981). Skewness and kurtosis were calculated using SPSS program 'Condescriptive'; simple linear regression was performed using 'Scattergram'; multiple linear regression and polynomial regression were performed using 'Regression'; paired t-tests were done by SPSS program 't-test'; and analysis of variance was undertaken using 'ANOVA'. The Y_S^2 *test and* χ^2 analysis of trend were performed manually.

Results

Psychometric tests (Fig. 3)

The scattergram of HDRS and dMTS showed that 13% of subjects were 'demented only', 8% were 'depressed only',

TABLE I
Prevalence of depression and dementia in 100 male and 100 female elderly people

	Depressed only	*Demented only*	*Depressed and demented*	*Normal*	*Total*	*All depressed*[1]	*All Demented*[2]
Male	1	16	2	81	100	3 (3%)	18 (18%)
Female	15	10	7	68	100	22 (22%)	17 (17%)
Total	16	26	9	149	200	25 (12.5%)	35 (17.5%)

1. Sum of 'depressed only' and 'depressed and demented'.
2. Sum of 'demented only' and 'depressed and demented'.

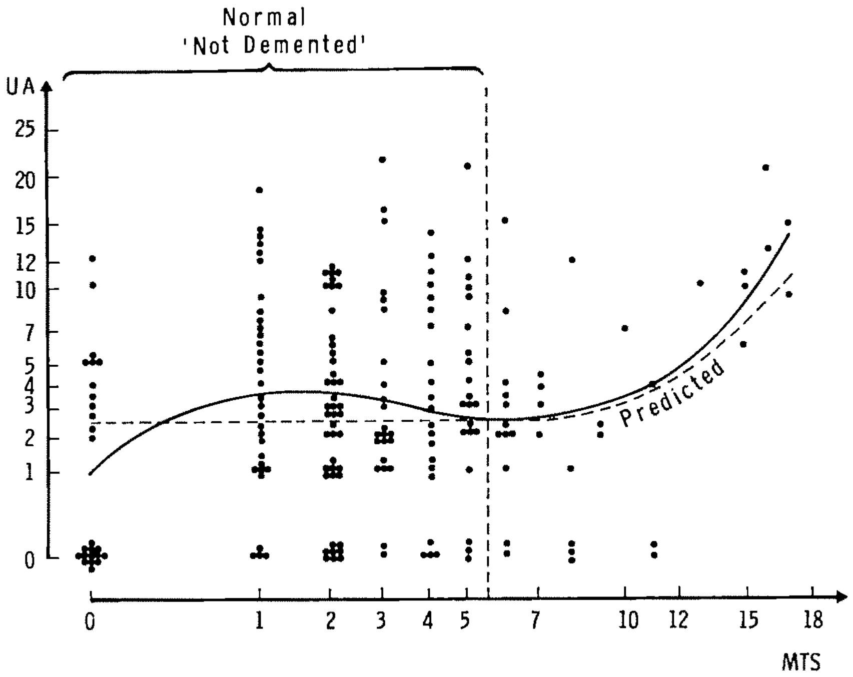

FIG. 4 Relationship between usual activities (UA) score and Mental Test Score (MTS) in 200 elderly people. Square-root transforms of the data are plotted; the axes are labelled with the untransformed values.

and 4.5% were 'demented and depressed'. The overall prevalence was 17.5% with dementia and 12.5% with depression; the prevalence of dementia in males and females was similar (M = 18%; F = 17%), but the prevalence of depression in males and females was disparate (M = 3%; F = 22%). The apparently lower prevalence of dementia alone in females was an artefact of the higher prevalence of depression with dementia (Table I). Small changes in criteria would produce disproportionately greater changes in prevalence. If the criterion of abnormality of the dMTS was altered from 6 (our criterion) to 9 (Hodkinson's criterion, *vide supra*), the overall prevalence of dementia fell from 17.5 to 10%.

A cubic polynomial provided a better model of the relationship between depression and dementia than did a quadratic ($F = 8.10$; d.f. = 3, 196; $P < 0.001$); introduction of a quartic term did not produce significant improvement. Raised HDRS was associated with raised dMTS (cubic regression, $R = 0.39$; F = 35.90; d.f. = 3, 196; $P < 0.001$). After extraction of the effects of sex, age and UA, HDRS was significantly related to dMTS (one-way ANOVA, $P < 0.002$).

Usual activities (disability rating)

Scores ranged between 0 and 24; 50% of subjects had significant disability in more than two items of the schedule. Forty-one (20.5%) of subjects had no disability. The median was 2.5, which reflected the competence of our sample; it would be expected that, in certain groups such as the housebound, the distribution would shift towards greater disability.

Relationship of dMTS to usual activities score (Fig. 4)

A cubic polynomial provided a significantly better model of the relationship between dementia and disability than did a quadratic ($F = 14.33$; d.f. = 3, 196; $P < 0.001$). Introduction of a quartic term did not provide significant improvement. Disability appeared to be related significantly to dementia (cubic regression, $R = 0.33$; $F = 24.94$; d.f. = 3, 196; $P < 0.001$).

However, after extracting the effects of sex, age and HDRS, there was no association between UA and dMTS (one-way ANOVA).

The regression equation produced by solving the separate equations for HDRS on dMTS, and UA on HDRS (*vide infra*) produced a predicted description of the relationship of UA to dMTS, which is also shown in Fig. 4. This predicted relationship was constructed by substituting the equation:

$$\sqrt{\mathrm{HDRS}} = a + b\,\sqrt{\mathrm{dMTS}} + c\,\mathrm{dMTS} + d(\sqrt{\mathrm{dMTS}})^3$$

into the equation:

$$\sqrt{\mathrm{UA}} = e + f\,\sqrt{\mathrm{HDRS}} + g\mathrm{HDRS}$$

This was remarkably consistent with the observed data.

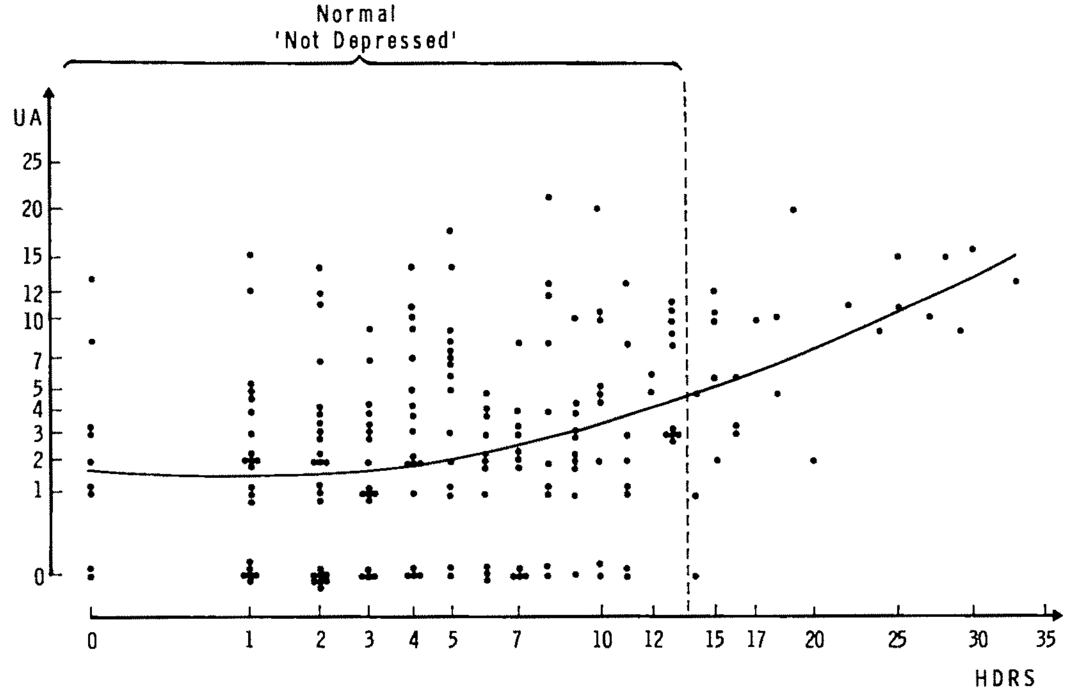

FIG. 5 Relationship between usual activities (UA) score and Hamilton Depression Rating Scale (HDRS) score in 200 elderly people. Square-root transforms of the data are plotted; the axes are labelled with the untransformed values.

Since UA and dMTS were not correlated (one-way ANOVA, *vide supra*), then a secondary relationship, of each with HDRS, was implied.

Relationship of usual activities score to HDRS (Fig. 5)

A quadratic polynomial provided a better model of the relationship between disability and depression than did a linear polynomial ($F=6.73$; d.f.$=3$, 197; $P<0.005$); introduction of a cubic term did not provide a significant improvement. Raised UA was associated with raised HDRS (quadratic regression, $R=0.41$; $F=40.28$; d.f.$=3$, 197; $P<0.001$).

After extracting the effects of sex, age and dMTS, UA was significantly associated with HDRS (one-way ANOVA, $P<0.001$).

Age and sex differences

In this structured sample, the age distribution of males and females was not different, either in the individual quinquennia or in the whole sample (paired *t*-test).

Hamilton depression rating scale (Figs 6 and 7)

Depression increased with age (two-way ANOVA, $P<0.01$): in females, depression increased from 60 to 69 years. At age 60–64 years, males and females did not differ but, at 65 years and over, females scored higher than males (paired *t*-test, $P<0.01$).

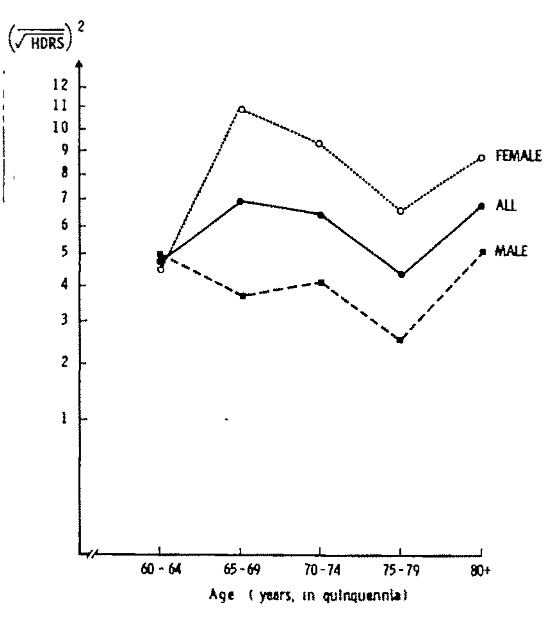

FIG. 6 Relationship between depression and age in 200 elderly people (100 male, 100 female). Square-root transforms of the data are plotted on the ordinate: the abscissa is linear.

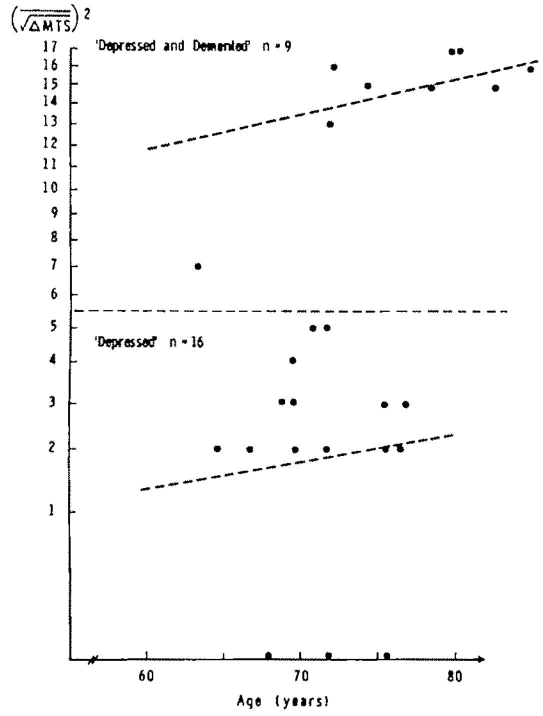

FIG. 7 Relationship between Mental Test Score (MTS) and age in depressed and demented elderly people. Square-root transforms of the data are plotted on the ordinate: the abscissa is linear.

Two groups of depressed patients are seen in Fig. 3. One group, the 'depressed only', had normal dMTS; the other, the 'depressed and demented', showed a relationship of HDRS to dMTS. The 'depressed and demented' group showed a trend of association between dMTS and increasing age (Fig. 7) (χ^2 analysis of trend, $P<0.01$).

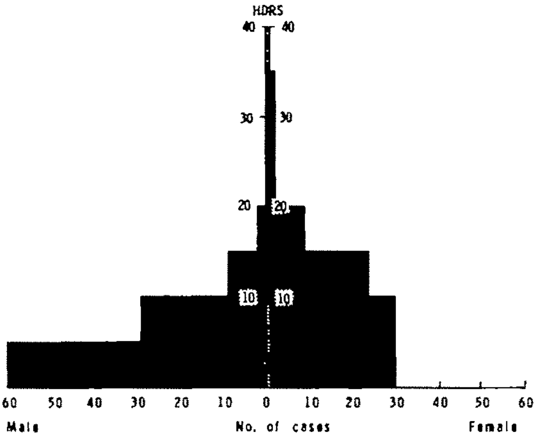

FIG. 8 Distribution of the Hamilton Depression Rating Scale (HDRS) score by sex in 200 elderly patients.

Insufficient cases of 'depressed' and 'depressed and demented' subjects were available to perform satisfactory ANOVA on these subjects separately from the whole group.

The distribution of HDRS score by sex is shown in Fig. 8 and SMR scores for both HDRS and dMTS in Table II. Females scored higher than males on every item of the HDRS. Females scored 14.5% of the maximum score and males 7.8%. In particular, 71% of those reporting sensations of depression were female. There was no significant difference between males and females in the MTS score total, nor in pattern of response (two-way ANOVA). On the HDRS, females scored more highly than males did ($P<0.001$); increased HDRS score was related to increased decrement of MTS ($P<0.02$; two-way ANOVA).

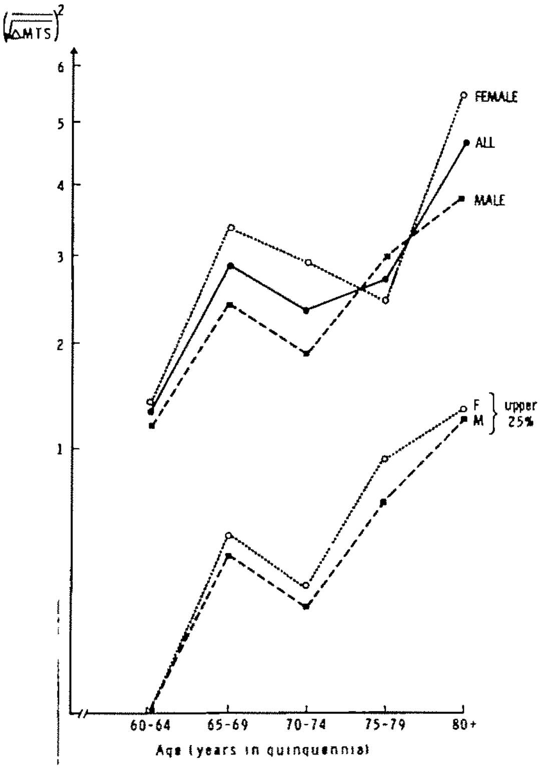

FIG. 9 Relationship between Mental Test Score (MTS) and age in 200 elderly people (100 male, 100 female). The square-root transform of the data is plotted on the ordinate: the abscissa is linear.

Mental test score (Fig. 9)

Dementia increased with age ($P<0.001$), but not significantly in those aged less than 75 years. Although females were more impaired, this was not significant (two-way ANOVA). The upper quartile of these patients showed a significant age-related increase in dMTS (linear regression, $P<0.01$).

TABLE II
Psychometric test scores in males and females

	Hamilton Depression Rating Scale score (HDRS)			*Decrement of Mental Test Score (dMTS)*		
	$(\overline{\sqrt{HDRS}})^2$	*Back-transformed 95% confidence limits*	*Range*	$(\overline{\sqrt{dMTS}})^2$	*Back-transformed 95% confidence limits*	*Range*
Male	3.98	3.24–4.80	0–20	2.36	1.84–2.94	0–15
Female	7.83	6.55–9.22	0–34	2.98	2.38–3.64	0–16

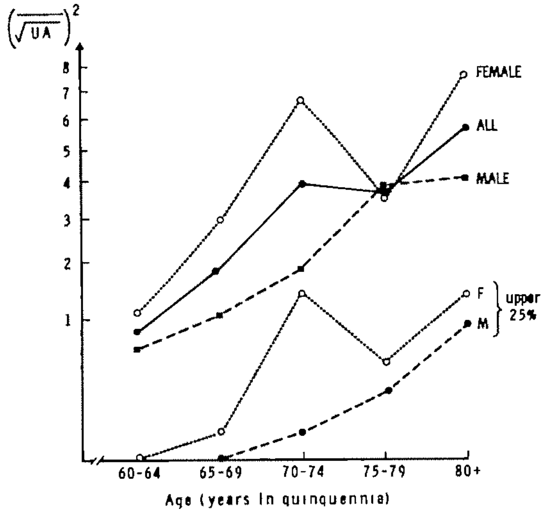

FIG. 10 Relationship between usual activities (UA) score and age in 200 elderly people (100 male, 100 female). Square-root transforms of the data are plotted on the ordinate: the abscissa is linear.

Usual activities (disability rating; Fig. 10)

Disability increased with age ($P<0.001$) and females were more impaired than males ($P<0.001$; two-way ANOVA). The upper quartile showed a significant trend with age (χ^2 analysis of trend, $P<0.02$); the difference between males and females was not significant (paired t-test).

Annual review

All patients were called for review in 1980. The scores discussed are those measured in 1979.

Response

Ten (5%) patients had died and two (1%) had moved from the practice. Non-repliers were those who did not reply to the follow-up request; refusers were those who replied but declined to participate.

Hamilton depression rating scale (Fig. 11)

Non-repliers had a higher HDRS than responders (χ^2, $P<0.01$), and patients who died also had a raised HDRS (χ^2, $P<0.01$). The refusers had a lower HDRS than the responders, but this was not significant. However, after extraction of the effects of age, sex, UA and dMTS, response type ('responded', 'did not reply', 'refused' or 'died') was not correlated with HDRS (two-way ANOVA).

Mental test score (Fig. 11)

Refusers did not differ from responders. Non-repliers and the patients who died had a greater dMTS in 1979 (χ^2, $P<0.01$; two-way ANOVA, $P<0.002$). After extraction of the effects of other factors and covariates, response type was significantly correlated with dMTS (two-way ANOVA, $P<0.002$).

Usual activities (disability rating; Fig. 11)

There was no significant difference between responders and non-repliers; refusers and those who died had shown a significantly higher degree of disability in 1979 (χ^2, $P<0.02$ and $P<0.005$ respectively). After extraction of the effects of other factors and covariates, response type was correlated significantly with UA (two-way ANOVA, $P<0.05$). The adjusted deviations for each response type show that refusal was not significantly correlated with increased UA, but death was.

Discussion

In the elderly, rating scale measures of depression, dementia and disability are inter-related and age-dependent. The criterion adopted for depression, HDRS > 13, showed a prevalence of depression of 12.5% in the sample, which is in agreement with previous community studies. The criterion we adopted for dementia, MTS < 29, showed a prevalence of dementia of 17.5% in the sample. If the criterion suggested by Lloyd (1970) and Hodkinson (1973), MTS < 26, was substituted, the prevalence of dementia was 10%. The latter figure was more in keeping with previous studies.

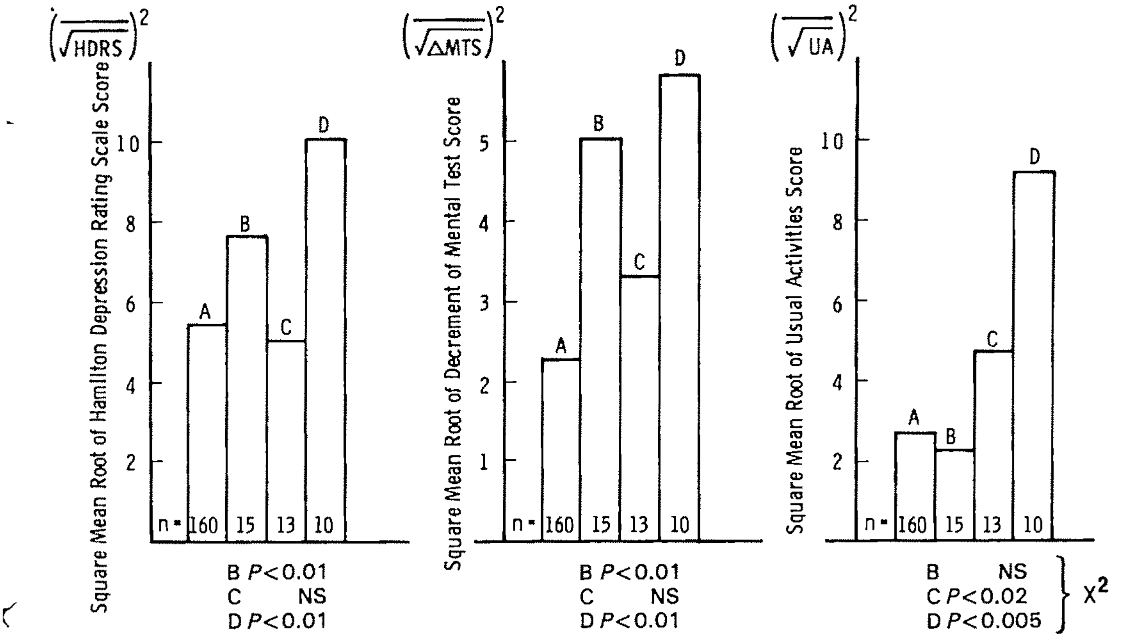

FIG. 11 Depression, dementia and disability at annual review. A, responders; B, did not reply; C, refused; D, died.

The prevalence of dementia did not differ between males and females throughout the age range, but the prevalence of depression did differ, with females scoring significantly higher on the HDRS than did males over the age of 65 years. In the 25% of 'best' patients, only 0.33% of all total scores showed values outside our normal range. Therefore, the 'best' 25% of patients may be regarded as unimpaired.

Figure 3 shows that increasing dementia is associated with increasing depression. There was insufficient inter-correlation between the items of the MTS and HDRS scales to explain this association, which suggests that these patients might have a particular type of depression associated with dementia. It also indicates that, taken in isolation, surveys of depression or dementia will produce overlap, which may account for some of the differences in community studies. A comparable study from New York (Gurland *et al*, 1980) showed a similar prevalence of depression and dementia and a similar age-related change to our study; a synthesis of the data from the New York and London studies confirms the necessity of combined assessment of depression and dementia (Gurland *et al*, 1983), as does a Washington study (Reifler *et al*, 1982). Considerably improved differential diagnosis between depression and dementia is obtained by multidimensional discriminant analysis (Gurland *et al*, 1982). However, it is assumed that depression and dementia are mutually exclusive diagnoses – a necessary concomitant of taking only one discriminant function. Analysis on the first two discriminant functions would permit classification as depressed, demented, or depressed and demented.

In this sample, the prevalence of depression was increased in females over the age of 65, being significantly higher than in males thereafter. There was no age-related trend in the least depressed quartile.

In this sample, females were not more demented than males; there was a significant overall increase in dementia over the age of 75 years. There was a slow increase in dMTS in the upper quartile of both males and females, which underlaid a change of greater magnitude in the more impaired. We consider this to be age-related benign senescent memory loss (Kral, 1978); the prevalence of maximum MTS (34/34) at age 80 was only 9% of that at age 60. Disability increased with age, and females were more impaired than males; the 'best' quartile showed a gradual increase in disability with age.

Depression and dementia are related. Depressed patients fall into two groups – those with normal dMTS in which dMTS is not age-related, and those with higher dMTS in which the dMTS is age-related. Depression and disability are also related; there was a correlation between increasing depression and increasing disability. By therapeutic modification of the HDRS, it might be possible to determine the degree of causality attributable to depression in this relationship.

While there is an association between disability and dementia, analysis of variance shows that this is secondary to the relationship of both these variables to depression. The predicted relationship between UA and dMTS, calculated from the regression equations of these with HDRS, corresponds closely to the directly calculated relationship.

Differences in disability do not influence response or refusal, but differences in MTS do; non-repliers and patients who died were more demented than were responders or refusers. Differences in HDRS do not influence response or refusal. As would have been expected, deaths occurred among the more disabled; physical disability is one determinant of poor outcome of depressive illness (Murphy, 1983).

Refusal is different from non-response; Akhtar (1972) found in a random sample that depression was more common among acceptors; dementia and anxiety were more common among refusers, but the differences were not statistically significant. It was concluded that refusers do not introduce significant bias into random sample estimates of mental health of the elderly patient.

Our figures of annual review show that non-response is probably allied to dementia and that refusal came from the least depressed. Disability played no part in either non-response or refusal.

Acknowledgements

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References

Akhtar, A. J. (1972) Refusal to participate in a survey of the elderly. *Gerontologia Clinica*, **14**, 205–211.

——, Broe, G. A., Crombie, A., McLean, W. M. R., Andrews, G. R. & Caird, F. I. (1973) Disability and dependence in the elderly at home. *Age and Ageing*, **2**, 102–111.

Åsberg, M., Montgomery, S. A., Ferris, C., Schalling, D. & Sedvall, G. (1978) A comprehensive psychopathological rating scale. *Acta Psychiatrica Scandinavica, Supplementum*, **271**, 5–27.

Bech, P., Gram, L. F., Dein, E., Jacobsen, O., Vitger, J. & Bolwig, T. G. (1975) Quantitative rating of depressive states: Correlation between clinical assessment, Beck's self-rating scale and Hamilton's objective rating scale. *Acta Psychiatrica Scandinavica*, **51**, 161–170.

Beck, A. T. (1967) *Depression: Causes and Treatment*. Philadelphia: University of Pennsylvania Press.

——, Ward, C. H., Mendelson, M., Mock, J. & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–571.

Blazer, D. G. (1980) The diagnosis of depression in the elderly. *Journal of the American Geriatric Society*, **28**, 52–58.

—— (1982) *Depression in Late Life*. St. Louis: Mosby.

Blessed, G., Tomlinson, B. E. & Roth, M. (1968) The association between quantitative measures of dementia and of senile change in the cerebral gray matter of elderly subjects. *British Journal of Psychiatry*, **114**, 797–811.

Bowman, K. O. & Shenton, L. R. (1975) Omnibus test contours for departures from normality based on $\sqrt{b_1}$ and b_2. *Biometrika*, **62**, 243–250.

Carney, M. W. P., Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of E.C.T. response. *British Journal of Psychiatry*, **111**, 659–674.

Copeland, J. R. M., Kelleher, M. J., Kellett, J. M., Gourlay, A. J., Gurland, B. J., Fleiss, J. L. & Sharpe, L. (1976) A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly: the geriatric mental test schedule. I. Development and reliability. *Psychological Medicine*, **6**, 439–449.

Freedman, N., Bucci, W. & Elkowitz, E. (1982) Depression in a family practice elderly population. *Journal of the American Geriatric Society*, **30**, 372–377.

Garrad, J. & Bennett, A. E. (1971) A validated interview schedule for use in population surveys of chronic disease and disability. *British Journal of Preventive & Social Medicine*, **25**, 97–104.

Gurland, B., Dean, L., Cross, P. & Golden, R. (1980) The epidemiology of depression and dementia in the elderly: the use of multiple indicators of these conditions. In *Psychopathology of the Aged* (eds J. O. Cole & J. E. Barrett). New York: Raven Press.

——, Golden, R. & Challop, J. (1982) Unidimensional and multidimensional approaches to the differentiation of depression and dementia in the elderly. In *Aging, Volume 19, Alzheimer's Disease: A Report of Progress in Research* (eds S. Corkin, K. L. Davis, J. H. Growden, E. Usdain & R. J. Wurtman). New York: Raven Press.

——, Copeland, J., Kuriansky, J., Kelleher, M., Sharpe, L. & Dean, L. L. (1983) *The Mind and Mood of Ageing*. New York: Haworth Press.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

—— (1976) Comparative value of rating scales. *British Journal of Clinical Pharmacology*, 3 (Supplement 1), 58–60.

Herbst, K. G. & Humphrey, C. (1980) Hearing impairment and mental state in the elderly living at home. *British Medical Journal*, **281**, 903–905.

Hodkinson, H. M. (1972) Evaluation of a mental test score for assessment of mental impairment in the elderly. *Age and Ageing*, **1**, 233–238.

—— (1973) Mental impairment in the elderly. *Journal of the Royal College of Physicians, London*, **7**, 305–317.

Kay, D. W. K. & Roth, M. (1955) Physical accompaniments of mental disorder in old age. *Lancet*, **ii**, 740–745.

—— & Bergmann, K. (1980) Epidemiology of mental disorders among the aged in the community. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, NJ: Prentice Hall.

Kearns, N. P., Cruickshank, C. A., McGuigan, K. J., Riley, S. A., Shaw, S. P. & Snaith, R. P. (1982) A comparison of depression rating scales. *British Journal of Psychiatry*, **141**, 45–49.

Kral, V. A. (1978) Benign senescent forgetfulness. In *Aging, Volume 7, Alzheimer's Disease: Senile Dementia and Related Disorders* (eds R. Katzman, R. D. Terry & K. L. Bick). New York: Raven Press.

LLOYD, C. M. (1970) *Royal College of Physicians' Study of Mental Disease in the Elderly. Report of Findings of a Pilot Study.* London: Royal College of Physicians.

MILLER, N. E. (1980) The measurement of mood in senile brain disease: examiner ratings and self-reports. In *Psychopathology in the Aged* (eds J. O. Cole & J. E. Barrett). New York: Raven Press.

MILNE, J. S., MAULE, M. M. & WILLIAMSON, J. (1971) Method of sampling in a study of older people with a comparison of respondents and non-respondents. *British Journal of Preventive & Social Medicine*, **25**, 37–41.

MORTIMER, J. A., SCHUMAN, L. M. & FRENCH, L. R. (1981) Epidemiology of dementing illness. In *The Epidemiology of Dementia* (eds J. A. Mortimer & L. M. Schuman). New York: Oxford University Press.

MURPHY, E. (1983) The prognosis of depression in old age. *British Journal of Psychiatry*, **142**, 111–119.

NIE, N. H. & HULL, C. H. (1981) *SPSS Update 7-9: New Procedures and Facilities for Releases 7-9.* New York: McGraw Hill.

——, ——, JENKINS, J. G., STEINBRENNER, K. & BENT, D. H. (1975) *Statistical Package for the Social Sciences*, 2nd ed. New York: McGraw Hill.

REIFLER, B. V., LARSON, E. & HANLEY, R. (1982) Coexistence of cognitive impairment and depression in geriatric outpatients. *American Journal of Psychiatry*, **139**, 623–626.

ROTH, M. & KAY, D. W. K. (1956) Affective disorder arising in the senium. II Physical disability as an aetiological factor. *Journal of Mental Science*, **102**, 141–150.

STONIER, P. D. (1973) A comparison of two mental status questionnaires used in the assessment of dementia. Elective period project report, University of Manchester.

WEISSMAN, M. M., SHOLOMSKAS, D., POTTENGER, M., PRUSOFF, B. A. & LOCKE, B. Z. (1977) Assessing depressive symptoms in five psychiatric populations: a validation study. *American Journal of Epidemiology*, **106**, 203–214.

WILLIAMSON, J., STOKOE, I. H., GRAY, S., FISHER, M., SMITH, A., McGHEE, A. & STEPHENSON, E. (1964) Old people at home. Their unreported needs. *Lancet*, **i**, 1117–1120.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms.* London: Cambridge University Press.

ZUNG, W. W. K. (1965) A self-rating depression scale. *Archives of General Psychiatry*, **12**, 63–70.

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Communicability and Thought Disorder in Schizophrenics and Other Diagnostic Groups

A Follow-up Study

A. BARNETT RAGIN and T. F. OLTMANNS

To evaluate qualitative differences in the nature of thought disorder, the 'cloze' procedure and the Scale for the Assessment of Thought, Language and Communication were used to compare speech samples from schizophrenic, depressive, manic, schizo-affective and normal subjects at two different times. At the acute phase, thought-disordered subjects (schizophrenics, manics and schizo-affectives) were less communicable than non-thought-disordered subjects (depressives and normals). Communicability increased with remission of the more flagrant features of disturbance. Comparison of the thought-disordered diagnostic groups in the rate and pattern of remission of specific features of thought disorder indicated that factors reflecting goal-disrupted cognition distinguished the groups.

The concept of formal thought disorder has figured prominently in psychiatric classification systems and in formulations of schizophrenia, including those of Bleuler and Kraepelin, but recent findings challenge the view that it is specific to schizophrenia. Comparable levels of thought disorder have been reported in manic and other psychotic patients (Andreasen, 1979*b*; Harrow & Quinlan 1977; Marengo & Harrow, 1984).

Efforts to assess diagnostic differences in the nature of thought disorder have suggested that traditional clinical descriptions of thought disorder vary in their diagnostic utility and specificity. Andreasen (1979*a*,*b*) found that certain phenomena traditionally regarded as indicators of thought disorder in schizophrenia, such as tangentiality, derailment, incoherence, illogicality and loss of goal, occurred with equal frequency in manic subjects. Of the 18 subtypes examined, only two – poverty of speech and poverty of content of speech – distinguished schizophrenics from depressives and manics. Other investigators have also found it difficult to identify diagnostic differences in the nature of thought disorder (Harrow *et al*, 1982; 1986; Harvey, 1983). As a consequence, investigators have argued that differences may emerge only through follow-up evaluation (Andreasen, 1979*b*; Harvey, 1983).

In a recent study, Harvey *et al* (1984) demonstrated that thought disorder is relatively stable in both manic and schizophrenic subjects during the first 10 days of hospital admission. Other studies have found indications of thought disorder in schizophrenic and in manic subjects 7 weeks following admission, during partial recovery (Harrow & Quinlan, 1977; Harrow *et al*, 1982; Grossman & Harrow, in press). However, it is not yet clear whether the course of thought disorder differs for schizophrenic and other thought-disordered groups over longer periods of time.

Reduced communicability, as measured by the 'cloze' procedure, has been found to distinguish thought-disordered from non-thought-disordered schizophrenics (Manschreck *et al*, 1979). In this procedure, raters attempt to supply words that have been deleted from a speech segment; the success of the raters in supplying the missing words provides an index of the predictability or communicability of the speech sample. This provides a method for quantifying the degree of listener confusion which is generated by a specific speech segment. In a previous study (Ragin & Oltmanns, 1983), we found that reduced communicability was associated with traditional clinical indicators of thought disorder, such as derailment, as assessed by the Scale for the Assessment of Thought, Language and Communication (TLC). The TLC is an instrument that can be used to evaluate 18 features of thought disorder in spontaneous speech samples (Andreasen, 1979*a*).

This paper extends our previous report with a follow-up examination of communicability and its relationship to specific features of thought-disordered speech in five diagnostic groups: schizophrenics, depressives, manics, schizo-affectives and non-psychiatric subjects. Further subjects were added to those represented in the previous report. All subjects were assessed at two instances: at admission and following hospital discharge. Post-hospital evaluation was necessary to determine whether thought disorder is only associated with acute disturbance in some psychotic groups, but is more persistent in schizophrenics, and to provide important evidence concerning the relative persistence of specific features of thought disorder among different diagnostic groups. Failure to demonstrate differences among groups might suggest that thought disorder is a general feature of psychosis. Alternatively, specification of diagnostic differences in the rate and pattern of remission of clinical features of thought disorder could further understanding of schizophrenia and other psychotic disorders.

Method

Speech samples

Subjects in the study included 12 schizophrenic, 12 depressive, 12 manic and 12 schizo-affective patients, interviewed shortly after admission to a psychiatric hospital. The composition of the current sample was as follows: eight schizophrenics, five depressives, ten manics and eight schizo-affectives participated in a previous study (Ragin & Oltmanns, 1983), while others were added in order to ensure adequate sample sizes. The non-psychiatric subjects were sampled specifically for the purposes of this study; therefore, none of these subjects were represented in the prior investigation. In addition, a non-psychiatric control sample of 12 subjects (municipal employees and clerical workers) was also interviewed at two instances. The non-psychiatric subjects were screened for prior personal or family psychiatric history before being included. At the beginning of each audiotaped interview, each subject was asked to describe how he or she had been feeling lately. The psychiatric patients were asked to provide an account of the events surrounding admission to the hospital, and the non-psychiatric ones to describe how they had come to their present employment. The interviewer tried to avoid interrupting the subject, and provided only minimal prompts necessary to facilitate discourse. For the psychiatric patients, the remainder of the interview followed the Schedule for Affective Disorders and Schizophrenia (SADS) (Spitzer & Endicott, 1978). Diagnoses were determined by two independent judges (a psychologist and a psychiatrist) using the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1978). Disagreements were resolved through further discussion. The groups did not differ significantly on any of the demographic features. Demographic information and information on length of hospital stay is presented in Table I.

TABLE I
Summary of subjects' demographic characteristics

	Schizophrenics	*Manic*	*Schizo-affectives*	*Depressives*	*Normals*
Sex: male/female	8/4	10/2	9/3	7/5	9/3
Age: years					
mean	28.4	30.1	26.3	30.2	37.0
s.d.	7.7	9.4	5.5	4.9	11.8
Education: years					
mean	12.6	13.1	13.0	11.7	13.0
s.d.	2.5	1.7	1.4	2.4	1.2
Number of past admissions					
mean	2.6	2.8	1.7	0.5	NA
s.d.	1.5	0.7	2.2	1.4	NA
Months between initial and follow-up testing					
mean	8.2	7.1	5.6	6.6	7.6
s.d.	2.9	2.2	2.8	3.8	0.9
Medication: % initial follow-up					
Neuroleptic	83/58	08/0	58/25	0/08	NA
Antidepressant	0/0	0/08	08/0	75/50	NA
Lithium Carbonate	0/08	33/50	25/08	0/0	NA
Neuroleptic & lithium	17/0	58/33	08/33	0/0	NA
Antidepressant & neuroleptic	0/0	0/0	0/0	08/08	NA
None	0/33	0/08	0/33	17/33	NA

TABLE II
Definitions of clinical thought-disorder categories (Harvey & Neale, 1983)

Category	*Definition*
Poverty of speech	Restriction in the amount of spontaneous speech
Poverty of content	Speech adequate in amount but low in information of speech
Pressure of speech	Increase in amount of spontaneous speech
Distractibility	Interruption of a train of discourse, with focus shifted to an external object
Tangentiality	Replying to a question in a manner not related to the question
Derailment	Ideas expressed in spontaneous speech are obliquely related to previous speech
Illogicality	Overtly expressed reasoning that breaks logical rules
Circumstantiality	Indirect and lengthy speech gets to a goal slowly if at all
Loss of goal	Speech that never reaches logical end points
Blocking	Interruption of a train of speech, with comment from patient that thought is blocked

Thought disorder was assessed at both the initial and follow-up time periods, using Andreasen's (1979*a*,*b*) Scale for the Assessment of Thought, Language and Communication. The ratings include 18 separate subcategory scores and a global measure of thought disorder. Subcategories include among others: derailment, tangentiality, distractibility and loss of goal. A summary of the rating system, including definitions of the most frequent subcategories, is presented in Table II. Ratings were based on the independent judgements of two research assistants, who listened to each audiotaped interview and assessed either the actual number of instances in the 200-word sample, or, where appropriate, provided an ordinal rating of 0–4 for each of the thought disorder categories. At the initial time period, all of the schizophrenic, manic and schizo-affective subjects and none of the depressive and non-psychiatric subjects in the study would be considered thought disordered, based on one common criterion employed in previous reports – a score of two or higher on the Andreasen global rating (Andreasen, 1979*a*,*b*; Harvey 1983). Reliabilities for the more frequently occurring ratings ranged from 0.46 to 0.96, with a weighted kappa value for the global rating of 0.91. These reliability ratings were based on a fairly large sample, including a subsample of the subjects represented in the present study, in addition to subjects not included in this group, for a total n of 51.

The 48 psychiatric subjects were all contacted following discharge from the hospital to arrange follow-up testing. There were other subjects who were tested initially, but were unwilling to participate in follow-up testing due to relocation or lack of interest; these included two schizophrenic, two depressive, one manic and three schizo-affective subjects. The interviews were conducted in the subject's home, between 3 and 13 months following the initial testing: at the beginning, each subject was asked to describe how he had been feeling and what he had been doing since the last interview. The remainder of the psychiatric interviews followed the SADS; the follow-up interviews were also rated for thought disorder, using Andreasen's system.

The first uninterrupted 200-word segment from each interview was transcribed; to ensure confidentiality, it was necessary to change proper names or personal information that appeared in the original speech segment (e.g. birthdate, place of birth, employer). These changes were made consistently throughout the cloze transcript, so that they did not alter the communicability of the passage. The transcripts were prepared for cloze analysis by omitting all punctuation and replacing every fourth word with a blank.

Cloze procedure

Four introductory psychology students served as judges; each was asked to cloze all 120 passages — two from each of the 60 subjects, passages being presented in random order. All raters received the same instructions: they were asked to fill in the missing words, were urged to complete every blank and were given as much time as they needed to rate all 120 samples (averaging about 20 h over the course of 1 month).

The proportion of correctly filled blanks was computed to produce a cloze score, which was determined without knowledge of diagnosis or time period. For the most part, the criterion for scoring was a perfect match with the word in the original segment, but some allowances were made in scoring. If the rater supplied a synonym, it was scored as a correct response, since the speaker's meaning was communicated. In some instances, the word to be supplied was a proper name or a number that the rater could not guess, because the exact name or number could not be inferred from the context. In these instances, the blank was considered correctly rated if any proper name was supplied, where a proper name had occurred in the original passage, or if any number was supplied where a number had occurred. However, this practice was only necessary when it was impossible for the rater to infer the correct proper name or number from the context. In the few cases where a rater failed to fill in a blank, the omission was treated as an incorrect response.

Results

The means and standard deviations for the communicability measures (the cloze scores) at each of the two time periods are presented in Table III. Initially, the cloze scores at each time period were examined independently. One-way analysis of variance at the initial time period indicated a significant main effect for diagnosis ($F(4, 55) = 3.5$, $P < 0.05$). Table III indicates that when interviewed at the

TABLE III
Means and standard deviations for the cloze measure of communicability[1]

	Initial		*Follow-up*	
	Mean	*s.d.*	*Mean*	*s.d.*
Schizophrenics	0.549	0.061	0.649	0.092
Manics	0.500	0.071	0.578	0.083
Schizo-affectives	0.553	0.121	0.657	0.093
Depressives	0.590	0.113	0.598	0.138
Normals	0.633	0.076	0.655	0.083

1. Based on proportion correct.

initial time period, the normal subjects had the highest communicability scores, followed by depressives, schizo-affectives, schizophrenics and then manics. Planned comparison of the means for the schizophrenics and normals resulted in a significant difference ($P<0.01$). At the initial time period, schizophrenics were less communicable than were non-psychiatric subjects, which is in accord with earlier studies demonstrating reduced communicability among schizophrenics (Salzinger *et al*, 1964, Ragin & Oltmanns, 1983). Because the remaining comparisons were *post hoc*, they were assessed using the Newman–Keuls procedure, which resulted in a significant difference between the extreme groups: normals and manics. Table III indicates that the depressive subjects had generally higher communicability scores than the thought-disordered subjects had; in this, they resembled the normal subjects. Planned comparison of the thought-disordered (schizophrenics, manics and schizo-affectives) *vs* the non-thought-disordered groups (depressives and normals) resulted in a significant main effect for the grouping factor ($F(1, 58)=10.10$, $P<0.01$). Schizophrenics, manics and schizo-affectives (mean = 0.534, s.d. = 0.089) were less communicable than were depressives and normals (mean = 0.611, s.d. = 0.096).

One-way analysis of variance at the follow-up time period indicated no main effect for diagnosis. The groups did not differ significantly with respect to communicability following remission of psychiatric symptoms.

To assess the stability of communicability between the acute phase of disturbance and following hospital discharge, cloze scores from each of the two time periods for the 60 subjects were submitted to a repeated measures analysis of variance, with diagnosis and time as factors; this indicated a significant main effect for time period ($F(1, 58)=18.36$, $P<0.001$). Communicability increased for all groups between the initial and follow-up testing. As might be expected, correlations of the initial cloze scores with change scores for the individual psychotic groups were significantly negative. Thus, the subjects who were the least communicable at the initial time period (i.e. those who had the most potential for change) did in fact change the most. The interaction of time and diagnosis failed to reach significance at the 0.05 level ($P=0.13$). The means in Table III suggest that, on the measure of communicability, the groups tended to converge with the passage of time and remission of psychiatric symptoms.

Table IV presents the results of the correlational analysis of cloze scores and the Andreasen ratings. Of the 18 Andreasen subcategory scores, eight occurred infrequently; correlations with cloze measures were not computed for these ratings. Examination of the remaining ratings for the initial time period indicated that four subcategory scores were significantly correlated with communicability: derailment ($r=-0.29$, $P<0.05$), loss of goal ($r=-0.35$, $P<0.001$), pressure of speech ($r=-0.30$, $P<0.05$), and poverty of speech ($r=0.37$, $P<0.001$).

TABLE IV
Correlations of cloze scores with Andreasen thought-disorder ratings

Time period:	*Initial*	*Follow-up*
Poverty of speech	0.37***	0.37**
Pressure of speech	−0.30*	0.15
Distractibility	−0.13	0.11
Tangentiality	−0.03	0.13
Derailment	−0.29*	−0.18
Circumstantiality	−0.18	−0.23*
Loss of goal	−0.35***	−0.18
Blocking	0.06	—[1]
Poverty of content	−0.19	−0.38***
Illogicality	0.04	−0.07

1. A correlation could not be computed for this measure.
*$P<0.05$; **$P<0.01$; ***$P<0.0001$.

To assess potential diagnostic differences in the nature of thought disorder, the groups were compared on the four indices of thought disorder which were linked significantly to communicability. This included a cross-sectional analysis at the initial time period, as well as a longitudinal examination across the time periods. One-way analyses of variance at the initial time period resulted in one significant main effect: at the initial testing, the manic subjects received significantly higher scores on pressure of speech than did the other subjects ($F(3, 42)=4.4$, $P<0.01$).

Next, the subcategory ratings were submitted to a repeated measures analysis of variance, in order to evaluate diagnostic differences in the course of particular indices of thought disorder over time. Thus, the effect of interest was the interaction of time with diagnosis: significant interactions were obtained for two of the four subcategories: derailment and loss of goal ($F(4, 55)=2.90$, $P<0.05$; $F(4, 55)=3.78$, $P<0.01$, respectively). Means and standard deviations for these measures at each of the two time periods are presented in Table V: the significant interactions for derailment and loss of goal were due to the convergence of the scores between the two time periods. Among the thought-disordered diagnostic groups, the schizo-affective and schizophrenic subjects demonstrated a relatively greater degree of change than did the manic subjects on these measures. At the initial testing, during the acute phase of disorder, the schizo-affective and schizophrenic subjects received high scores on derailment and loss of goal, but by the time of follow-up, the scores for these subjects were very similar to those of the non-thought-disordered

TABLE V
Means and standard deviations for derailment and loss of goal

	Derailment				*Loss of goal*			
	Initial		*Follow-up*		*Initial*		*Follow-up*	
	Mean	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*
Schizophrenics	3.67	2.74	1.17	1.95	1.80	1.83	0.25	1.40
Manics	2.25	1.54	1.58	1.73	1.33	0.65	0.92	1.00
Schizo-affectives	4.42	3.45	1.08	1.44	1.33	1.43	0.42	0.67
Depressives	1.33	1.38	1.42	2.06	0.25	0.45	0.25	0.62
Normals	2.25	1.42	1.25	1.29	0.25	0.45	0.00	0.00

diagnostic groups (depressives and normals). The manic subjects displayed moderate levels of these features at the initial testing, but in contrast to the other thought-disordered subjects, did not demonstrate significant declines in derailment or in loss of goal between initial testing and follow-up. Paired *t*-tests of these differences supported these impressions. Schizophrenic and schizo-affective subjects demonstrated significant declines on the measures of derailment and loss of goal ($P<0.05$), while the manic subjects did not.

Discussion

Are there diagnostic differences during the acute inhospital phase of the psychiatric episode?

The interview transcripts of recently admitted schizophrenics were less communicable than those of either depressives or non-psychiatric subjects. Normal raters experienced greater difficulty producing correct inferences concerning the speaker's intended message, on the basis of the provided context; this is consistent with previous reports of reduced communicability among schizophrenics (Salzinger *et al*, 1964; Ragin & Oltmanns, 1983).

Schizophrenics were not unique in this regard. The transcripts of manic and schizo-affective subjects also posed difficulties for normal raters. The results of the 'cloze' analysis were consistent with findings of thought disorder among non-schizophrenics, particularly manic and schizo-affective subjects, who were distinguished from the depressive and normal subjects, in whom thought disorder has generally been reported to be minimal (Harrow & Quinlan, 1977; Andreasen, 1979*a*). Based on definitions employed in previous reports (e.g. Harvey, 1983), all the schizophrenic, manic and schizo-affective subjects in the present study would have been considered thought-disordered at the initial time period. At that time, the thought-disordered subjects were less communicable than were the non-thought-disordered subjects (depressives and normals). However, there were no diagnostic differences among thought-disordered groups (schizophrenics, manics and schizo-effectives) on this measure.

The manic subjects did not achieve higher communicability scores than the schizophrenics did; this is inconsistent with clinical descriptions of manic speakers as easier to understand than schizophrenics (Redlich & Freedman, 1966; Janowsky *et al*, 1970; Lipkin *et al*, 1970). In fact, the means in Table III indicate that manic speakers were generally the least communicable at both time periods. This finding also fails to support the conclusions of several studies in which manic and schizophrenic speech was compared in the incidence of various structural linguistic features. On the basis of differences between schizophrenics and manics in the incidence of analytically derived linguistic measures (e.g. cohesive ties), investigators have argued that manic speech is more fluent (Morice & Ingram, 1982) and more cohesive (Wykes & Leff, 1982).

Are there diagnostic differences in the course of thought disorder?

When interviewed 5–8 months after the acute phase, following remission of the more flagrant features of disturbance, psychiatric subjects could not be distinguished from non-psychiatric subjects in communicability. The following pattern emerged: depressive and normal subjects remained relatively stable, while schizophrenic, manic and schizo-affective subjects demonstrated significant increases in communicability between the initial and follow-up testing.

Thought disorder, as measured by the cloze procedure, appears to be associated with the acute phase of the psychiatric episode; it does not appear to be a stable deficit among all schizophrenics or other thought-disordered populations. The Kraepelinian view of progressive deterioration among schizophrenics and the related view of better outcome among affective disorders has been challenged by a growing body of literature. It has been found that not all schizophrenics continue to display thought disorder when examined over extended post-hospital periods (Marengo & Harrow, 1984).

Longitudinal evaluation of communicability in the three thought-disordered diagnostic groups examined in this study did not reveal diagnostic differences in the course of thought disorder over time. The schizophrenic, manic and schizo-affective groups all demonstrated significant increases in communicability at follow-up testing. The hypothesis of a differential course for thought disorder among manic and schizophrenic subjects, suggested by Andreasen (1979*a*,*b*), which would be consistent with traditional views of schizophrenia and the affective disorders (Bleuler, 1911; Kraepelin, 1919), was not supported.

What specific clinical indices of thought disorder were associated with reduced communicability at the acute phase?

The association between communicability and specific clinical indices of thought disorder at the initial time period suggests an important role for pressure of speech, poverty of speech, derailment and loss of goal. Pressure of speech and poverty of speech reflect phenomena that are opposite in nature: the latter characterises speech that is inappropriately restricted and laconic, whereas the former is characterised by an inappropriate excess of verbal production. Pressured speech can be described as a rapid, incessant monologue which is difficult for the listener to interrupt. The association between these measures and the cloze scores were in opposite directions: high scores on poverty of speech were associated with increased communicability, while high scores on pressure of speech were associated with reduced communicability. Although both reflect inappropriate verbal behaviour, pressured speech appears to be more closely associated with confusion on the part of the listener. The association between poverty of speech and communicability is consistent with the view that reduced or slowed verbal production may be an attempt to compensate for disorganised thinking.

To assess potential differences in the nature of thought disorder, the schizophrenic, manic and schizo-affective subjects were compared on the features which were associated with communicability at the acute phase: pressure of speech, poverty of speech, derailment and loss of goal. Examination of the specific indices of thought disorder at the initial time period failed to uncover any features of thought disorder that were specific to the diagnosis of schizophrenia. Of the four features examined, only pressure of speech distinguished among the thought-disordered diagnostic groups at the initial time period: the manic subjects received the highest scores on this measure. Associative errors have been demonstrated to increase under time pressure among thought-disordered subjects (Hemsley & Hawks, 1974). Descriptions of mania have emphasised the increase in verbal production (Redlich & Freedman, 1966) and the push of speech (Kraepelin, 1919). The negative association between communicability and pressure of speech may indicate that the incomprehensibility of manic speech, or manic thought disorder, is tied to this feature and supports views of a manic rate disorder (Harrow *et al*, 1986).

Do differences between thought-disordered diagnostic groups emerge when the significant clinical indices are examined over time?

The three thought-disordered diagnostic groups demonstrated a similar course for the features that were linked to quantity of speech: poverty and pressure. However, the groups differed in the features that were associated with goal-disrupted discourse: derailment and loss of goal. When initially tested during the acute psychiatric episode, the schizophrenic and schizo-affective subjects received high ratings on these features, while the manic subjects received more moderate scores. Between the initial and follow-up testing, 5–8 months following the acute phase of disturbance, the manic subjects remained fairly stable on the measures. However, the incidence of derailment and loss of goal declined significantly for the schizophrenic and schizo-affective subjects, dropping below the scores of the manic subjects.

In contrast to many features such as neologisms and clanging, described by Andreasen, derailment and loss of goal reflect communication deviance at the level of discourse planning and organisation. Discourse planning has been described by Deese (1978) as a process by which the speaker maps out his intended message. This form of communicative difficulty is in contrast to linguistic anomalies that occur at more immediate levels of discourse, such as the level of the individual word (e.g. neologisms), or at the level of the individual proposition (e.g. word salad). In the current study, features based on word-to-word difficulties occurred only rarely, if at all. Findings that syntactic organisation is maintained in thought-disordered subjects also suggest that thought disorder is not adequately explained by deviance at the more immediate levels of discourse (Gerver, 1967; Rochester, 1973).

Previous research comparing thought-disordered groups has generally found similarities rather than differences between the diagnostic groups (Andreasen & Powers, 1974; Harrow *et al*, 1982). Diagnostic-specific impairments have proved difficult to identify. The strong relationship that was found between thought disorder and clinical indices reflecting goal-disrupted cognition suggests that further research should be directed at these features specifically. More detailed evaluation of these phenomena and their relationship to other features of schizophrenia, schizo-affective disorder and mania may help to clarify important diagnostic and aetiological issues.

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References

Andreasen, N. C. (1979*a*) Thought, language, and communication disorders I. Clinical assessment, definition of terms, and evaluation of their reliability. *Archives of General Psychiatry*, 36, 1315–1321.

—— (1979*b*) Thought, language and communication disorders II. Diagnostic significance. *Archives of General Psychiatry*, 36, 1325–1330.

—— & Powers, P. S. (1974) Overinclusive thinking in mania and schizophrenia. *British Journal of Psychiatry*, 125, 452–456.

Bleuler, E. (1911) *Dementia Praecox or the Group of the Schizophrenias*. Translated (1950) by J. Zinkin. New York: International Universities Press.

Deese, J. (1978) Thought into speech: Linguistic rules and psychological limitations in processing information determine how we put our ideas into words. *American Scientist*, 66, 314–321.

Gerver, D. (1967) Linguistic rules and the perception and recall of speech by schizophrenic patients. *British Journal of Social and Clinical Psychology*, 6, 204–211.

Grossman, L. S. & Harrow, M. (in press) The early course of manic thought disorder. In *A Multidimensional Approach to Clinical Research in Schizophrenia* (eds R. R. Grinker Sr & M. Harrow). Springfield, Ill.: Charles C. Thomas.

Harrow, M. & Quinlan, D. (1977) Is disordered thinking unique to schizophrenia? *Archives of General Psychiatry*, 34, 15–21.

——, Grossman, L. S., Silverstein, M. L. & Meltzer, H. Y. (1982) Thought pathology in manic and schizophrenic patients. *Archives of General Psychiatry*, 39, 665–671.

——, Grossman, L. S., Silverstein, M., Meltzer, H. Y. & Kettering, R. (1986) A longitudinal study of thought disorder in manic patients. *Archives of General Psychiatry*, 43, 781–785.

Harvey, P. D. (1983) Speech competence in manic and schizophrenic psychoses: The association between clinically rated thought disorder and cohesion and reference performance. *Journal of Abnormal Psychology*, 92, 368–377.

—— & Neale, J. M. (1983) The specificity of thought disorder to schizophrenia: research methods in their historical perspective. In *Progress in Experimental Personality Research* (eds B. A. Maher & W. B. Maher) vol. 12. New York: Academic Press.

——, Earle-Boyer, E. A. & Wielgus, M. A. (1984) The consistency of thought disorder in mania and schizophrenia: An assessment of acute psychotics. *Journal of Nervous and Mental Disease*, 172, 458–463.

Hemsley, D. R. & Hawks, D. V. (1974) Speed of response and associative errors in schizophrenia. *British Journal of Social and Clinical Psychology*, 13, 293–303.

Janowsky, D. S., Leff, M. & Epstein, R. S. (1970) Playing the manic game. *Archives of General Psychiatry*, 22, 252–261.

Kraepelin, E. (1919) *Dementia Praecox and Paraphenia*. Translated (1971) by R. M. Barclay. Huntington, NY: Krieger.

Lipkin, K. M., Dryud, J. & Meyer, G. G. (1970) The many faces of mania. *Archives of General Psychiatry*, 22, 262–267.

Manschreck, T. C., Maher, B. A., Rucklos, M. E. & White, M. T. (1979) The predictability of thought disordered speech in schizophrenic patients. *British Journal of Psychiatry*, 134, 595–601.

Marengo, J. & Harrow, M. (1984) Schizophrenic thinking: Two and four year follow-up. Presented at the annual meeting of the American Psychiatric Association, 5–11 May.

Morice, R. D. & Ingram, J. C. L. (1982) Language analysis in schizophrenia: diagnostic implications. *Australian and New Zealand Journal of Psychiatry*, 16, 11–21.

Ragin, A. B. & Oltmanns, T. F. (1983) Predictability as an index of impaired communication in schizophrenia and affective disorders. *British Journal of Psychiatry*, 143, 578–583.

Redlich, F. C. & Freedman, D. X. (1966) *Theory and Practice of Psychiatry*. New York, Basic Books.

Rochester, S. R. (1973) The role of information processing in sentence decoding of schizophrenic listeners. *Journal of Nervous and Mental Disease*, 157, 217–223.

Salzinger, K., Portnoy, S. & Feldman, R. S. (1964) Verbal behaviour in schizophrenic and normal subjects. *Annals of the New York Academy of Sciences*, 105, 845–860.

Spitzer, R. & Endicott, J. (1978) *Schedule for Affective Disorders and Schizophrenia (SADS)* 3rd ed. New York: Biometrics Research, New York State Psychiatric Institute.

——, —— & Robins, E. (1978) *Research Diagnostic Criteria for a Selected Group of Functional Disorders*. New York: Biometrics Research, New York State Psychiatric Institute.

Wykes, T. & Leff, J. (1982) Disordered speech: Differences between manics and schizophrenia. *Brain and Language*, 15, 117–124.

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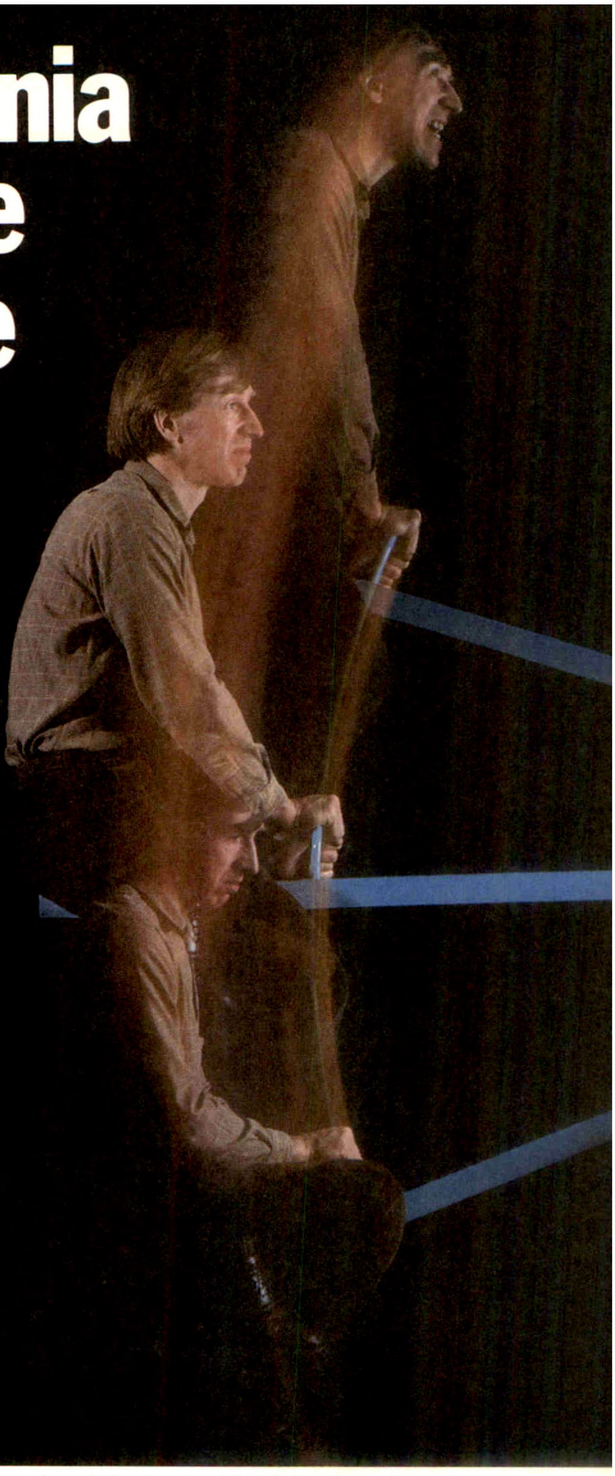

Schizophrenia therapy: the importance of balance

Too often, neuroleptic therapy swings the mood of the schizophrenic patient too far. Withdrawn patients' moods can be raised so high that they become agitated; whilst agitated patients may sink so low that they become withdrawn.

Experience shows that Piportil Depot treatment usually gets this critical balance right. Successful resocialization has been achieved even in patients who respond poorly to other neuroleptics.[1]

Piportil Depot contributes to putting the patient back into everyday life in other ways, too. Thanks to its relative freedom from extra-pyramidal effects,[2-5] and because injections need only be given every four weeks, patients hardly know they are being treated.

Piportil

DEPOT PIPOTHIAZINE PALMITATE

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Dosage: *(Adults)* Initially 25mg (0.5ml) by deep intramuscular injection into the gluteal region to assess susceptibility of patient. Dosage should be increased by increments of 25 to 50mg at appropriate intervals until a satisfactory response is obtained. Most patients respond to a dose of 50-100mg (1-2ml) every 4 weeks. Patients should be stabilised under psychiatric supervision. **Contra-indications:** Marked cerebral atherosclerosis, phaeochromocytoma, renal or liver failure, severe cardiac insufficiency or hypersensitivity to other phenothiazine derivatives. **Precautions:** A history of convulsive disorders or marked extra-pyramidal reactions to oral phenothiazines, in pregnancy and lactation. **Side-effects:** Reversible extra-pyramidal reactions, sleep disturbance, depression, blurred vision, asthenia, impotence, dry mouth, nausea, amenorrhoea, galactorrhoea, hypotension, hyperhydrosis and weight gain have been observed occasionally. Corneal or lenticular cloudiness have not been observed with Piportil alone, but in treatment with phenothiazine preparations in high dosage over a long period, there is a risk of such side-effects occurring. Local reactions at site of injection are rare. **Presentations and Hospital Prices:** Boxes of 10 x 1ml (50mg) £48.33 and 10 x 2ml (100mg) £79.08½ ampoules (PL0012/0117). **References:** 1. Imlah, N.W. (Awaiting publication). 2. Villeneuve, A. *et al.*, *Clin. Ther. Res.*, 1972, **14,** No.11, 696. 3. St. Laurent, J. *et al.*, *Can. Psychiatr. Assoc. J.*, 1974, **19,** 583. 4. Singh, A.N. & Saxena, B. *Curr. Ther. Res.*, 1979, **25,** 121. 5. Villeneuve, A. & Fontaine, P. *Curr. Ther. Res.*, 1980, **27,** 411. Piportil Depot is a Trade Mark. Further information is available on request.

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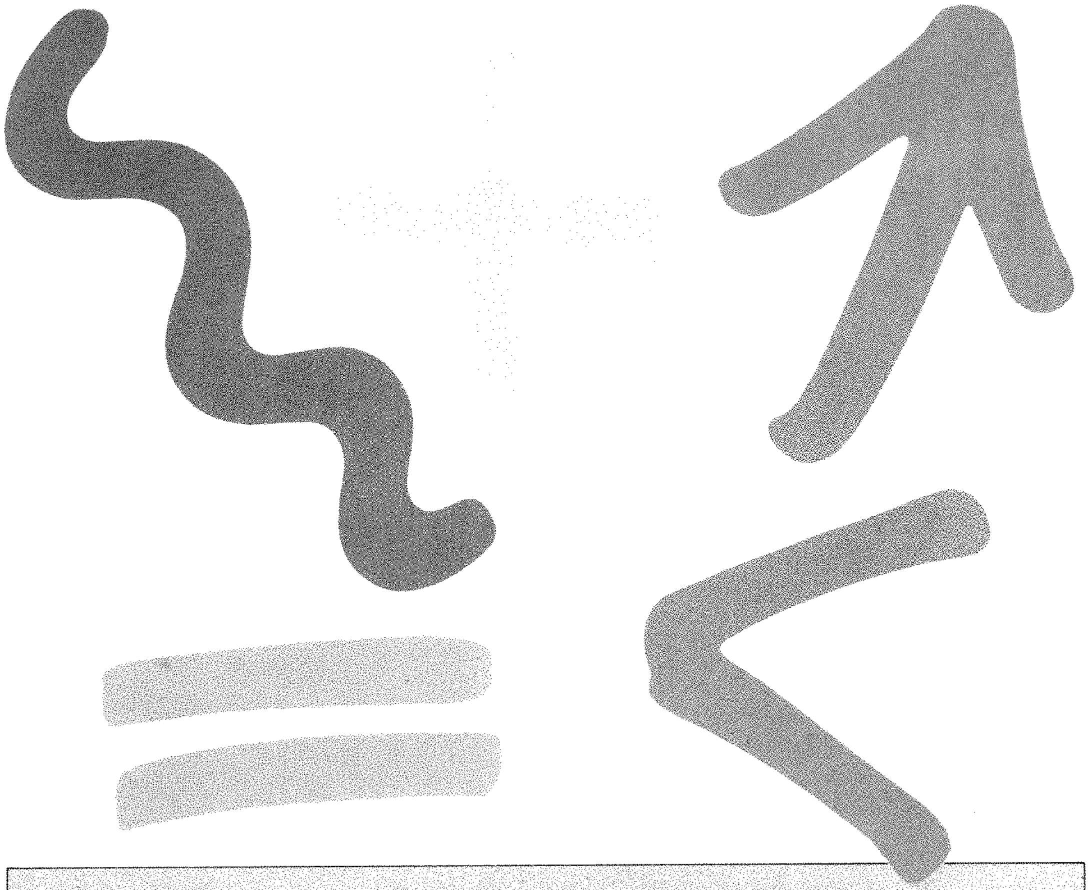

DIFFERENCE

= *Proven antipsychotic effect* (1-7)

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REFERENCES

1. Johnson D A W and Malik N A. Acta Psychiat Scand 1975, 51, 257-267
2. Pinto R, Bannerjee A and Ghosh N. Acta Psychiat Scand 1979, 60, 313-322
3. Hamilton M, Card I R, Wallis C G and Mahmood M R. Psychopharmacol 1979, 64, 225-229
4. Chowdhury M E H and Chacon C. Comp Psychiat 1980, 21, 135-139
5. Haslam M T, Bromham B M and Schiff A A. Acta Psychiat Scand 1975, 51, 92-100
6. Knights A et al. Brit J Psychiat 1979, 135, 515-523
7. Kelly H B, Freeman H L, Banning B and Schiff A A. Int Pharmacopsychiat 1977, 12, 54-64
8. Based on Basic NHS prices at September 1986

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A Comparative Hospital Survey of Psychotropic Drug Prescribing

M. MUIJEN and T. SILVERSTONE

In a survey of psychotropic drug prescribing for in-patients in three different types of psychiatric hospitals, the prevalence of combinations of more than one psychotropic drug varied from 45% in one hospital to 94% in another, with significant consistent differences between the use of drug combinations in the various hospitals. Combinations of two antipsychotic drugs were particularly frequent; the hospital with the lowest prevalence of polypharmacy was the only one with an associated psychopharmacology unit. Access to clinical pharmacology teaching may be an important factor in determining appropriate drug-prescribing habits.

A generally accepted principle of good therapeutic practice is not to prescribe more drugs to an individual patient than are strictly necessary. In psychiatry, the prescription of more than one or two psychotropic drugs to a given patient is unlikely to be beneficial and may be harmful (Merlis *et al*, 1970, 1972*a*; Hollister, 1974; Fann & Wheless, 1978), yet previous surveys (Sheppard *et al*, 1969; Merlis *et al*, 1970; Hemminski, 1977; Prien *et al*, 1973; Yosselson-Superstine *et al*, 1979; Michel & Kolakowska 1981; Morgan & Gopalaswamy, 1984; Edwards & Kumar, 1984) have shown that the habit of multiple drug prescribing is widespread.

Different studies have revealed wide variation in prescribing patterns: in a State Hospital in the USA 28% of chronic patients received multiple drug prescription (Sheppard *et al*, 1969), while in a Finnish mixed psychiatric population, 69% did so (Hemminski, 1977). There is a similar variation for specific drug combinations: approximately 10% of patients were given two antipsychotics in three studies (Sheppard *et al*, 1969; Hemminski, 1977; Michel & Kolakowska, 1981) but, in the fourth, Yosselson-Superstine *et al* (1979) found an incidence as high as 67% in an Israeli hospital. Despite such variations, there appear to be some general trends in multiple drug prescribing. For example, Hemminski (1977) noted a relationship with age: the incidence of polypharmacy among younger and older patients was lower than that in the age-group 51–60 years. Sheppard *et al* (1969) found that multiple drug prescribing occurred in 39% of female patients, compared to 19% of males.

There are difficulties in comparing one study with another, due to differences in sample selection and methodology: the American survey (Sheppard *et al*, 1969) was confined to chronic patients, the three English studies (Michel & Kolakowska, 1981; Edward & Kumar, 1984; Morgan & Gopalaswamy, 1984); involved acute, chronic and day patients, and the Finnish survey (Hemminski, 1977) included out-patients. The Israeli survey (Yosselson-Superstine *et al*, 1979) gave little precise information about the patients' characteristics.

The present study compared similar populations of patients in three different types of hospital in south-east England, to determine whether the pattern of psychotropic drug prescribing within the UK varies much from one hospital to another, or whether there are general overall similarities. Where appropriate, we have compared our findings with those of other recent UK surveys, one undertaken in an academic department of psychiatry (Michel & Kolakowska, 1981) and the two others in mental hospitals (Edwards & Kumar, 1984; Morgan & Gopalaswamy, 1984).

Method

The three hospitals which participated in our study were a London teaching hospital department with an inner city catchment area (hospital A), a provincial mental hospital with a university town and rural catchment-area, which provides a teaching facility for the local medical school (hospital B), and a large mental hospital in the Greater London Area with both an inner city and suburban catchment area (hospital C).

Only patients on acute admission wards were included. There were six such wards in hospital A (89 patients), three in hospital B (57 patients), and five in hospital C (104 patients), all serving a designated sector within the overall catchment area. Wards with specialist functions (alcohol, geriatric, adolescence, rehabilitation or long-stay) were excluded. Also excluded were patients on long-leave (i.e. longer than 1 week), as were patients who had been admitted less than 3 days before the survey.

Each ward was visited on a particular day, and the following information for each patient was recorded from the case notes and treatment sheets: diagnosis, sex, age and length of stay in hospital. All psychotropic drugs given the day before the survey were recorded (name of drug, dosage, number of administrations). As required (p.r.n.) medication was only included if given, it was otherwise recorded separately. The medication which had been given on the third day after admission was also recorded. Comparing this to the survey day medication allowed an observation of the change in medication which had taken place during the admission.

Results

Characteristics of patients

The characteristics of the patients in the three hospitals in terms of numbers, sex ratio, age distribution and diagnosis are given in Table I. The lower proportion of elderly (over 65) in hospital C is due to the fact that it has an active psychogeriatric department, to which most such patients are admitted. There was some variation in sex ratios between the hospitals, with a higher percentage of female patients in hospital B. Depressive illness was more predominant in women; the other diagnoses were equally represented in the two sexes.

General patterns of prescribing

The number of drugs given per patient in each of the three hospitals is shown in Table II, which includes the corresponding figures from two of the previous single-centre UK studies (Michel & Kolakowska, 1981; Morgan & Gopalaswamy, 1984) for comparison. More than one psychotropic drug was being given to less than half of the patients in hospital A (48%), compared to 82% in hospital B, and 74% in hospital C ($\chi^2 = 25.9$, $P < 0.001$). Similarly, more than two drugs were prescribed to 13% in hospital A, compared to over 40% in hospitals B and C. Within each hospital, there was consistency in the degree of multiple drug use between the different wards. Neither the age nor sex of the patients significantly affected the pattern of prescribing.

If the number and types of drugs given per diagnosis are examined (Table III), the same overall variation in prescribing pattern is seen, with a higher number of patients in hopsitals B and C receiving more than one drug than in hospital A. Many of these differences reach statistical significance. However, there were two exceptions to this general trend: the medication given to patients diagnosed as having personality disorders and to those diagnosed as having organic disorders was similar in all three hospitals, with more than one drug being given to only four of the 12 patients with the former diagnosis and four of the 24 with the latter.

Table I
Patient characteristics

Characteristics	*A (n = 89)*	*B (n = 57)*	*C (n = 104)*
Sex ratio (F:M)	1.5	3.7	0.9
Proportion aged			
18–39	39	37	40
40–64	36	45	52
65 and over	25	18	8
Percentage diagnosed as:			
schizophrenic	33	23	35
manic	12	14	21
depressed	24	45	21
organic	14	6	7
other	17	12	16

Table II
Number of drugs prescribed in each hospital

Percentage of patients receiving	*Hospital A*	*B*	*C*	*Michel & Kolakowska (1981)*	*Morgan & Gopalaswamy (1984)*
No drugs	25	5	4	19	8
1 drug	27	12	22	28	26
2 drugs	35	40	30	30	30
3 drugs	11	26	29	18	24
>3 drugs	2	16	15	5	12

The most frequent combination of drugs was that of an antipsychotic and an anticholinergic drug, while the next most frequent was the prescription of more than one antipsychotic drug, which occurred in 69% of the patients in hospital B (Table III).

For mania, most patients were prescribed a combination of lithium carbonate and an antipsychotic drug, with some patients receiving more than one antipsychotic.

Table III
Type of drugs used per diagnosis for number of patients

	A		*B*		*C*	
	n	*%*	*n*	*%*	*n*	*%*
Schizophrenia	(*n* = 30)		(*n* = 13)		(*n* = 37)	
1 antipsychotic only	7	23	0	0	6	16
1 antipsychotic (+ other drugs)	15	50	4	31	14	38
⩾2 antipsychotics (+ other drugs)	8	27	9	69	17	46
anticholinergic (+ other drugs)	14	47	10	77	25	67
Mania	(*n* = 11)		(*n* = 8)		(*n* = 22)	
1 antipsychotic only	0	0	1	12	3	14
1 antipsychotic (+ other drugs)	6	55	6	75	14	64
⩾2 antipsychotics (+ other drugs)	2	18	1	12	6	27
lithium alone	0	0	0	0	0	0
lithium and antipsychotic	5	45	6	75	7	32
Depression	(*n* = 20)		(*n* = 26)		(*n* = 22)	
no antidepressants	9	45	7	27	2	9
1 antidepressant only	3	15	2	8	4	18
antidepressant and antipsychotic	3	15	15	58	8	36
antidepressant and benzodiazepine	2	10	11	42	15	68

Seventy-five per cent of the patients diagnosed as suffering from depression were on at least one antidepressant. Antidepressants were given to five manic-depressive patients in a depressed phase of their illness, after having been admitted earlier in a manic state. Four patients, all on the same ward, were prescribed more than one antidepressant.

Twenty-two patients (14%) were on a depot antipsychotic as the only antipsychotic, while 41 patients (26%) were on a combination of oral and depot antipsychotics.

Some interesting patterns emerged when the specific use of the different antipsychotics was analysed. Depot neuroleptics were mainly used for schizophrenic patients; only four manic and five depressed patients were on this type of preparation, with all but one of these depressed patients being on flupenthixol decanoate.

For the treatment of schizophrenia and mania, chlorpromazine and haloperidol were the most popular drugs. The expectation that haloperidol would be the drug of choice for treating mania was not fulfilled – chlorpromazine was used as often. For depression both thioridazine and chlorpromazine were often prescribed, with chlorpromazine predominating in the younger patients. The use of thioridazine was largely restricted to a group of patients who were female and elderly, with a diagnosis of depression or dementia; within the brain-damaged group, this drug had a monopoly amongst the anti-psychotics.

Benzodiazepines were prescribed to 18 patients in hospital A (20%), 25 in hospital B (44%) and 54 in hospital C (52%), usually as hypnotics. Only seven patients were on benzodiazepines as the only form of medication, all given as hypnotics. Three different hypnotics (flurazepam, nitrazepam and temazepam) were prescribed in approximately equal numbers. Day-time benzodiazepines were given to five patients in hospital A (5.5%), nine in hospital B (16%), and eight in hospital C (6%). All but three of these were given diazepam. Eleven patients in the three hospitals were receiving two different benzodiazepines, one being used as a day-time anxiolytic and the other as a hypnotic.

Antiparkinsonian drugs were given to 84 of the 150 patients on antipsychotics (53%); 52 of these had been on such drugs from the time of admission. When only the 52 patients who had been in hospital for longer than 3 months at the time of this survey were considered, it was found that 34 had been on antiparkinsonian drugs continuously. Duration of treatment with antipsychotics did not appear to be an important factor in the decision whether or not to discontinue treatment – only seven patients had had the anticholinergic drugs stopped, all were in hospital C and, in all of these, it followed reduction in the dose of neuroleptics.

A total of 99 patients had a prescription for one or more p.r.n. drugs. In 52, this involved a drug which was not being given as regular medication: an antipsychotic in 16 cases, of which seven constituted a potential second antipsychotic, in 16 an anticholinergic, and in 23 an hypnotic. Six patients could have had two new drugs added, and one patient even three. The incidence was similar in all the hospitals, although there was a marked variation between wards, especially in hospital A.

Discussion

In this, the first comparative survey of psychotropic drug prescribing undertaken in the UK, we found a wide variation from one hospital to another. The overall percentage of patients on a combination of more than one psychotropic drug in the three hospitals surveyed, although high (68%), was comparable with other studies in which figures varying from 28% (Sheppard *et al*, 1969) to 69% (Hemminski, 1977) were quoted. However, there was a marked difference in this respect between the three hospitals – half as many of the patients in hospital A as in hospitals B and C were prescribed more than one drug, which was true for almost every type of drug combination. The difference was even more marked for combinations of more than two drugs. Within each hospital, there was little difference in the prescribing of drug combinations from one ward to another.

Among combinations of psychotropic drugs, one that was especially common was that of two antipsychotic drugs. There is little evidence of any benefit being derived from the use of two antipsychotics rather than one (Freeman, 1967; Hemminski, 1977) and those studies which have shown a favourable result with combinations (Casey *et al*, 1962; Bennett & Hamilton, 1963; Talbot, 1967) suffer from serious methodological weakness. Furthermore, such combinations increase the risk of unpredictable interactions and side-effects (Hollister, 1974). An exception may be made for patients on depot drugs, for whom an oral antipsychotic may be added temporarily, although it should generally be possible to add the same drug orally as is being given in depot form.

Anticholinergic drugs were prescribed to a high proportion of patients on antipsychotics, many of them continuing to take anticholinergics for longer than 3 months.

Another popular combination was that of antipsychotic and an antidepressant in depressive illness. In the comprehensive review by Boulanger & Zarifian (1981) of the pharmacological and clinical aspects of the antipsychotic/antidepressant combinations, two indications are given for this: psychotic or agitated depression and clinical depression in schizophrenia. One of the few common findings in our three hospitals was the low number of antidepressants prescribed for schizophrenics; on the other hand, the combination of an antipsychotic and an antidepressant in depressed patients varied between 10 and 54%.

The finding of considerable differences in prescribing between, but not within the three hospitals surveyed suggests a consistent policy for prescribing within each hospital. There are a number of possible explanations for such a variation. First, there is a difference in the population served by the hospitals. Hospital A serves a deprived inner-city area with a working-class population, while hospital B serves a country, city and rural area, with a predominantly middle-class population. Hospital C serves two distinct areas, a deprived one comparable with that of hospital A and a suburban one more comparable with that of hospital B. As there was no difference in prescribing habits between the wards of hospital C, in spite of marked variations in socioeconomic status of patients from one ward to another, it is unlikely that variation in population accounts for the findings. Secondly, there were differences in the proportions of diagnostic categories: hospitals A and C had similar proportions of schizophrenic, depressed and manic patients, whereas hospital B had a relative excess of depressed patients. Nevertheless, more drugs were prescribed per patient over all the diagnostic categories in hospitals B and C, compared to A. This would suggest that the variation in terms of drug prescribing could not be explained simply on the basis of a difference in distribution of diagnostic categories.

Finally, the three hospitals have a different academic input. Hospital A is the only one in this survey with an academic department specialising in psychopharmacology, and this may have resulted in a greater emphasis being placed on more rational drug prescribing. In keeping with this possibility is the similar lower rate of polypharmacy seen in the Oxford University Department, which also has an active psychopharmacology unit (Michel & Kolakowska, 1981). Thus, the further development of clinical psychopharmacology teaching could have a real impact in reducing the frequency of multiple psychotropic drug prescribing, to the benefit of both patients and staff, as well as to the finances of the National Health Service. There is good evidence to suggest that drug prescribing is more influenced by training and exchange of ideas between doctors than by the presenting symptoms of patients (Merlis *et al*, 1972*b*; Edwards, 1982).

Acknowledgement

We wish to thank all the consultants in the three hospitals who allowed us to survey their prescribing patterns in their wards.

References

BENNETT, J. L. & HAMILTON, D. L. (1963) Sequential use of antidepressant drugs with chlorpromazine in chronic schizophrenia. *Psychiatric Quarterly*, **37**, 53-65.

BOULANGER, J. Ph. & ZARIFIAN, E. (1981) L'association neuroleptique-antidepresseur tricyclique. *L'Encephale*, **8**, 5-28.

CASEY, J. F., HOLLISTER, L. E., KLETT, C. J., LASKY, J. & CAFFEY, E. M. (1962) Combined drug therapy of chronic schizophrenics. *American Journal of Psychiatry*, **117**, 997-1003.

EDWARDS, G. J. (1982) Factors influencing the psychotropic drug choices of teachers of psychiatry. *British Journal of Clinical and Social Psychology*, **2**, 24-26.

EDWARDS, S. & KUMAR, V. (1984) A survey of prescribing of psychotropic drugs in a Birmingham psychiatric hospital. *British Journal of Psychiatry*, **145**, 502-507.

FANN, W. & WHELESS, J. (1978) Dangers of psychotropic polypharmacy. *Current Psychiatric Therapies*, 143-154.

FREEMAN, H. (1967) Therapeutic value of combinations of psychotropic drugs. *Psychopharmacology Bulletin*, **4**, 1-27.

HEMMINSKI, E. (1977) Polypharmacy among psychiatric patients. *Acta Psychiatrica Scandinavica*, **56**, 347-356.

HOLLISTER, L. (1974) Polypharmacy in psychiatry, is it necessary, good or bad. In *Rational Psychopharmacotherapy and the Right to Treatment* (ed. F. Ayd). Baltimore: Medical Communications.

MERLIS, S., SHEPPARD, C., COLLINS, L. & FIORENTINO, D. (1970) Polypharmacy in psychiatry: patterns of differential treatment. *American Journal of Psychiatry*, **126**, 123-127.

——, BEYEL, V., FIORENTINO, D., FRACCHIA, J. & SHEPPARD, C. (1972*a*) Polypharmacy in psychiatry: empiricism, efficacy and rationale. *American Psychiatric Therapy*, 89-97.

——, SHEPPARD, C. & FRACCHIA, J. (1972*b*) Psychiatrists characteristics and polypharmacy. *Canadian Psychiatric Association Journal*, **17**, (*Suppl. 2*), 89-92.

MICHEL, K. & KOLAKOWSKA, T. (1981) A survey of prescribing psychotropic drugs in two psychiatric hospitals. *British Journal of Psychiatry*, **138**, 217-221.

MORGAN, R. & GOPALASWAMY, A. K. (1984) Psychotropic drugs: another survey of prescribing patterns. *British Journal of Psychiatry*, **144**, 298-302.

PRIEN, R. F., BALTER, M. B. & CAFFEY, E. M. (1973) Hospital surveys of prescribing practice with psychotherapeutic drugs. *Archives of General Psychiatry*, **35**, 1271-1275.

SHEPPARD, C., COLLINS, L., FIORENTINO, D., FRACCHIA, J. & MERLIS, S. (1969) Polypharmacy in psychiatric treatment: Incidence at a State Hospital. *Current Therapeutic Research*, **11**, 765-774.

TALBOT, D. R. (1967) Are tranquilliser combinations more effective than a single tranquilliser? *American Journal of Psychiatry*, **117**, 997-1000.

YOSSELSON-SUPERSTINE, S., STERNIK, D. & LIEBENZON, D. (1979) Prescribing patterns in psychiatric hospitals in Israel. *Acta Psychiatrica Scandinavica*, **60**, 477-482.

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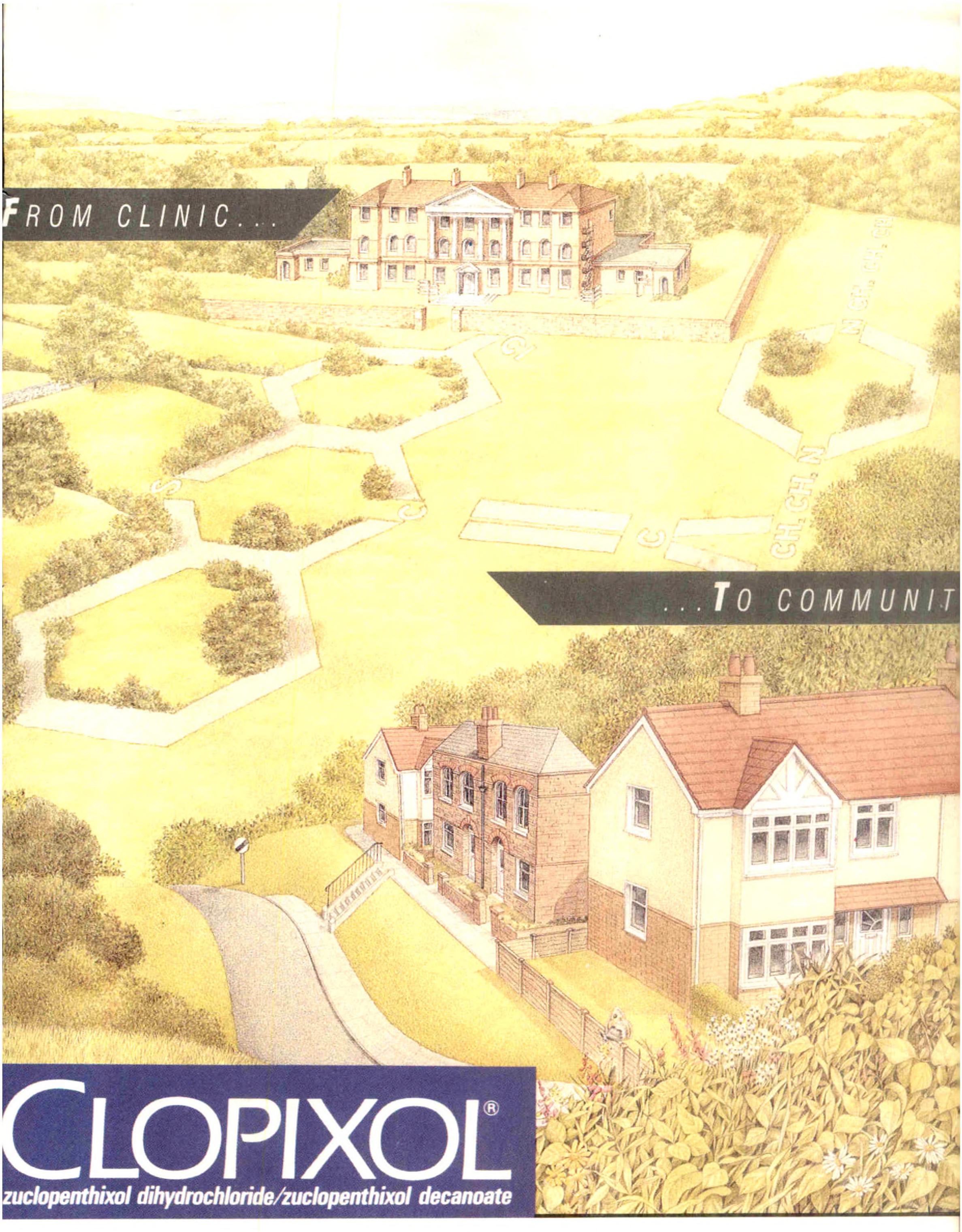

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Contraindications Comatose states.
Precautions Caution in patients with advanced renal, hepatic, or cardiovascular disease, Parkinson's disease, pregnancy, car driving or machine operation.
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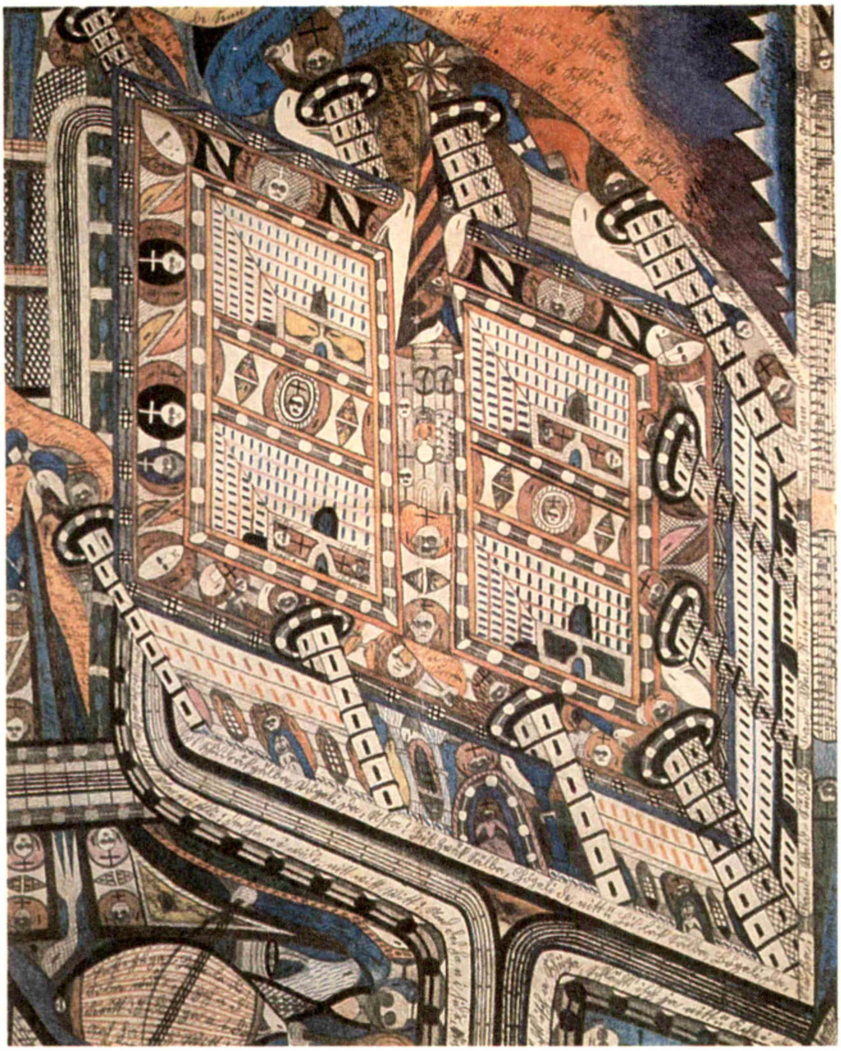

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The Compulsory Detention of Males of Different Ethnic Groups, with Special Reference to Offender Patients

D. McGOVERN and R. COPE

Compulsory detention rates of white, West Indian and Asian males under Part IV and Part V (offenders) of the 1959 Mental Health Act were compared: British-born West Indians and Asians were differentiated from migrants. Rates for Asians were similar to those for whites, but West Indians were significantly over-represented amongst compulsory detentions, especially as offender patients. A high total number of admissions and diagnostic differences accounted for the excess of West Indians admitted under Part IV, but not Part V.

The present study arose from the authors' impression that males of West Indian origin were over-represented, especially as offender patients, amongst compulsory admissions to psychiatric hospitals, compared with whites and Asians. This excess included both British-born and migrant West Indians.

The literature on immigrant groups and offender patient status is scarce: the only detailed study is that of Walker & McCabe (1973), who found nearly three times the expected number of West Indian immigrants, but fewer than expected Asians, in a sample of male offender patients. Norris (1984) found that 12% of male patients who left Broadmoor Hospital between 1974 and 1981 were 'non-white' (mostly West Indians). Other studies of detained patients have not separated offender patients for special analysis: Rwegellera (1970), Littlewood & Lipsedge (1977, 1981*a*) and Ineichen *et al* (1984) reported an excess of West Indians among compulsory admissions, and Pinto (1970) described the same for Asians. However, Szmukler *et al* (1981) found that ethnicity was not significantly associated with compulsory admissions, and Hitch & Clegg (1980) found no excess of formal admissions in a sample of New Commonwealth patients. Most of the studies considered migrants only, though Ineichen *et al* (1984) grouped together migrant and British-born West Indians. Significant numbers of the British-born children of immigrants are now adult, and warrant separate study to distinguish the effects of migration from other factors.

The present study examines three hypotheses:

1. That males of West Indian ethnic origin (both migrant and British-born) have significantly higher compulsory admission rates than have whites. This occurs for both non-offender patients who were detained under Part IV of the 1959 Mental Health Act (Sections 29, 25, 26) and offender patients detained under Part V (Sections 60, 65, 72 and 73), and the excess is independent of age, the total number of admissions and diagnosis. However, Asians have similar detention rates to those for whites.
2. That if West Indians do have excessive compulsory admission rates, this excess is greater under Part V (offender patients) than under Part IV (non-offender).
3. That there are no differences in detention rates between British-born West Indians and migrant West Indians.

There is no satisfactory classification of ethnic groups, but in this paper we refer to the following:

(a) Those born in the United Kingdom of British or Irish ancestry – *white*.
(b) Those of West Indian ancestry, which includes *West Indian immigrants* born in the Caribbean, and *British West Indians* born in the United Kingdom.
(c) Those of Asian ancestry which includes *Asian immigrants* born in the Indian subcontinent or East Africa and *British Asians* born in the United Kingdom.
(d) Others.

Throughout the study, we refer to the 1959 Act, which was in operation during the research period. The equivalents in the 1983 Mental Health Act are Part II (non-offender patients detained under Sections 2, 3 and 4) and Part III (offender patients detained under Sections 37, 41, 47 and 48).

Method

The psychiatric hospital selected for the study, All Saints', Birmingham, was chosen because its catchment area includes a large proportion of West Indian and Asian immigrants and their children. There are no other psychiatric units in the catchment area.

Sample

Three samples were obtained from the case notes of male patients between the ages of 16 and 65 years. Patients residing outside the catchment area were excluded, as were admissions to the Alcohol and Addiction Unit, because this offers a Regional service and does not admit compulsory patients. The samples were:

(i) All male patients admitted under Part V of the Mental Health Act 1959 for the 8-year period 1 January 1975 to 31 December 1982. There were 67 individuals.
(ii) All male patients admitted under Part IV of the Act in the 4-year period 1 January 1979 to 31 December 1982, comprising 217 individuals.
(iii) A list was obtained of all male admissions during the 4-year period 1 January 1979 to 31 December 1982, and alternate cases chosen, giving a 50% sample of individuals (574 cases). For each individual, the first admission only during the period was studied. This sample will be called the 'general admission sample'.

Information obtained from case notes

The following were recorded:

(i) age on admission;
(ii) ethnic group: (white, West Indian, Asian or other), identified from nursing records, which included colour, place of birth and clinical histories;
(iii) country of birth;
(iv) legal status: informal and formal: Part IV (29, 25, 26) and Part V (60, 65, 72 or 73) of 1959 Mental Health Act. There were no admissions under Section 136;
(v) final case note diagnosis: under one of the following: schizophrenia, paranoid psychosis, affective disorder, neurosis and personality disorder, drug or alcohol abuse, organic psychosis, drug-induced psychosis, other.

Catchment area population

The catchment area includes Sandwell Metropolitan Borough and the whole or part of seven wards in north-west Birmingham. The main ethnic groups are whites, West Indians and Asians.

Estimation of whites and West Indian and Asian migrant males

Total numbers were calculated for the precise catchment area, using tables for country of birth, by sex in the 1981 Census (Office for Population Censuses and Surveys [OPCS], 1982*a*); small area statistics and figures for enumeration districts were used where necessary. Unfortunately, there is no direct published estimate of the breakdown into age groups for country of birth below West Midlands County level. After consultation with Birmingham Central Statistical Office and in order to avoid possible variation within the county, table 11 in the Census (OPCS, 1982*a*) was used to obtain a breakdown into age groups. This gives "persons usually resident in private households, whether born inside or outside the United Kingdom by age of residents by birth place of head of household". Those born outside the UK are assumed to be immigrants from the country of birth of the head of the household. In fact, the percentages for each age group for Sandwell and Birmingham differed very little from the equivalent in the West Midlands County table mentioned above; these percentages were applied to our previous estimate of total numbers in each group. The eventual estimate for British West Indians and Asians was subtracted from that for all residents in the equivalent age groups (apart from numbers of the appropriate foreign-born groups) to leave an estimate for the white group.

British West Indians and Asians

No question on ethnic origin was asked in the 1981 census. However, there is support for the use of the birth-place of the head of the household (OPCS, 1982*a*, table 11) as an indicator of the ethnic group of those living in the house from both the Labour Force Survey (OPCS, 1982*b*) and the Birmingham Central Statistical Office (1984). The Labour Force Survey asked a direct question on ethnicity, but results were published for large areas only. In the West Midlands Metropolitan County, it was estimated that 89% of persons living in houses where the head was born in the New Commonwealth were of the same ethnic origin. It concluded that the birth-place of the head of the household was a good indicator of ethnic group, especially in large urban areas. The Birmingham Central Statistical Office (*op cit*) came to a similar conclusion, after checking census data with separate estimates of ethnic minority births and school children.

The figures for Sandwell were derived directly from the census. Those people born in the UK living in a house whose head was born in the Caribbean were assumed to be British West Indians. The census does not provide this information for individual wards, and the estimate for Birmingham wards was provided by the Birmingham Central Statistical Office (1984). Their data are derived from knowledge of the immigrant figures for wards, birth statistics and census data on the birth-place of the head of the household by individual country for Birmingham as a whole.

Reference to the head of the household table as an indicator of ethnic group neglects minority householders who were born in the UK. It was important not to underestimate the numbers of British West Indians, because this would introduce a bias in favour of the first hypothesis. Therefore we consulted the only other recent estimate of population by ethnic group – that in the 1978 National Housing & Dwelling Survey (Department of the Environment, 1979), which was based on a 1.6% sample in Birmingham. Having adjusted for population trends between 1978 and 1981, we found that this survey's estimate for the West Indian ethnic group exceeded the one described above by just over 10%. We therefore increased the estimate of British West Indians by this amount and applied the breakdown into age groups given in the head of household table, assuming equal numbers for each sex.

Analysis of data

Attention will be focused upon comparisons of the age-specific section rates (per 10 000 population) for immigrant groups with those for the white population. For Part IV rates, differences are tested for significance using the χ^2 test with the continuity correction. However, this test is inappropriate for testing differences between Part V age-specific rates, because the expected values for the immigrant groups are too small (near to unity or less). An overall weighted 'risk' ratio has therefore been computed for the West Indian migrants admitted under this section (excluding those born in Britain), after first testing for homogeneity.

Results

Table I shows the breakdown by ethnic group of the local male population between the ages of 16 and 65.

TABLE I

Age and ethnic group distribution of male residents (age 16–65) of catchment area

	Age group				
Ethnic group	*16–29*	*30–44*	*45–65*	*All Ages*	*%*
White	32 790	32 120	44 100	109 010	82.4
West Indian migrant	1210	1830	2550	5590	4.2
British West Indian	2440	140	—	2580	1.9
Asian migrant	5830	4330	3230	13 390	10.1
British Asian	1000	90	50	1140	0.9
Others				610	0.5
All groups				132 320	100

Detentions under Part IV

Numbers detained and detention rates per 10 000 of the population at risk are shown in Table II. The rates give the risk of being on a section at least once in the 4-year period, regardless of the number of admissions.

Highest rates are found in the West Indian migrants. The youngest group has a rate 17 times that for whites, and the middle group seven times; both these differences are highly significant ($P<0.001$). Unlike whites, the West Indians show sharply decreasing rates with increasing age.

The difference between the British West Indians and whites is also highly significant ($P<0.001$), but there is no significant difference between the British West Indians and the West Indian migrants.

No British Asians were detained. The 4-year rates for Asian migrants slightly exceed those for whites, but only for the 30–44-year group is this significant ($P<0.01$).

Detentions under Part V

Table III gives numbers detained and rates per 10 000 of the local population at risk. The rates give the risk of being detained under Part V at least once over the 8-year period.

The most striking differences are found once more between the West Indian migrants and whites. Rates for migrants all grossly exceed the comparable rates for whites, even in the oldest group. (Although it is likely that the ratio of the rate for immigrants to the rate for whites decreases with age, on these data the χ^2 test for heterogeneity gives a value of 4.37, which for two degrees of freedom gives $P>0.1$. The weighted mean ratio is 13.65 with 95% confidence limits of 9.02 and 23.23. using a method described by Armitage [1980].)

No British Asians were detained and only three Asian migrants; little can be made of such small numbers, but they would appear not to be over-represented. Only four British West Indians were detained; again, we were reluctant to make much of these numbers.

There is an additional problem with the British West Indian group. The population estimate is for 1981, but this particular sample of patients is derived from as far back

TABLE II

Male patients detained under Part IV and 4-year detention rates per 10 000 population at risk

	Age group							
	16–29		*30–44*		*45–65*		*All ages*	
Ethnic group	*n*	*Rate*	*n*	*Rate*	*n*	*Rate*	*n*	*Rate*
White	29	8.8	41	12.8	55	12.4	125	11.5
West Indian migrant	18	148.8**	16	87.4**	5	19.6	39	69.7
British West Indian	19	77.9**					19	73.6
Asian migrant	8	13.7	15	34.6*	6	18.6	29	21.7

*$P<0.01$; **$P<0.001$. The asterisked significance levels apply to ethnic minority group vs white, for similar age groups (N.B. differences for 'all age groups' not tested).

For 16–29 year old West Indian migrant versus British West Indian $\chi^2=3.38$ ($0.1>P>0.05$).

TABLE III

Males detained under Part V and 8-year detention rates per 10 000 population at risk

	Age group							
	16–29		*30–44*		*45–65*		*All ages*	
Ethnic group	*n*	*Rate*	*n*	*Rate*	*n*	*Rate*	*n*	*Rate*
White	12	3.7	18	5.6	7	1.6	37	3.4
West Indian migrant	11	90.9	7	38.3	5	19.6	23	41.1
British West Indian	4	16.4		—		—	4	15.2
Asian migrant	1	1.7		—	2	6.2	3	2.2

Weighted risk ratio of West Indian migrant : white = 13.65, with 95% confidence limits 9.0 to 23.2

as 1975 in order to obtain sufficient numbers of Part V detentions. The British West Indian group is a young one, with the 16–29 age group most probably growing each year from 1975. The population figures are therefore almost certainly an over-estimate as far as the early years of the 1975–1982 period. The true British-born rates will therefore be higher.

Comparison between Part V and Part IV detention rates

The West Indian migrants have increased rates for both Part V and Part IV compared to those for whites, but the increase is greater for Part V than for Part IV for all age groups, markedly so for the youngest and oldest groups. The former have a rate 25 times that of young whites for Part V, while the comparable rate for Part IV is 17 times that for whites; the latter have a rate 12 times that of whites for Part V, whereas the comparable Part IV rate is 1.5 times that for whites.

There were no British Asians detained under either Part, and insufficient numbers of migrant Asians and British West Indians detained under Part V to make meaningful comparisons.

TABLE IV

General admission sample (50% sample over 4 years)

		Part IV sections				
	Admissions	*n*	*Rate*[1]	*Part IV section rate per 100 admissions*	*Part V sections*	*Part V section rate per 100 admissions*
White						
16–29	92	13	4.0	14.1	1	
30–44	142	14	4.4	9.9	3	
45–P	186	18	4.7	9.7	2	
Total	420	45	4.3	10.7	6	1.4
West Indian migrant						
16–29	19	6	49.6**	31.5	1	
30–44	22	3	16.3	18.2	1	
45–P	6	2	8.0	16.6	1	
Total	47	11	19.9	23.4	3	6.4*
British West Indian						
16–29	24	6	24.6**	25.0	2	
30–44	1	0				
45–P						
Total	25	6	22.7	25.0	2	
Asian migrant						
16–29	25	2	3.4	8.0	0	
30–44	26	5	11.5	19.2	0	
45–P	14	2	6.5	14.2	0	
Total	65	9	6.8	13.8	0	

1. Part IV section rate was based on a 50% sample, double to obtain the full section rate.

*$P = 0.05$; **$P < 0.001$. Significance levels apply to ethnic minority group *vs* white for similar age groups (differences for total age groups not tested apart from Part V section rate per 100 admissions).

Diagnosis under Part IV and Part V

Schizophrenia was the commonest case note diagnosis under both Parts; 110 Part IV patients (51%) and 52 Part V patients (78%) were diagnosed as schizophrenic.

General admission sample

This sample was obtained to determine whether differences in compulsory detention rates between groups are affected by taking into account the total numbers of admissions and diagnostic differences. Details were recorded from the first admission for each individual in the 4-year period. Table IV shows numbers of admissions, detentions on Part IV and Part IV rates. Part V detentions are given, but Part V rates are not shown because numbers are so small. In order to take account of total number of admissions, the section rates per 100 admissions are calculated for Parts IV and Part V.

Numbers in the British Asian, mixed Asian/white and other groups total 17 and are excluded from the table, being too small for analysis.

Part IV section rates

These rates were calculated from the 50% sample of admissions. The overall pattern of Part IV sections is, as expected, similar to that given in Table III, but, in this sample, the rate refers to the risk of being on a section on one particular admission only. Therefore the differences between the groups are not quite so large and, with the smaller numbers, only the youngest group of West Indian migrants, in addition to the British West Indians, have rates significantly higher than those for whites ($P<0.001$). The rates for Asians are no longer significantly different from those for whites.

Part IV section rate per 100 admissions

Once the total number of admissions in each group is taken into account, differences between the groups diminish. Although there is still a trend for West Indians of both groups to be detained more frequently on sections, the differences are not statistically significant.

Part V detention (offender patients)

These comprised 13% of all compulsory detentions. The fact that numbers are so small should be taken into account in interpreting the analysis.

Part V detentions per 100 admissions

Because of the small number in individual age groups, these are given only for the total group of whites and West Indian migrants. The rate for West Indian migrants was 6.4, compared with 1.4 for whites. Applying Fisher's exact test, the probability of getting a result for the West Indians which exceeds the white by this extent or more is 0.05, i.e. the difference is just significant at the 5% level on a one-tail test.

Diagnosis

The final case note diagnosis for each of the main ethnic groups is given in Table V. The percentage of Asian migrants diagnosed as schizophrenic is slightly higher than the corresponding percentage for whites; otherwise, the two groups have similar diagnostic patterns. There are large differences between the West Indians and the Asian and white groups.

Approximately two-thirds of the West Indian migrants (64.5%) and British West Indians (68%) were diagnosed as schizophrenic, compared with one-third of the whites (32%) and Asians (39%). Thus, the proportion of West Indians with a diagnosis of schizophrenia is twice that of the other two groups. In addition, eight West Indian patients (four from each group) have diagnoses of drug-induced psychosis – a diagnosis not given to either whites or Asians; in each case, the drug implicated was cannabis. A lower proportion of West Indians have diagnoses of affective disorder, alcoholism and neurosis/personality disorder. There is little difference in the diagnoses given to the two West Indian groups.

TABLE V
Distribution of diagnosis in general admission sample

	White		*West Indian migrant*		*British West Indian*		*Asian migrant*	
Diagnosis	*n*	%	*n*	%	*n*	%	*n*	%
Schizophrenia	134	32	30	64.5	17	68	25	39
Paranoid psychosis	9	2	—	—	—	—	—	—
Affective disorder	124	29.5	7	15	2	8	19	29
Neurosis/personality disorder	71	17	3	6	2	8	9	14
Alcohol/drug abuse	49	11.5	—	—	—	—	8	12
Organic brain syndrome	24	6	1	2	—	—	—	—
Drug-induced psychosis	—	—	4	8.5	4	16	—	—
Other	9	2	2	4	—	—	4	6
Total	420		47		25		65	

Two methods were used to assess the effect of diagnosis on compulsory detention. First, each individual from the minority group was matched by age (within 3 years) and diagnosis with a member of the white group. Cannabis psychosis was matched with schizophrenia (one in four cases of cannabis psychosis was detained under Part IV, compared with between one in five and one in six cases of schizophrenia). Apart from age and diagnosis, the matching was random. Part V patients were removed from each group before matching. Because of their small numbers, no corresponding matching was done for these offender patients. Of the 44 matched pairs of whites and West Indian migrants, nine whites and 11 West Indians were on Part IV orders. Of the 23 matched pairs of whites and British West Indians, four of the former and six of the latter were detained on Part IV orders. Although the West Indians were compulsorily detained slightly more often, the differences did not approach statistical significance.

Secondly, the main diagnostic difference between the groups was the excess of West Indian schizophrenics. All patients with this diagnosis were identified and the numbers on Part IV and Part V determined. Because of the small numbers, the groups were split into two age groups only (16–34 and 35–54). In the youngest age group (16–34), 13 out of 55 white schizophrenics (24%) were on Part IV, compared with five out of 22 West Indian migrants (23%) and five out of 16 British West Indians (31%). In the older age group (35–54), seven out of 59 white schizophrenics (12%) were on Part IV, compared with one out of ten West Indian migrants (10%). None of these differences approached statistical significance. The numbers of offender patients were so small that they could only be compared for the total age groups. Two out of 114 white schizophrenics (2%) were detained under Part V, compared with three out of 32 West Indian migrants (9%). Applying Fisher's exact test, the probability of getting such a result or higher is 0.07 on a *one-tail test*.

Because there were no white patients detained under Part V in the 16–34 age group, no comparison could be made with the British West Indians, all of whom were aged under 34 years.

Discussion

Our study differs from comparable studies in two ways. First, special efforts were made to gather a sample of offender (Part V) as well as non-offender (Part IV) patients and, secondly, British-born children of migrants were analysed separately. We limited the study to males because observations suggested that the hypotheses were less applicable to females. In this discussion, we shall consider first the results in relation to the main hypotheses.

The results broadly confirmed that West Indian males, both migrant and British, had high compulsory admission rates compared to those for whites. In accordance with the hypothesis, the excess under Part V was considerably higher than under Part IV for West Indian migrants. Contrary to the hypothesis, the Part IV differences were accounted for by differences in total number of admissions and diagnosis; the differences were greater in the younger age groups. It seems unlikely that the Part V differences can be explained in the same way. The third hypothesis predicted no differences in detention rates between migrant and British West Indians. Differences were found, but these did not reach statistical significance. Overall, results for Asians were similar to those for whites, as predicted.

Several aspects merit further discussion. The Part IV results suggest that the high rate of West Indian detentions can be explained by the excess of admissions with schizophrenia. This differs from previous work (Littlewood & Lipsedge, 1981a), which found excessive West Indian detentions to be independent of diagnosis. Ineichen *et al* (1984) suggested there was no excess of voluntary admissions among West Indians in their sample; however, the comparison group comprised white inner city residents, and no account was taken of differences in age structure between the two populations. No reference was made to diagnosis.

A pattern of high admission and first admission rates for West Indian migrants and a preponderance of schizophrenia has often been noted (Cochrane, 1977; Rwegellera, 1977; Carpenter & Brockington, 1980; Dean *et al*, 1981). There has been much debate about the nature of the illness – is it true schizophrenia (Rwegellera, 1977), paranoid psychosis (Carpenter & Brockington, 1980), an acute psychotic reaction (Littlewood & Lipsedge, 1981b) or a neurotic condition (*British Medical Journal*, 1980)? These different interpretations of the illness cast doubt on the validity of hospital diagnosis. Since our study relied on case note diagnosis, our data are subject to these uncertainties. The diagnosis of cannabis psychosis occurred uniquely in the West Indian groups; we included it as a separate entity only after noting its frequent occurrence. There has been debate about its validity (Carney & Lipsedge, 1984), but the fact that it is being made so often on West Indian males makes the phenomenon worthy of further study.

The results revealed large differences between whites and West Indians. However, no significant differences were found between British West Indians and migrants for Part IV detention rates. Both had a high number of admissions and a similar pattern of diagnoses. Further study of these groups may help clarify the problem of mental illness in immigrants. Only one of these groups has experienced a pre-migration culture as well as migration, albeit several years earlier than the period of study. However, both groups may share cultural interests and values, as well as the experience of social deprivation and racial antagonism. There may also be genetic similarities. Some of these factors may be important aetiologically, while others may be pathoplastic.

Although it is unclear how much they differ, there is every indication that both groups have a high morbidity for major psychiatric illness, particularly the youngest members. There would appear to be no support for Cochrane's (1983) hypothesis that "The deliberate creation of a counter culture among West Indian youths . . . may provide some protection from psychological problems".

The last part of the discussion will concentrate on the offender patients. The main difference between Part IV and V patients is that the latter are offenders: the West Indian excess under Part V may merely reflect excessive involvement in crime and the 1975 arrest rates by the London Metropolitan Police (Stevens & Willis, 1979) provide some evidence for this. Blacks had disproportionately high rates for all crime, but arrest rates do not necessarily correlate with rates of criminality or conviction; they may reflect police attitudes or an increase in 'arrest-prone groups'. In addition, these authors emphasise that measures of social disadvantage are crucial in determining crime rates, and these vary between ethnic groups.

There are other possible explanations for the difference. First, once in custody, West Indians may be more prone to developing mental illness than whites; we know of no evidence either for or against this. Another possible explanation of our finding is that the manifestation of mental illness in West Indians leads to arrest and criminal charges, rather than referral to a mental hospital. There are certainly reports in the literature of West Indians presenting a more disturbed or violent picture for psychotic illness in general (Hitch & Clegg, 1980; Rwegellera, 1980; Harrison *et al*, 1984), acute psychotic reaction (Littlewood & Lipsedge, 1981*b*), hypomania (Rack, 1982) and cannabis psychosis (Knight, 1976). This may lead to early police involvement, particularly if the disturbance occurs in a public place. (It is important to remember that there are many possible explanations for any increase in disturbed behaviour; one is that it is a result of a poor relationship between psychiatric and other agencies and West Indians.) Whilst it is known that police referrals to psychiatric hospitals include an excess of West Indian migrants (Sims & Symonds, 1975), it is not known how many are placed 'inappropriately' in the penal system. Evidence from the US penal system (Scheiffer *et al*, 1968) suggested that blacks needed to be more obviously ill before the police referred them to psychiatric facilities; if the illness was less overt, they were more likely than whites to be dealt with in the penal system. There has been no similar work in the UK, but such a phenomenon may contribute to our results.

These differences between the Part V and Part IV results have implications for forensic psychiatrists and merit further study. To obtain more Part V patients, it would be necessary to include more hospitals in the study, including Special Hospitals. In an attempt to answer some of the questions raised by this paper, we have recently completed a study of first admissions to the same psychiatric hospital and plan a further study in a local prison to determine psychiatric morbidity of members of ethnic minorities in custody.

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References

ARMITAGE, P. (1980) *Statistical Methods in Medical Research*. Oxford: Blackwell Scientific Publications.

BIRMINGHAM CENTRAL STATISTICAL OFFICE (1984) Report no. 24.

BRITISH MEDICAL JOURNAL (1980) Editorial. *British Medical Journal*, **281**, 1513.

CARNEY, P. & LIPSEDGE, M. (1984) Psychosis after cannabis abuse. *British Medical Journal*, **288**, 1381.

CARPENTER, L. & BROCKINGTON, I. F. (1980) A study of mental illness in Asians, West Indians and Africans living in Manchester. *British Journal of Psychiatry*, **137**, 201–205.

COCHRANE, R. (1977) Mental illness in immigrants in England and Wales: An analysis of mental hospital admissions, 1971. *Social Psychiatry*, **12**, 25–35.

—— (1983) *The Social Creation of Mental Illness*. London: Longman.

DEAN, G., WALSH, D., DOWNING, H. & SHELLEY, E. (1981) First admissions of native-born and immigrants to psychiatric hospitals in South-East England 1976. *British Journal of Psychiatry*, **139**, 506–512.

DEPARTMENT OF THE ENVIRONMENT (1979) *National Dwelling and Housing Survey*. London: HMSO.

HARRISON, G., INEICHEN, B., SMITH, J. & MORGAN, H. G. (1984) Psychiatric hospital admission in Bristol. II Social and clinical aspects of compulsory admission. *British Journal of Psychiatry*, **145**, 605–611.

HITCH, P. J. & CLEGG, P. (1980) Modes of referral of overseas immigrant and native born first admissions to psychiatric hospital. *Social Science and Medicine*, **14**, 369–374.

INEICHEN, B., HARRISON, G. & MORGAN, H. G. (1984) Psychiatric hospital admissions in Bristol. I. Geographical and ethnic factors. *British Journal of Psychiatry*, **145**, 600–604.

KNIGHT, F. (1976) Role of cannabis in psychiatric disturbance. *Annals of the New York Academy of Sciences*, **282**, 64–71.

LITTLEWOOD, R. & LIPSEDGE, M. (1977) Compulsary hospitalisation and minority status. *5th Biennial Meeting of the Caribbean Psychiatric Association*.

—— & —— (1981*a*) Some social and psychological characteristics of psychotic immigrants. *Psychological Medicine*, 11, 289–302.

—— & —— (1981*b*) Acute psychotic reactions in Caribbean born patients. *Psychological Medicine*, 11, 303–318.

Norris, M. (1984) *Integration of Special Hospital Patients into the Community*. Aldershot: Gower.

Office Of Population Censuses And Surveys (1982*a*) *Census 1981, County Reports, West Midlands*. London: HMSO.

—— (1982*b*) *Labour Force Survey 1981*. London: HMSO.

Pinto, R. T. (1970) A study of psychiatric illness among Asians in the Camberwell area. MPhil Thesis, University of London.

Rack, P. (1982) *Race, Culture and Mental Disorder*. London: Tavistock.

Rwegellera, G. G. C. (1970) Mental illness in Africans and West Indians of African origin living in London. MPhil Thesis, University of London.

—— (1977) Psychiatric morbidity among West Africans and West Indians living in London. *Psychological Medicine*, 7, 317–329.

—— (1980) Differential use of psychiatric services by West Indians, West Africans and English in London. *British Journal of Psychiatry*, 137, 428–432.

Scheiffer, C., Derbyshire, L. & Martin, J. (1968) Clinical change in jail referred mental patients. *Archives of General Psychiatry*, 18, 42–46.

Sims, A. C. P. & Symonds, R. L. (1975) Psychiatric referrals from the police. *British Journal of Psychiatry*, 127, 171–178.

Stevens, P. & Willis, C. (1979) *Race, Crime and Arrests*, Home Office Research Study No. 58. London: HMSO.

Szmukler, G. I., Bird, A. S. & Button, E. J. (1981) Compulsory admissions in a London borough: I. Social and clinical features and a follow-up. *Psychological Medicine*, 11, 617–636.

Walker, N. & McCabe, S. (1973) *Crime and Insanity in England*, Vol II. Edinburgh: Edinburgh University Press.

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Anticonvulsant Medication in a Mental Handicap Hospital: 1972–1982

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The prescription sheets for all patients in a mental handicap hospital on one day in 1972 and on the same date in 1982 were examined to identify trends in anticonvulsant medication over a 10-year period. There was a reduction in size of the hospital population associated with fewer very young and milder handicapped persons. Prescription of anticonvulsants fell slightly during the decade under study with a dramatic fall in the use of phenobarbitone, and a parallel increase in the prescription of carbamazepine. Other changes included the use of sodium valproate in a significant minority of patients and the occasional use of clonazepam in 1982, while phenytoin remained a first-rank anticonvulsant throughout the period under review. The proportions of patients on polytherapy did not change over the 10-year period, though the contribution of phenobarbitone to the combined drug regimes was significantly reduced.

Epilepsy is common in mentally handicapped patients, especially the severely retarded and those who are institutionalised (Corbett, 1981). Hence anticonvulsant drugs are widely used in hospitals for the mentally handicapped. There is evidence that the latter are specially prone to become intoxicated with anticonvulsant drugs and may show side-effects, such as intellectual deterioration and psychomotor slowing with serum levels within the accepted optimum range (Reynolds & Travers, 1974: Trimble & Corbett, 1980). The probability of such complications is increased by polytherapy – the combined use of two, three or more anticonvulsants (Reynolds, 1975). Yet, surveys of anticonvulsant prescribing practices in mental handicap hospitals are scarce and most have surveyed drugs prescribed on a single day only (Spencer, 1974; Kirman, 1975; Hughes, 1977). Even fewer studies of prescribing changes over time have been carried out. James (1983) studied changes over a 9-month period following the introduction of British National Formulary prescribing guidelines and Gowdey *et al* (1984) investigated drug prescribing between 1977 and 1982 in a Canadian residential mental retardation centre.

The present investigation reviews anticonvulsant prescribing changes in a mental handicap hospital over a rather longer 10-year period, 1972–1982, a decade that witnessed significant advances in anti-epileptic drug therapy, a greater understanding of their clinical pharmacology, the development of effective serum level monitoring methods and a recognition of the therapeutic benefits of monotherapy and adverse consequences of polytherapy. In 1982, estimation of serum anticonvulsant drug levels had become routine; a decade before, such serum monitoring was not customary.

Method

The Drug Monitoring Unit at the Royal Dundee Liff Hospital was set up in November, 1969 to monitor drug prescribing and adverse drug reactions. A continuous record of all prescriptions for in-patients in the Dundee Psychiatric Service and the Tayside Region Mental Handicap Service is kept. The drug prescription sheets for all patients in Strathmartine Hospital on the following two dates were scrutinised: 31 December 1972 and 31 December 1982. Strathmartine Hospital accommodates all mentally handicapped patients within Tayside who need in-patient care.

The following data was extracted from each prescription sheet and recorded on a specially prepared data sheet: patient's name, ward, sex, date of birth, consultant's name and all drugs prescribed on the date of interest. For each drug the following information was obtained: date of commencement of the prescription, total daily dose, route of administration and whether the prescription was 'as required' or to be administered at regular intervals throughout the 24 hours.

Patients discharged on the day of interest were not included. Those on 'leave' from the hospital were. 'As required' or 'once only' prescriptions were only included if the drug was actually given to the patient on the day of interest.

The date of admission and diagnosis for each patient identified as receiving medication on the two days of interest were recorded from the medical records at Strathmartine Hospital. The severity of mental handicap was graded according to the Ninth Revision of the International Classification of Diseases. An additional Borderline category was used to describe those patients whose IQ was rated as higher than the Mild range but less than the lower limit of the average IQ range (Reid 1982). Those for whom no specific diagnostic category was available were defined as having 'unspecified mental retardation'.

The numbers of patients receiving anticonvulsants on the two dates of interest were calculated. The frequency of use of the individual drugs and common drug combinations were tabulated and compared for the two dates. The associations between severity of handicap and drug prescribing were also examined. χ^2 Tests were used where appropriate.

Results

Table I shows the total in-patient population changes. The total number of in-patients decreased from 605 in 1972 to 535 in 1982, the sex distribution remaining unchanged. Those under 20 years fell from 158 to 83 ($P<0.001$). A non-significant increase in those over 60 years was observed (50 to 72). No other changes in age distribution were apparent. There were significantly fewer patients with borderline handicap in 1982 (155 in 1972, 109 in 1982, $P<0.02$). Although there were fewer severely and profoundly retarded people in 1982 (213 as compared to 225 in 1972), they represented a slightly higher proportion of the population (37% in 1972, 40% in 1982). However, these differences were not statistically significant.

The main features of those receiving anticonvulsant medication are displayed in Table II. The total number receiving anticonvulsant medication was 179 (30%) in 1972 and 130 (24%), this decrease being significant at the 5% level. The sex distribution was similar to the in-patient population as a whole with an excess of males and did not change significantly over time. The anticonvulsant drug prescription rates increased with severity of the mental retardation. The numbers of each category on anticonvulsants are less in 1982 but this change is not statistically significant.

TABLE I

Total in-patient population changes

	1972	*1982*
Number of patients	605	535
Proportion of males	55%	58%
***Number under 20 years	158 (26%)	83 (15%)
Number over 60 years	50 (8%)	72 (13%)
Degree of handicap		
Borderline MR	29 (5%)	9 (2%)
Mild MR	126 (21%)	100 (19%)
Moderate MR	203 (34%)	208 (39%)
Severe MR	145 (24%)	137 (26%)
Profound MR	80 (13%)	76 (14%)
Unspecified MR	22 (4%)	5 (1%)

Significant differences between 1972 and 1982: $***P<0.001$.

TABLE II

Anticonvulsant prescribing changes

	1972	*1982*
*Number of patients on anticonvulsants (% total population)	179 (30%)	130 (24%)
Sex ratio		
Male	102 (57%)	77 (59%)
Female	77 (43%)	53 (41%)
Diagnoses		
Borderline MR	3 (2%)	1 (1%)
Mild MR	29 (16%)	18 (14%)
Moderate MR	56 (31%)	38 (29%)
Severe MR	47 (26%)	39 (30%)
Profound MR	40 (22%)	33 (25%)
Unspecified MR	4 (2%)	1 (1%)
Percentage of each diagnostic subgroup receiving anticonvulsants		
Borderline MR	10%	11%
Mild MR	23%	18%
Moderate MR	28%	19%
Severe MR	32%	28%
Profound MR	50%	44%
Unspecified MR	18%	20%

*Significant difference between 1972 and 1982: $P<0.05$.

TABLE III

Number of patients receiving each drug alone or in combination

Anticonvulsant	*1972*	*1982*
***Phenobarbitone	160 (89%)	63 (48%)
**Primidone	24 (13%)	6 (5%)
Phenytoin	60 (34%)	65 (50%)
***Carbamazepine	2 (1%)	52 (40%)
Sodium Valproate	—	22 (17%)
Clonazepam	—	6 (5%)
Ethosuximide	5 (3%)	1 (1%)
Sulthiame	9 (5%)	—
Beclamide	1 (<1%)	—
Troxidone	1 (<1%)	—
Paraldehyde	5 (3%)	—

Significant differences between 1972 and 1982: $***P<0.001$, $**P<0.01$.

Table III illustrates the prescribing changes between 1972 and 1982. These are as follows:

(a) A significant reduction in the number of people prescribed phenobarbitone (from 160 to 63: $\chi^2=39.5$; d.f. $=1$, $P<0.001$) and primidone (from 24 to 6: $\chi^2=9.6$; d.f. $=1$, $P<0.01$).

(b) A significant increase in the number of patients prescribed carbamazepine (from 2 to 52: $\chi^2=57.0$; d.f. = 1, $P<0.001$).
(c) An increase in the number of people prescribed phenytoin (from 60 to 65), although this was not statistically significant at the 0.05 level.
(d) The introduction of sodium valproate and clonazepam as anticonvulsants.
(e) Ethosuximide is rarely prescribed and its use is declining. Sulthiame, Beclamide, Troxidone and Paraldehyde, already unpopular in 1972, are not prescribed in 1982.

TABLE IV
Monotherapy and polytherapy changes

	1972	1982
Number of patients on one or more anticonvulsants		
One only	102 (57%)	68 (52%)
Two	66 (37%)	44 (34%)
Three	10 (6%)	13 (10%)
Four	1 (<1%)	5 (4%)
Diagnosis of polytherapy patients (% anticonvulsant subgroup)		
Borderline MR	2 (1%)	1 (1%)
Mild MR	12 (7%)	9 (7%)
Moderate MR	26 (15%)	22 (17%)
Severe MR	20 (11%)	17 (13%)
Profound MR	15 (8%)	12 (9%)
Unspecified MR	2 (1%)	1 (1%)

Tables IV and V show the changes in anticonvulsant polytherapy between 1972 and 1982. There is a slight decrease in number (77 to 62) but increase in proportion of patients prescribed two or more anticonvulsants (43–48%) over the 10-year period. These differences are not significant. Table IV illustrates the number of patients prescribed two or more anticonvulsants within each diagnostic category; around 80% were moderately, severely or profoundly retarded with little change between 1972 (79%) and 1982 (80%). Table V shows changes in certain combined drug regimes. The following significant differences between 1972 and 1982 are apparent:

(a) A significant decrease in the number of patients prescribed phenobarbitone and phenytoin together (from 48 to 21: $\chi^2=4.9$; d.f. = 1, $P<0.02$).
(b) A significant increase in the number of patients prescribed phenytoin and carbamazepine together (from 3 to 12: $\chi^2=10.5$; d.f. = 1, $P<0.001$).
(c) A significant increase in the number of patients prescribed phenytoin, carbamazepine and phenobarbitone together (from 2 to 9: $\chi^2=6.1$; d.f. = 1, $P<0.01$).

TABLE V
Combined medication changes

Anticonvulsant combinations	*1972*	*1982*
*Phenobarbitone and phenytoin	48 (27%)	21 (16%)
***Phenytoin and carbamazepine	3 (2%)	12 (9%)
**Phenytoin, carbamazepine and phenobarbitone	2 (1%)	9 (7%)

Significant differences between 1972 and 1982: ***$P<0.001$, **$P<0.01$, *$P<0.02$.

Discussion

Our anticonvulsant prescribing rates of 30% in 1972 and 24% in 1982 are essentially similar to those reported in previous surveys (Spencer, 1974; Kirman, 1975; Hughes, 1977; Gowdey *et al*, 1984).

If the assumption that most mentally handicapped patients receiving anticonvulsant medication are being treated for recurrent seizures is valid, it can be concluded that around 25% of our in-patients suffer from epilepsy, the number having fallen slightly over the 10-year period under review. These prevalence figures are in agreement with previously published series, as is the association between occurrence of fits and the severity of handicap (Corbett, 1981).

The proportion of severe and profoundly handicapped patients on anticonvulsants tended to increase slightly between 1972 (48%) and 1982 (55%). Hence the decrease in epilepsy prevalence cannot be accounted for by the accumulation of a residual pool of more severely handicapped patients in the hospital. Perhaps a more critical and conservative approach to the diagnosis of epilepsy and the use of anticonvulsant drugs explains the lower prescribing rate in 1982.

Probably the most dramatic change in anticonvulsant prescribing is the decline in use of phenobarbitone (from almost 90% of patients in 1972 to slightly less than 50% in 1982). Doubtless, these changes are a reflection of growing awareness of the adverse effects of phenobarbitone on behaviour and cognitive function (Reynolds & Travers, 1974; Reynolds, 1975; Stores, 1975; Trimble & Reynolds, 1976; Snaith *et al*, 1979). The declining popularity of primidone is almost certainly a consequence of phenobarbitone being one of its major metabolites. It also has acquired a reputation amongst influential clinicians for oversedation in therapeutic doses and for causing occasional psychotic episodes. Phenytoin has maintained its established place as a first-rank anticonvulsant, being used in 33–50% of patients.

The decline in phenobarbitone prescriptions is paralleled by a marked increase in the use of carbamazepine. Though available in 1972, it was rarely used. By 1982, its prescription (40%) was almost as frequent as phenobarbitone (48%) and phenytoin (50%). Over the 10-year period of this study, careful clinical trials have established carbamazepine as a first-rank anticonvulsant drug (Troupin, 1983). Further studies of both healthy subjects and patients have demonstrated that carbamazepine has much less effect on cognitive function than phenobarbitone or phenytoin (Dodrill & Troupin, 1977; Thompson *et al*, 1981; Thompson & Trimble, 1982; Hirtz & Nelson, 1985). The wide discussion of the possibility of a psychotropic action is also an important determinant in its use (Dalby, 1975; Ballenger & Post 1980). Another factor may be the recent local interest in using carbamazepine to control the behaviour of mentally handicapped patients (Reid *et al*, 1981).

The use of sodium valproate in a significant minority of patients in 1981 and not in 1972 is consistent with its relatively late introduction into the UK from the continent and its recognition as an effective drug in secondary generalised epilepsy (Driefuss, 1983). The foothold gained by clonazepam in 1982 is small. Can this be due to the problems of oversedation, irritability and tolerance that occur with this drug? (Browne, 1976; Schmidt, 1985). It is of interest that ethosuximide makes a minimal and decreasing contribution to anticonvulsant medication - presumably a reflection of the paucity of patients with absence seizures due to primary generalised epilepsy in a mental handicap hospital. Finally, the disappearance of sulthiame, beclamide, troxidone and paraldehyde by 1982 reflects current trends in anticonvulsant drug prescribing in epilepsy generally.

Polytherapy, the combination of two or more anticonvulsant drugs, continues to be the prescribing pattern in a substantial minority of patients; slightly less than half, a proportion that has not changed significantly over the 10 years being studied. As with monotherapy, the contribution of phenobarbitone to the drug combinations has reduced significantly (27% to 16%) and that of carbamazepine is rising (2% to 9%). Hence, even though the combinations of drugs used are changing, polytherapy persists. Indeed, the proportion of patients on three or more drugs is increasing (6% to 14%).

Is this worrying trend due to the intractible nature of epilepsy in a significant minority of the mentally handicapped with consequent greater difficulty in achieving seizure control? Certainly, polytherapy is a feature of the medication of the more severely handicapped (around 80% in those with moderate, severe or profound mental handicap). If and when satisfactory control is reached, are clinicians reluctant to withdraw drugs in the mentally handicapped because of the risk of status? Of course, intractible epilepsy in less severely impaired patients is likely to make discharge to community care difficult. This may lead to an ever-growing pool of patients with severe epilepsy who can only be managed by polytherapy. A further research project has been initiated in order to identify the characteristic features of the polytherapy patients and to investigate the questions raised in this paragraph.

A final question that requires answering is the extent to which polytherapy contributes to cognitive impairment and behaviour disorder in the mentally handicapped. Neuroleptic medication has been shown to affect adversely the daily living skills and workshop performance of mentally handicapped patients (Breuning *et al*, 1982; Heistad *et al*, 1982). Does polytherapy cause similar problems?

It should be emphasised that our data relate to a highly selected population of mentally handicapped patients due to their requiring institutional care. Therefore, the findings cannot be generalised to the anti-epileptic treatment of patients living in the community. Indeed, it would be of interest to study a community sample.

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References

BALLENGER, J. C. & POST, R. M. (1980) Carbamazepine (Tegretol) in manic-depressive illness: a new treatment. *American Journal of Psychiatry*, 137, 782-790.

BREUNING, S. E., DAVIS, V. J., MATSON, J. L. & FERGUSON, D. G. (1982) Effects of thioridazine and withdrawal dyskinesias on workshop performance of mentally retarded young adults. *American Journal of Psychiatry*, 139, 1447-1454.

BROWNE, T. R. (1976) Clonazepam — a review of a new anticonvulsant drug. *Archives of Neurology*, 33, 326-332.

CORBETT, J. (1981) Epilepsy and mental retardation. In *Epilepsy and Psychiatry* (eds E. R. Reynolds & M. R. Trimble). Edinburgh: Churchill Livingstone.

DALBY, M. A. (1975) Behavioural effects of carbamazepine. In *Advances in Neurology* (eds. J. K. Penry & D. D. Daly). New York: Raven Press.

DODRILL, C. B. & TROUPIN, A. S. (1977) Psychotropic effects of carbamazepine in epilepsy: a double blind comparison with phenytoin. *Neurology*, 27, 1023-1028.

DRIEFUSS, F. E. (1983) Sodium Valproate: a reappraisal. In *Recent Advances in Epilepsy* (eds T. A. Pedley & B. S. Meldrum). London: Churchill Livingstone.

GOWDEY, C. W., COLEMAN, L. M. & CRAWFORD, E. M. H. (1984) Survey of anticonvulsant and neuroleptic drug use on a residential mental retardation center in Ontario. In *Perspectives and Progress in Mental Retardation Vol. II—Biomedical Aspects* (ed. J. M. Berg). Baltimore: University Park Press.

HEISTAD, G. T., ZIMMERMANN, R. L. & DOEBLER, M. I. (1982) Long term usefulness of thioridazine for institutionalised mentally retarded patients. *American Journal of Mental Deficiency*, **87**, 243–251.

HIRTZ, D. G. & NELSON, K. B. (1985) Cognitive effects of anticonvulsant drugs. In *Recent Advances in Epilepsy* (eds T. A. Pedley & B. S. Meldrum). Edinburgh: Churchill Livingstone.

HUGHES, P. S. (1977) Survey of medication in a subnormality hospital. *British Journal of Mental Subnormality*, **13**, 88–94.

JAMES, D. H. (1983) Monitoring drugs in hospitals for the mentally handicapped. *British Journal of Psychiatry*, **142**, 163–165.

KIRMAN, B. (1975) Drug therapy in mental handicap. *British Journal of Psychiatry*, **127**, 545–549.

REID, A. H. (1982) *Psychiatry of Mental Handicap*. Oxford: Blackwell.

——, NAYLOR, G. J. & KAY, D. S. G. (1981) A double blind placebo controlled crossover trial of carbamazepine in overactive severely mentally handicapped patients. *Psychological Medicine*, **11**, 109–113.

REYNOLDS, E. H. (1975) Chronic antiepileptic toxicity: a review. *Epilepsia*, **16**, 319–332.

—— & TRAVERS, R. D. (1974) Serum anticonvulsant concentrations in epileptic patients with mental symptoms. *British Journal of Psychiatry*, **124**, 440–445.

SCHMIDT, D. (1985) Benzodiazepines—an update. In *Recent Advances in Epilepsy 2* (eds T. A. Pedley & B. S. Meldrum). Edinburgh: Churchill Livingstone.

SNAITH, R. P., JAMES, F. E. & WINOKUR, B. (1979) The drug treatment of mental illness and epilepsy in the mentally handicapped patient. In *Psychiatric Illness and Mental Handicap* (eds F. E. James & R. P. Snaith). London: Gaskell.

SPENCER, D. A. (1974) A survey of the medication in a hospital for the mentally handicapped. *British Journal of Psychiatry*, **124**, 507–508.

STORES, G. (1975) Behavioural effects of anti-epileptic drugs. *Developmental Medicine and Child Neurology*, **17**, 647–658.

THOMPSON, P. J. & TRIMBLE, M. R. (1982) Anticonvulsant drugs and cognitive functions. *Epilepsia*, **23**, 531–534.

THOMPSON, P., MUPPERT, F. A. & TRIMBLE, M. (1981) Phenytoin and cognitive function: effects on normal volunteers and implications for epilepsy. *British Journal of Clinical Psychology*, **20**, 155–162.

TRIMBLE, M. & CORBETT, J. A. (1980) Behaviour and cognitive disturbances in epileptic children. *Irish Medical Journal Supplement*, **73**, 21–28.

TRIMBLE, M. R. & REYNOLDS, E. H. (1976) Anticonvulsant drugs and mental symptoms: a review. *Psychological Medicine*, **6**, 169–178.

TROUPIN, A. S. (1983) Carbamazepine re-examined. In *Recent Advances in Epilepsy 1* (eds T. A. Pedley & B. S. Meldrum). London: Churchill Livingstone.

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Behavioural Treatment of Life-threatening Masochistic Asphyxiation: A Case Study

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Asphyxia as a means of heightening sexual pleasure unfortunately comes to notice more often in the coroner's court than in the psychiatrist's consulting room. The victims are usually adolescent men who strangle or hang themselves while masturbating; transvestism is frequently associated with this behaviour (Simpson, 1979; Friedmann & Faguet, 1982; Mason, 1983). Few such people ask for help, and behavioural treatment in such a case has not been previously described.

Case report

A married man aged 35, with two young children, sought help when his wife discovered his deviant practice and he realised that he had come near to death on several occasions. He was trying to suffocate himself thrice weekly.

The patient's father was an aggressive man, possibly schizophrenic, who had received prolonged psychiatric treatment, including a leucotomy. His mother was very close to the patient emotionally during his early years and adolescence. He had a sister several years his senior. Early childhood was unremarkable.

His first recollection of any unusual sexual interest was of a vivid dream at age 13 that he was being swallowed and thus constricted by a snake, leading to intense sexual arousal and orgasm. He sought to reproduce this sensation by dressing in his mother's corsetry and masturbating, and later used a rubber diving suit to this end. He found that by holding his breath he could increase the intensity of orgasm and this led to his covering his face with a plastic bag.

The deviant behaviour became more complex in stages until he was routinely dressing in tight, women's clothing, securing a plastic bag over his head with a stocking and then binding his wrists in previously prepared slip knots in a length of rope so that he was not able to remove the plastic bag easily. He then achieved orgasm at an intensity far greater than with sexual intercourse, the excitement being increased by the risk that he might not be able to release his hands from the rope in order to remove the bag from his head. Indeed, on a few occasions he almost lost consciousness before succeeding in removing it.

Marital coitus occurred about twice a month, was felt to be unsatisfactory by both partners and was usually accompanied by deviant fantasies. In contrast, coitus with a mistress was frequent, satisfying and unaccompanied by deviant fantasies.

Treatment

We aimed first to reduce the patient's deviant urges and practices to such a level that he was able to resist his urges to act out the fantasy, and secondly, to improve his level of heterosexual functioning. The patient was admitted with his wife to a family flat on the ward, staying there for 6 weeks.

To reduce the deviant fantasy, two self-regulatory techniques were employed: covert sensitisation and the use of coping strategies. The patient's wife was closely involved throughout to encourage the patient and to help monitor his progress. The patient terminated the relationship with his mistress so that he could concentrate on bettering his relationship with his wife, which was good but needed some improvement.

Covert sensitisation

The patient was instructed, over 40 sessions, first in the presence of the therapist, then alone, to imagine carrying out his deviant routine. When he had a clear and strong image of the deviant fantasy, he was asked to produce an aversive fantasy which, with repeated practice, became linked with the deviant one and reduced its accompanying intensity of arousal. Several aversive fantasies were used, e.g. discovery by his children or by his boss, or of being attacked by wasps (the patient was phobic of wasps).

Coping strategies

These were taught to prevent his getting into tempting situations and later, as self-control improved, to enable him to disrupt any deviant behaviour. The first strategy was based on the fact that he stayed up late after his wife had gone to bed and then frequently masturbated with bondage and suffocation. He was encouraged to go to bed at the same time as his wife, who was encouraged to insist that he did so. Secondly, the in-patient nursing staff arranged that there would always be someone on duty familiar with the patient and prepared to talk to him whenever he felt an urge to engage in deviant behaviour. Such contact enabled him to resist carrying it out. Thirdly, a cassette tape recording was made of the voices of nurses and doctors encouraging him to resist his deviant urges and to phone the ward if they occurred when he was outside hospital. Fourthly, heterosexual functioning was improved by sexual skills training of the patient and his wife, together with marital counselling and the use of sexually educational videotapes.

After discharge from in-patient treatment, the patient maintained weekly contact by phone and was seen at decreasing intervals lengthening from weeks to 6 months. He remained greatly improved, both in his heterosexual relationship with his wife and in the reduction of his deviant fantasies and activities.

After 7 months, he suddenly developed strong urges to dress in, and suffocate himself with, a plastic raincoat. This occurred after he had watched a young woman dress in one while he was on holiday abroad and was unable to telephone the ward. He found watching this act intensely erotic, immediately bought such a garment for himself, and then

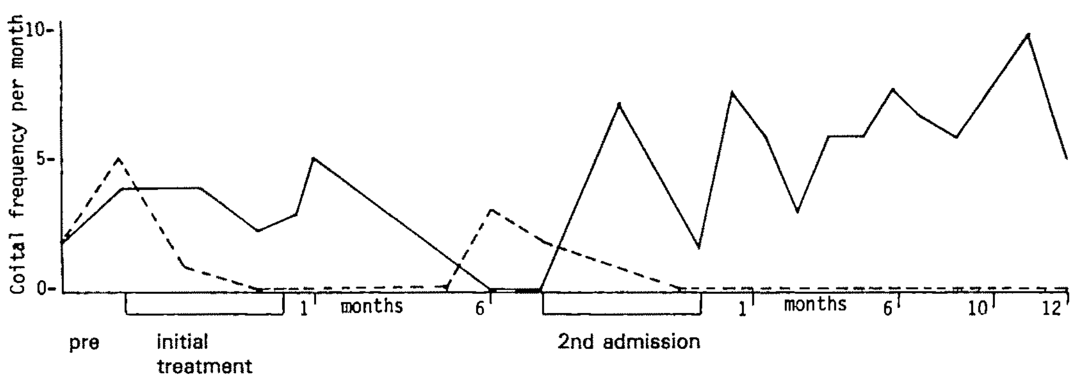

FIG. 1 The effect of behavioural treatment on frequency of coitus with (----) and without (——) deviant fantasies.

wore it, once only, back to front in a constricting manner while masturbating.

Readmission for 4 weeks was arranged for booster therapy. The patient had 45 more sessions along the lines described above and improved quickly. The swallowing fantasy disappeared for the first time. Thereafter the patient was followed up at rapidly widening intervals (6 months at the time of writing). During this time, the patient remained very well and described his marital relationship as the best it had ever been.

Outcome

As treatment progressed, the patient's attitude to his wife and to normal sex, which had been positive initially, became even more so, while his attitudes to deviant concepts became more negative. His urges to engage in deviant behaviour decreased, and the frequency of deviant fantasies accompanying masturbation diminished. The fantasy of plastic bags decreased more rapidly than that of being swallowed by a woman.

The frequency of sexual intercourse with his wife increased from once every 2 weeks prior to treatment to two or three times per week at follow-up, and orgasm during marital coitus ceased to be accompanied by deviant fantasies (see Fig. 1). Concomitantly, masturbation to deviant fantasties decreased from three times daily at pretreatment to less than once per month at follow-up.

Discussion

The patient had a combination of transvestism and masochism which is not infrequent. Of a series of transvestites, 15% had masochistic interests, and a liking of tight garments was common (Prince & Bentler, 1972).

Transvestites with features of fetishism have been treated with some success by electric aversion during fantasied and real cross-dressing, as assessed after 2-year follow-up (Marks & Gelder, 1967; Marks *et al*, 1970). However, the use of aversion in fantasy (covert sensitisation), rather than external electrical aversion, has certain advantages. It needs no special apparatus and is easier to self-administer. Electrical aversion, even if self-administered, may be seen by some patients and therapists as punishment and manipulation, while aversion in fantasy is seen more easily as a form of self-regulation. The covert sensitisation was used together with other self-regulatory methods to disrupt tempting urges, e.g. phoning the ward after discharge when the urges grew in strength, upon which the nursing staff would 'talk him down' until he felt he could cope. On such occasions he was also encouraged to talk with his wife, but only became able to do so after 1 year, even though she was extremely cooperative.

The patient's improvement requires a continuing vigilance and self-control. At 2½-year follow-up deviant urges were infrequent and could be controlled.

References

FRIEDMANN, C. & FAGUET, R. A. (1982) *Extraordinary Disorders of Human Behaviour*. London: Plenum.

MARKS, I. M. & GELDER, M. G. (1967) Transvestism and fetishism: clinical and psychological changes during faradic aversion. *British Journal of Psychiatry*, 113, 711–729.

——, —— & BANCROFT, J. (1970) Sexual deviants two years after electrical aversion. *British Journal of Psychiatry*, 117, 173–185.

MASON, J. M. (1983) *Forensic Medicine for Lawyers*. London: Butterworth.

PRINCE, V. & BENTLER, P. M. (1972) Survey of 504 cases of fetishism. *Psychological Reports*, 31, 903–917.

SIMPSON, K. (1979) *Forensic Medicine* 8th ed. London: Edward Arnold.

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Classification of Parasuicide by Cluster Analysis

Types of Suicidal Behaviour, Therapeutic and Prognostic Implications

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A classification of suicidal behaviour was derived by cluster analysis from descriptive data on parasuicide patients. The grouping was meaningful in terms of clinical interpretation and had both therapeutic and prognostic implications. The sample was broken down into three groups, identified as repeated, serious and non-serious attempts. The repeated attempters had the worst prognosis at 1-year follow-up. When the sample was classified into five groups according to seriousness of the attempts, the repeater and non-serious groups were retained, while the serious attempts were split into three subtypes, which differed on nosological characteristics, treatment disposal and outcome. An excessively high successful suicide rate was found in a small group of elderly attempters.

In many investigations, attempted suicide has been treated as a uniform entity; in particular much of the research into aetiology and prediction of self-destructive behaviour has set out from this premise. On the other hand, efforts have been made to distinguish separate behavioural patterns within attempted suicide. Classifications have been proposed on the dimensions of suicidal intent (Dorpat & Boswell, 1963; Silver *et al*, 1971; Beck *et al*, 1976), seriousness of the act (Schmidt *et al*, 1954; McHugh & Goodell, 1971; Pallis & Birtchnell, 1977; Goldney, 1981) and repetitiveness (Batchelor, 1954; Buglass & Horton, 1974). A more complex typology was suggested by Wold (1971) and by Litman (1972). Clustering techniques have been applied to the classification of suicidal behaviour by only a few investigators (Kiev, 1976; Henderson *et al*, 1977; Paykel & Rassaby, 1978; Katschnig & Fuchs-Robetin, 1984). In the present report, 493 cases of parasuicide were grouped by cluster analysis and the nosological characteristics and pattern of treatment disposal examined in each group. The prognostic value of the classification was assessed by the rates of repeated suicide attempts and of successful suicides, after a follow-up period of 1 year.

Method

Subjects

The sample comprised 485 patients, consecutively admitted to the poisoning treatment unit of a large university hospital in Munich. They fulfilled the criteria of parasuicide (Kreitman *et al*, 1969). Eight cases of drug experimentation, in whom the diagnosis of parasuicide was equivocal, were also included. The patients were representative of suicide attempters in Western European cities: a detailed description has been published elsewhere (Kurz *et al*, 1985).

Collection of data

Using a pre-coded data recording form, information was gathered on demographic characteristics, psychiatric history, pre-suicidal situation, circumstances of the suicide attempt and psychiatric diagnosis. Severity of intoxication was rated in accordance with the classification proposed by Reed *et al* (1952). A severe intoxication was defined by stages IV and V of this scheme or, where the operationalisation did not apply (e.g. in alkyl phosphate poisoning), was specified by high probability of lethal outcome without medical intervention. The Inpatient Multidimensional Psychiatric Scale (Lorr & Klett, 1967) was used for psychopathological evaluation. Psychiatric diagnoses refer to the German version of the International Classification of Diseases (ICD-9, Degkwitz *et al*, 1980). A personal follow-up investigation was performed 1 year after the key admission. Information on repeated suicide attempts and on completed suicides was obtained from 421 patients (85% of the sample).

Therapeutic management

Following initial psychiatric assessment, immediate crisis intervention was offered where appropriate. In addition to this treatment, outpatient psychotherapy or psychological counselling was considered necessary for most patients. Roughly one-fifth of the cases were referred for psychiatric hospital care; few patients were discharged without a treatment proposal. An account of the therapeutic management has been given by Möller *et al* (1984).

Selection of variables for cluster analysis

The set of input variables was adapted from Paykel & Rassaby (1978): of the original 14 items, ten could be matched on item content and scaling. The variable list covered age and sex, number of previous suicide attempts, frequency of suicidal intent in the past month (rated on a four-point scale), precautions against discovery (rated on a four-point scale), severity of intoxication (rated on a three-point scale), clinician's judgement of interpersonal motivation (e.g. appeal for help, rated on a five-point scale) and clinician's judgement of auto-aggressive motivation (intent-to-die, rated on a five-point scale), depression and hostility at initial assessment (percentage scores of the factors 'anxious intropunitiveness' and 'hostile belligerence' on the Inpatient Multidimensional Psychiatric Scale).

Clustering procedures

The data were analysed with the PKM program of the BMDP statistical software (Dixon, 1983); this uses a clustering technique which establishes mutually exclusive classes (Everitt, 1974). The procedure is composed of a hierarchical (divisive) and an optimising (hill-climbing) method. Starting from the initial set of individuals, the number of clusters is successively increased by the splitting of one group, until the pre-fixed number of clusters is reached; subsequently, cases are reassigned to the groups by iterative reallocation. The sum of squared distances serves as the clustering criterion. For the calculation of distance measures, the raw data were transformed to unit variance. In Tables I and III, the clustering results were retransformed to raw means and percentages. The number of clusters was chosen to be 3, 4 and 5; the three-group solution was selected for cross-validation. The original sample was divided in two subsets, each of which was subjected to the clustering procedure separately.

Discriminant function analysis

A discriminant function analysis was performed on the three-cluster solution to determine the quality of group separation and to assess the discriminant power of each input variable. The P7M program of the BMDP statistical software was used. Three linear classification functions were computed, serving to classify each case into one of the three groups. The percentage of correct allocation was examined by comparison with the original cluster composition.

Results

Three-group classification

Characteristics of the groups (Table I)

Compared to the total patient sample, group A was characterised by low mean age, more males, more previous suicide attempts, more suicidal pre-meditation and more hostility. Group B was distinguished by above-average mean age, more males, more precautions against discovery, more severe intoxications, low interpersonal and high auto-aggressive motivation, high depression and low hostility scores. The outstanding features of group C were low mean age, more females, low percentage of previous suicidal episodes, low rates of recent suicidal intent and of concealment, less severe intoxications, and high interpersonal and

TABLE I

Three-group classification: characteristics of the groups on classification variables

		Clusters					
	All patients n=493	*A n=52*		*B n=203*		*C n=238*	
Variable	*Mean*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*
Age: years	33.2	28.0	10.7	39.5	17.8	29.1	13.8
Number of previous attempts	0.6	3.3	2.1	0.4	0.7	0.2	0.5
Frequency of recent suicidal intent	1.6	2.2	1.0	1.7	0.9	1.4	0.7
Precautions against discovery	1.8	1.7	0.6	2.3	0.6	1.4	0.6
Severity of intoxication	2.1	1.9	0.5	2.3	0.5	1.9	0.4
Interpersonal motivation	3.6	3.4	1.4	2.7	1.3	4.3	0.9
Auto-aggressive motivation	4.1	4.1	0.8	4.7	0.5	3.6	0.9
Depression score	25.9	27.2	10.5	28.6	11.2	23.3	7.6
Hostility score	12.9	22.8	17.7	7.9	10.2	14.9	14.8
Sex: male	39%	46%		47%		31%	
female	61%	54%		53%		69%	

Difference between groups significant at $P<0.001$ on nine variables (analysis of variance), on sex distribution significant at $P<0.01$ (χ^2).

low self-directed motivation. Differences between groups were statistically significant on all variables.

Cross-validation

The cluster triplets obtained from each subset were very similar and consistent with the original clusters; differences between groups were statistically significant on all variables except for sex distribution in both subsets.

Discriminant function analysis

A total of 96% of all cases could be correctly reclassified into the three clusters, which indicates that the groups were well separated. The variables that contributed most to the discrimination were the number of previous parasuicide episodes, the extent of precautions against discovery and the clinician's rating of interpersonal motivation.

Psychiatric diagnosis

Since psychiatric diagnosis was not included in the set of input variables, the nosological characteristics of the clusters were examined separately. A relatively high incidence of personality disorder (31%) was found in cluster A. In cluster B, neurosis, addictive behaviour and reactive disorder were equally frequent, each of these diagnostic categories accounting for one-quarter of the patients. Cluster C was distinguished from the two other groups by a high percentage of reactive disorders (44%).

Treatment disposal

From the above, differences in the therapeutic management of the groups can be suspected. This was confirmed by comparing the major categories of treatment disposal. Admission to psychiatric hospital was frequent in cluster A (44%) and was rare in cluster C (5%).

Follow-up data

To determine the prognostic relevance of the classification, the rates of repeated suicide attempts and of successful suicides within 1 year after key admission were calculated for each group (Table II). Further suicide attempts were three times as frequent in cluster A as in the remaining groups. The suicide rate was roughly 4% in clusters A and B, compared with only 0.4% in group C.

TABLE II

Three-group classification: outcome at 1-year follow-up

	Clusters					
	A		*B*		*C*	
	n	*%*	*n*	*%*	*n*	*%*
Patients who repeated suicide attempts	13	25.0	18	8.9	20	8.4
Patients who committed suicide	2	3.8	8	3.9	1	0.4

Five-group classification

When the number of clusters was increased successively, groups A and C remained fairly stable with their respective characteristics including psychiatric diagnosis, treatment disposal and follow-up data. The clusters which corresponded to A and C on the four- and five-group levels of analysis were not considered. Subdivision occurred in cluster B: in a first step, it was split into two groups, containing 88 and 154 patients, which were difficult to interpret. In a second step, the larger group was subdivided, while the

TABLE III

Five-group classification: major differences between the subgroups of cluster B

	Clusters					
	B1 *n = 27*		*B2* *n = 88*		*B3* *n = 127*	
Variable	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*
Age*	75.4	14.4	32.8	14.0	33.3	10.7
Frequency of recent suicidal intent*	1.5	0.7	2.7	0.8	1.1	0.3
Depression score*	22.6	7.4	37.5	11.2	22.9	7.4
	n	*%*	*n*	*%*	*n*	*%*
Sex: male	10	38	26	30	65	51
female**	17	62	62	70	62	49
Patients who repeated suicide attempts	—	—	9	10.2	16	13.1
Patients who committed suicide	2	7.4	2	2.3	4	3.1

*Differences between groups significant at $P<0.001$ (analysis of variance).

**Differences between groups significant at $P<0.01$ (χ^2).

minor group was retained. Eventually, three different patterns of suicidal behaviour were distinguished (Table III).

Characteristics of the groups

Group B1, the smallest in number, deviated from the remaining groups by a high mean age (75 years). Group B2 was discernible by long-standing suicidal pre-meditation and by a high depression score. Group B3 was distinguished by a sex ratio of 1:1 and by almost complete absence of suicidal pre-meditation. Differences between groups were statistically significant on all variables.

Psychiatric diagnosis

Relatively high rates of organic brain syndrome (21%) and of endogenous depression (14%) were found in group B1. Neurotic disorder was the most frequent diagnosis in group B2 (35%). In group B3, one-third of the patients were given the diagnosis of substance misuse or addiction.

Treatment disposal

In groups B1 and B2, all patients were referred to outpatient or inpatient psychiatric therapy, whereas no further treatment was considered necessary for 9% in group B3. The rate of admission to hospital was high in group B2 (44%) and low in group B3 (9%).

Follow-up data

In group B1, no further suicide attempts occurred, but the suicide rate was high (7.4%). In both other groups, roughly 10% of the patients had repeated parasuicidal acts and 3% had committed suicide.

Discussion

Did the classification make sense from a clinical point of view? Cluster A contained 11% of all patients and showed the known features of parasuicide repeaters (Batchelor, 1954; Kessel & McCulloch, 1966; Buglass & Horton, 1974; Kurz *et al*, 1982). According to the differentiation between serious and non-serious suicide attempts (Schmidt *et al*, 1954; Greer & Lee, 1967;McHugh & Goodell, 1971; Pallis & Birtchnell, 1977; Torhorst *et al*, 1983), cluster B, which comprised 41% of all cases, shared the characteristics of serious acts, while cluster C, which accounted for 48% of the cohort, was identified as a group characterised by non-serious suicidal behaviour.

These results are compatible with previous cluster analysis classifications of suicide attempts. Henderson *et al* (1977) distinguished a group of alienated hostile individuals, whose acts were highly operant, from a depressed alienated group with high life-endangerment, and from a third group, which could not be interpreted clinically. Paykel & Rassaby (1978) found a group taking overdoses, showing little risk to life, little psychiatric disturbance and more evidence of interpersonal rather than self-destructive motivation; a second group made more severe attempts, with more self-destructive motivation; and by violent methods rather than overdose, while a third group had previous histories of many attempts, made relatively mild attempts and were overtly hostile. Katschnig & Fuchs–Robetin (1984) described an 'appeal' group, characterised by low mean age, female sex, first-ever attempts and mild acts motivated by interpersonal conflict. The second group was interpreted as 'failed suicides' and was distinguished by high mean age, hard methods, and motives other than interpersonal friction. The third group – the 'chronic' cluster – was comprised of relatively young repeaters. There is remarkable agreement between the data of Paykel & Rassaby, Katschnig & Fuchs-Robetin and our own findings concerning the main properties of different types of suicidal behaviour, which adds to the evidence that the classification is more than a methodological artifact.

When the original sample of patients was broken down into four and five groups respectively, the clusters interpreted as repeaters and as non-serious attempts were slightly changed in size, but their characteristics were preserved – another point in favour of the consistency of these two clusters.

The prognostic relevance of the classification can be seen from the follow-up data. The cluster of repeaters turned out to be a high-risk group with respect to future attempts and completed suicides, while the cluster of non-serious acts was identified as a low-risk group. The suicide rate was higher in the serious than in the non-serious cluster. Katschnig & Fuchs-Robetin evaluated their classification in a 10-year follow-up study and found similar results. In their sample, no suicide occurred in the 'appeal' group, the suicide rate was 10.7% in the 'failed suicides' and 13.9% in the 'chronic' group; figures on repeated attempts were not reported.

The high rate of further suicidal behaviour in the repeater group is not surprising. There is general agreement in the literature that a history of multiple suicide attempts is associated with an increased risk of repetition and of suicide (Buglass & McCulloch, 1970; Bagley & Greer, 1971; Buglass & Horton, 1974; Morgan *et al*, 1976; Pino *et al*, 1979; Siani *et al*, 1979). In the present sample, however, the 'repeater' group involved less than one-half of the patients who had a history of suicidal behaviour prior to the key attempt; the remaining repeaters were dispersed among the other clusters. From this, it may be hypothesised that there are several subtypes of parasuicide repeaters, as suggested by Bancroft & Marsack (1977).

The difference in prognosis between the serious and non-serious groups requires attention, since discrepant data have been reported on this issue. Whereas Jansson (1962) found no association between the seriousness of suicide attempts and repetition, both Beck *et al* (1976) and Pierce (1981) demonstrated an increased probability of repeated attempts in serious compared to non-serious acts. According to Rosen (1976), patients who made serious attempts were more likely to commit suicide than were non-serious attempters. However, Pino *et al* (1979) failed to confirm this finding. The results of the present study strengthen the assumption that the seriousness of self-destructive behaviour, as judged from ratings of suicidal intent and of life-endangerment, is relevant to prognosis.

The follow-up data of five groups show that the probability of suicide was not evenly distributed within the original group of serious attempts. The elderly patients did not repeat parasuicides, but had a very high suicide rate; this finding is consistent with the fact that the suicide rate increases with age, while the probability of parasuicide is reduced (Kreitman, 1981). As a group, the elderly did not score higher on depression than did the patients who had made non-serious attempts, but this does not preclude an association between suicidal behaviour in old age and depression (Barraclough, 1971; Kockott, 1981). Looking at individual cases, one of the two elderly patients who committed suicide had been given the diagnosis of endogenous depression, and the second was an alcoholic.

The classification of suicide attempts into repeated, serious and non-serious acts, which follows both from previous cluster analysis investigations and from our data, is not novel. Indeed, it is familiar to clinical experience. However, the grouping by mathematical procedures demonstrates that the typology is an objective property of the complex phenomenon of suicidal behaviour, rather than a matter of intuitive judgement. The practical importance of the classification is implied in its relation to outcome: by allocating a patient to a particular behavioural pattern, the level of probability for future suicidal acts is determined, and therapeutic measures can be taken accordingly. Since the classification can be accomplished on a minimum of information, it may be helpful in the management of parasuicide patients, when rapid decisions have to be made. Our data suggest further that the discrimination of smaller and more specific subtypes of suicidal behaviour, such as suicide attempts in old age, may be valuable. More empirical evidence is desirable on this issue.

References

Bagley, C. & Greer, S. (1971) Clinical and social predictors of repeated attempted suicide. *British Journal of Psychiatry*, **119**, 515–521.

Bancroft, J. & Marsack, P. (1977) The repetitiveness of self-poisoning and self-injury. *British Journal of Psychiatry*, **131**, 394–399.

Barraclough, B. M. (1971) Suicide in the elderly. In *Recent Developments in Psychogeriatrics* (eds D. W. K. Kay & A. Walk). Ashford, Kent: Headley Brothers.

Batchelor, I. R. C. (1954) Repeated suicidal attempts. *British Journal of Medical Psychology*, **27**, 158–162.

Beck, A. T., Weissman, A., Lester, D. & Trexler, L. (1976) Classification of suicidal behaviors. *Archives of General Psychiatry*, **33**, 835–837.

Buglass, D. & McCulloch, J. W. (1970) Further suicidal behaviour. The development and validation of predictive scales. *British Journal of Psychiatry*, **116**, 483–491.

—— & Horton, J. (1974) The repetition of parasuicide. A comparison of three cohorts. *British Journal of Psychiatry*, **125**, 168–174.

Degkwitz, R., Helmchen, H., Kockott, G. & Mombour, W. (eds) (1980) *Diagnosenschlüssel und Glossar psychiatrischer Krankheiten. Deutsche Ausgabe der Internationalen Klassifikation der Krankheiten der WHO (ICD). 9th Revision, Kapitel V.* Berlin: Springer.

Dixon, W. J. (1983) *BMDP Statistical Software*. Berkeley: University of California Press.

Dorpat, T. L. & Boswell, J. W. (1963) An evaluation of suicide intent in suicide attempts. *Comprehensive Psychiatry*, **4**, 117–125.

Everitt, B. (1974) *Cluster Analysis*. London: Heinemann.

Goldney, R. D. (1981) Attempted suicide in young women. Correlates of lethality. *British Journal of Psychiatry*, **139**, 382–390.

Greer, S. & Lee, H. A. (1967) Subsequent progress of potentially lethal attempted suicides. *Acta Psychiatrica Scandinavica*, **43**, 361–371.

Henderson, A. S., Hartigan, J., Davidson, J., Lance, G. N., Duncan-Jones, P., Koller, K. M., Ritchie, K., McAuley, H., Williams, C. L. & Slaghuis, W. (1977) A typology of parasuicide. *British Journal of Psychiatry*, **131**, 631–641.

Jansson, B. (1962) A catamnestic study of 476 attempted suicides, with special regard to the prognosis of cases of drug automatism. *Acta Psychiatrica Scandinavica*, **38**, 183–198.

Katschnig, H. & Fuchs-Robetin, G. (1984) Prognose nach Suizidversuch. In *Aktuelle Intensivmedizin 1: Diagnose, Verlaufskontrolle und Therapie schwerer exogener Vergiftungen* (eds E. Deutsch, G. Kleinberger, R. Ritz & H. P. Schuster). Stuttgart: Schattauer.

Kessel, N. & McCulloch, W. (1966) Repeated acts of self-poisoning and self-injury. *Proceedings of the Royal Society of Medicine*, **59**, 89–92.

Kiev, A. (1976) Cluster analysis profiles of suicide attempters. *American Journal of Psychiatry*, **133**, 150–153.

Kockott, G. (1981) Der Selbstmordversuch im Alter. *Fortschritte der Medizin*, **99**, 1049–1056.

Kreitman, N. (1981) The epidemiology of suicide and parasuicide. *Crisis*, **2**, 1–13.

——, Philip, A. E., Greer, S. & Bagley, C. R. (1969) Parasuicide. *British Journal of Psychiatry*, **115**, 746–747.

Kurz, A., Torhorst, A., Wächtler, C. & Möller, H. J. (1982) Vergleichende Untersuchung von 295 Patienten mit erstmaligem und wiederholtem Suizidversuch. *Archiv für Psychiatrie und Nervenkrankheiten*, **232**, 427–438.

——, Möller, H. J., Bürk, F., Torhorst, A., Wächtler, C. & Lauter, H. (1985) Determination of treatment disposal for parasuicide patients in a German university hospital. *Social Psychiatry*, **20**, 145–151.

LITMAN, R. E. (1972) Experiences in a suicide prevention center. In *Suicide and Attempted Suicide* (eds J. Waldenström, T. Larsson & N. Ljungstedt). Stockholm: Nordiska Bokhandelns Förlag.

LORR, M. & KLETT, C. J. (1967) *Manual for the Inpatient Multidimensional Psychiatric Scale* (revised). Palo Alto: Consulting Psychologists Press.

MCHUGH, P. R. & GOODELL, H. (1971) Suicidal behavior. A distinction in patients with sedative poisoning seen in a general hospital. *Archives of General Psychiatry*, **25**, 456–464.

MÖLLER, H. J., BÜRK, F., KURZ, A., TORHORST, A., WÄCHTLER, C. & LAUTER, H. (1984) Stationäre und poststationäre psychiatrische Versorgung von Patienten nach Suizidversuchen durch Intoxikation. In *Aktuelle Intensivmedizin 1: Diagnose, Verlaufskontrolle und Therapie schwerer exogener Vergiftungen* (eds E. Deutsch, G. Kleinberger, R. Ritz & H. P. Schuster). Stuttgart: Schattauer.

MORGAN, H. G., BARTON, J., POTTLE, S., POCOCK, H. & BURNS-COX, C. J. (1976) Deliberate self-harm. A follow-up study of 279 patients. *British Journal of Psychiatry*, **128**, 361–368.

PALLIS, D. J. & BIRTCHNELL, J. (1977) Seriousness of suicide attempts in relation to personality. *British Journal of Psychiatry*, **130**, 253–259.

PAYKEL, E. S. & RASSABY, E. (1978) Classification of suicide attempters by cluster analysis. *British Journal of Psychiatry*, **133**, 45–52.

PIERCE, D. W. (1981) The predictive validation of a suicide intent scale. A five year follow-up. *British Journal of Psychiatry*, **139**, 391–396.

PINO, R., KOCKOTT, G. & FEUERLEIN, W. (1979) Sechs-Jahres-Katamnese an hundert Patienten mit Suicidversuchen durch Tabletteneinnahme. *Archiv für Psychiatrie und Nervenkrankheiten*, **227**, 213–226.

REED, C. E., DRIGGS, M. F. & FOOTE, C. C. (1952) Acute barbiturate intoxication. A study of 300 cases based on a physiologic system of classification on the severity of the intoxication. *Annals of Internal Medicine*, **37**, 290.

ROSEN, D. H. (1976) The serious suicide attempt. Five year follow-up of 886 patients. *Journal of the American Medical Association*, **235**, 2105–2109.

SCHMIDT, E. H., O'NEAL, P. & ROBINS, E. (1954) Evaluation of suicide attempts as a guide to therapy. Clinical and follow-up study of 109 patients. *Journal of the American Medical Association*, **155**, 549–552.

SIANI, R., GARZOTTO, N., ZIMMERMANN-TANSELLA, C. & TANSELLA, M. (1979) Predictive scales for parasuicide repetition. Further results. *Acta Psychiatrica Scandinavica*, **59**, 17–23.

SILVER, M. A., BOHNERT, M., BECK, A. T. & MARCUS, D. (1971) Relation of depression of attempted suicide and seriousness of intent. *Archives of General Psychiatry*, **25**, 573–576.

TORHORST, A., WÄCHTLER, C. & MOLLER, H. J. (1983) Zum Problem der "Ernsthaftigkeit" von Suizidversuchen. *Archiv für Psychiatrie und Nervenkrankheiten*, **223**, 151–166.

WOLD, C. I. (1971) Sub-groupings of suicidal people. *Omega*, **2**, 19–29.

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An American Validation Study of the Newcastle Diagnostic Scale

II. Relationship with Clinical, Demographic, Familial and Psychosocial Features

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We completed the Newcastle Diagnostic Scale on 152 unipolar depressed in-patients: its validity was supported by the findings that endogenous depressives were, in contrast to neurotic depressives, older, more severely depressed, with better social support, fewer life events, less personality disorder, and a lower morbid risk of alcoholism and antisocial personality in their first-degree relatives. The relationship between Newcastle scores and the morbid risk for alcoholism was non-linear, such that a cut-off score of 4, rather than 5, maximised the difference between the endogenous and neurotic groups with respect to familial alcoholism rates as well as other validating variables.

In 1972, research workers at Washington University, St Louis offered a five-phase paradigm to validate psychiatric diagnoses (Feighner *et al*, 1972). Their five phases of diagnostic validity were: clinical description, laboratory studies, delimitation from other disorders, family studies, and follow-up studies. The present paper is the second in a series in which we examine the validity of the Newcastle diagnostic scale (Carney *et al*, 1965) according to the Washington University model. Previously, we focused on the second phase of validity (laboratory studies), and reported a significant association between the Newcastle scale and results of the dexamethasone suppression test (DST) (Zimmerman *et al*, 1986*c*). Here, we report our results for the first and fourth phases of validity (clinical description and family studies).

The clinical description phase of validity refers to both the signs and symptoms of a disorder, as well as to demographic characteristics such as age, education, and sex ratio, and psychosocial variables such as life events, social support, and personality disorder diagnoses. Based on our survey of the literature of the 1920s and 1930s, before the discovery of effective antidepressant treatments, and on studies during the 1960s and 1970s examining the correlates of a clinical distinction between endogenous and neurotic depressives, we concluded that a valid operational definition of endogenous depression should identify a group of patients who, in contrast to neurotic depressives, are: older; more severely depressed; less likely to make a non-serious suicide attempt during their index episode; and have a more stable and well integrated lifestyle, as reflected by lower rates of premorbid personality disorder, fewer dysfunctional attitudes, lower frequencies of stressful life events during the year prior to hospital admission, lower frequency of marital separation and divorce, and better social support (Zimmerman *et al*, 1986*a*).

From a family history perspective, the literature suggested that endogenous depressives have a higher morbid risk for depression in their first-degree relatives and a lower frequency of familial alcoholism and antisocial personality. There is more empirical support for the alcoholism prediction than for the conventional wisdom that endogenous depressives have a higher familial rate of affective disorder. Three studies found a clinical diagnosis of reactive/neurotic depression significantly associated with a family history of alcoholism (Perris *et al*, 1982; Winokur, 1983; Winokur & Pitts, 1964). In a cluster analysis study, Andreasen & Grove (1982) found a significantly higher frequency of alcoholism in the families of patients in the non-endogenous than endogenous clusters.

Method

We approached a consecutive series of DSM-III major depressives aged 18 and older, admitted to the 58-bed psychiatry in-patient unit at the University of Iowa Hospitals and Clinics between June, 1982 and June, 1983. Because the research protocol included a DST, patients were excluded if they had any of the medical or pharmacological conditions specified by Carroll *et al* (1981), had insulin-dependent diabetes, or were taking carbamazepine.

A detailed description of our diagnostic procedures is given in Zimmerman *et al* (1986*c*). Because the unipolar/bipolar distinction is related to family history of affective disorder (Gershon *et al*, 1982) and to personality (Akiskal *et al*, 1983), only the 152 unipolar major depressives are described in this report.

Measures

Two of the authors (D. S., M. Z.) completed the 17-item Hamilton Rating Scale for Depression (HAMD; Hamilton, 1967), Beck Depression Inventory (BDI; Beck *et al*, 1979), and the Global Assessment Scale (GAS; Endicott *et al*, 1976) within one week of admission ($\bar{x}=2.5$ days after admission). Sucidal ideation and behaviour during the index episode were also recorded; judgements about the seriousness of suicidal behaviour reflected both the medical lethality and seriousness of intent of the attempt. The raters (B. P., W. C.) followed the SADS (Spitzer & Endicott, 1978) guidelines and a serious attempt would be rated 4 or above on SADS items 249 or 250.

The life event instrument was the 152-item Positive and Negative Impact (PANI) life event interview (Zimmerman *et al*, 1986*b*). A unique feature of the PANI is that it incorporates all the items from the two widely used life event scales developed by Paykel *et al* (1971) and by Tennant & Andrews (1976). In the present study, we compared endogenous and neurotic depressives on the total number of events experienced on the PANI, Paykel, and Tennant scales. In addition, we compared the groups on two event subcategories that the New Haven group found to be important in distinguishing depressed patients, suicide attempters, schizophrenics, and normal controls (Jacobs *et al*, 1974; Paykel *et al*, 1975): exits from the social field (e.g. deaths, divorce, child leaves home) and undesirable events. Finally, the interviewer rated the degree to which each event's occurrence might be the result of the patient's behaviour (i.e. event independence); e.g. an event such as divorce would usually be rated as non-independent, because it could be due to the depression. Death of a spouse, in contrast, would be rated as independent of the patient's behaviour.

Personality disorder diagnoses were made using the Structured Interview for DSM-III Personality Disorder (SIDP, Pfohl *et al*, 1982). This instrument includes a 90-minute interview with the patient and a 30–60 minute interview with a knowledgeable informant – a parent, spouse, sibling, or close friend. With the SIDP, our chance-corrected inter-rater reliability (kappa) for the presence or absence of personality disorder was 0.71 in joint interviews and 0.66 in independent interviews (Stangl *et al*, 1985).

Social support was rated on the four-point scale developed by Brown & Harris (1978). The essence of this rating is whether the respondent has at least one close, confiding, intimate relationship, and if so, how frequent are the interactions between the respondent and the confidant.

We used the Dysfunctional Attitudes Scale (DAS) to assess cognitive distortion (Weissman, 1980). This is a 40-item scale designed to measure the degree to which individuals endorse the attitudes hypothesised by cognitive theory to be associated with depression (Beck, 1972).

Family history diagnoses were based upon information collected from the patient, and are described in greater detail by Zimmerman *et al* (1985). Briefly, each relative was diagnosed twice – according to both a low and a high diagnostic threshold. Our low-threshold diagnoses were based on a strict interpretation of the Family History Research Diagnostic Criteria (FH-RDC; Endicott *et al*, 1978) whilst our high-threshold diagnoses required hospital admission for depression and treatment for alcoholism.

Data analysis

Morbid risks were calculated with age-corrected denominators or Bezugsziffers (BZs) by the Weinberg shorter method. Thus, relatives over the age of risk for the particular illness were given a value of 1; those within the age risk were given a value of 1/2; and those below it given a value of 0. Limits for these ages of risk for depression, alcoholism, and antisocial personality were 15 to 59, 20 to 39, and 20 to 39 respectively (Tsuang *et al*, 1984). Morbid risks were compared according to the method proposed by Breborowicz & Trzebiatowski-Trzeciak (1976), which modifies the χ^2 statistic to accommodate the statistical artifacts introduced by age correction in the denominators of the morbid risk calculations. We used the χ^2 statistic for other categorical comparisons and the *t*-test for comparisons of continuous variables.

Results

The majority of the 152 patients were female (109/152), high school graduates (122/152), and had one or more prior psychiatric hospital admissions (103/152); one-quarter (40/152) of the sample were separated or divorced. The mean scores on the rating scales completed during the first week of admission reflect a moderate-severe level of symptoms (HRS 22.5±6.4; BDI 27.8±11.2; GAS 38.9±8.7). The Newcastle scores ranged from −2 to 12 ($\bar{x}=4.79$, S.D.=2.6). Figure 1 shows that the scores were normally distributed.

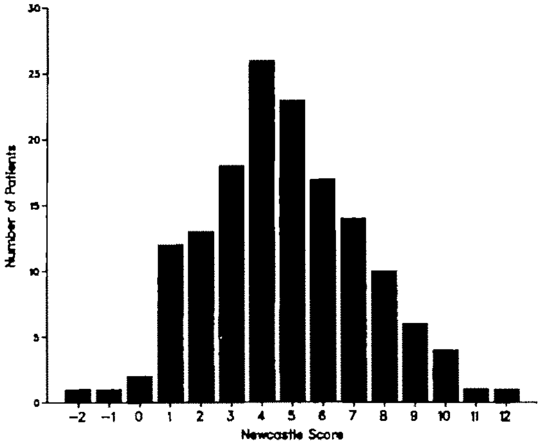

FIG. 1 Distribution of Newcastle scores in 152 unipolar depressed in-patients.

Family history

Kendell (1982) suggested that two syndromes, such as endogenous and neurotic depression, can be distinguished by demonstrating a non-linear relationship between symptomatology and some independent variable such as treatment response, family history, or a laboratory measure. The association between Newcastle scores and the morbid risk for FH-RDC alcoholism is non-linear (Fig. 2). (An identical relationship was also found with the narrower definition of treated alcoholism). This suggests that a cut-off of 4 should be used to distinguish endogenous and neurotic depression. The morbid risk for alcoholism was 2.5 times greater in the 26 patients with a Newcastle score of 4 than in the 23 patients with a score of 5 (11.7% *vs* 4.6%, $P<0.06$). The difference in familial rates of alcoholism between endogenous and neurotic depressives is greater with a cut-off of 4 in contrast to the conventional cut-off of 5 (Table I).

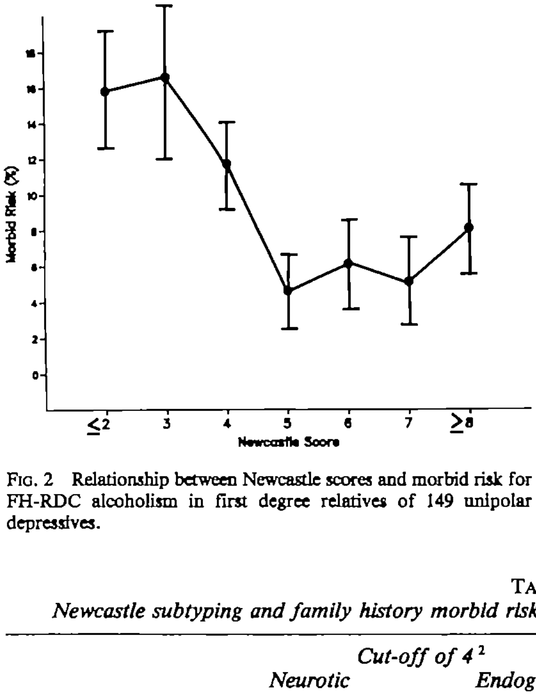

FIG. 2 Relationship between Newcastle scores and morbid risk for FH-RDC alcoholism in first degree relatives of 149 unipolar depressives.

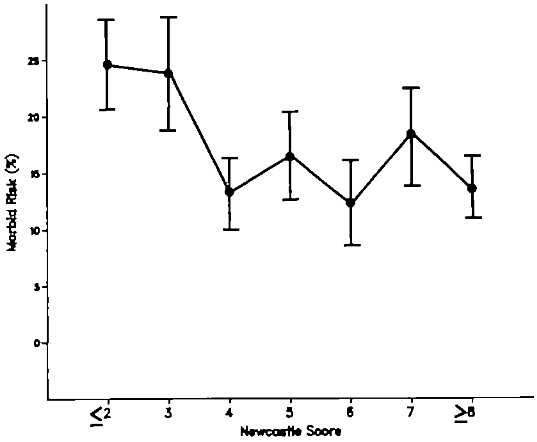

FIG. 3 Relationship between Newcastle scores and morbid risk for FH-RDC depression in first degree relatives of 149 unipolar depressives.

Inconsistent with our prediction, endogenous depressives had a *lower* morbid risk for affective disorder (Table I). Neurotic and non-endogenous depression cannot be clearly separated based on a family history of depression (Fig. 3).

Finally, we found that a family history of antisocial personality was greater in the neurotic depressives, although this was significant only when a cut-off of 4 was used (Table I). There were too few antisocial relatives ($n = 10$) to search for a point of discrimination between endogenous and neurotic depression.

In our previous report on the relationship between the Newcastle Scale and the DST, our results suggested that a cut-off of 4 was more valid than the conventional point of 5. The present findings for familial alcoholism rates are consistent with the DST analysis. In the remainder of the paper, we use both cut-offs in examining the relationship between Newcastle diagnosis and demographic, clinical, and psycho-social variables. The Newcastle-4 scale will refer to

TABLE I

Newcastle subtyping and family history morbid risks in first degree relatives of 149 unipolar depressives[1]

	Cut-off of 4[2]				Cut-off of 5[2]			
	Neurotic (n = 73)		*Endogenous (n = 76)*		*Neurotic (n = 96)*		*Endogenous (n = 53)*	
	BZ	*Morbid risk (%)*	*BZ*	*Morbid risk (%)*	*BZ*	*Morbid risk (%)*	*BZ*	*Morbid risk (%)*
FH-RDC depression	293.5	20.1	347	13.8*	391	19.2	249.5	12.8*
Hospitalised depression	283	12.4	341.5	8.8	378.5	11.6	246	8.5
FH-RDC alcoholism	325.5	14.4	386.5	5.9***	433.5	12.0	278.5	6.5*
Treated alcoholism	319.5	5.9	384	1.5***	426	4.5	277.5	2.2
Antisocial personality	380.5	2.1	448.5	0.4*	505	1.8	324	0.3

*$P<0.05$; **$P<0.01$; ***$P<0.001$

1. Family history information was not available for three patients.
2. For this table, and all subsequent tables, when a cut-off of 4 was used the neurotic depressives scored ≤4 and the endogenous patients ≥5 on the Newcastle scale. When a cut-off of 5 was used the neurotics scored ≤5 and the endogenous ≥ 6.

TABLE II

Newcastle subtyping and life events during the year prior to admission

	Cut-off of 4		*Cut-off of 5*	
	Neurotic (n=66)	*Endogenous (n=62)*	*Neurotic (n=84)*	*Endogenous (n=44)*
All events, $\bar{x}$: s.d.				
PANI	14.6 (7.9)	12.1 (9.0)*	14.6 (8.3)	11.2 (8.6)*
Tennant and Andrews	9.2 (5.2)	7.2 (5.7)*	9.1 (5.4)	6.6 (5.5)**
Paykel *et al*	7.5 (4.5)	6.0 (5.1)*	7.4 (4.6)	5.7 (5.2)*
Undesirable events	5.0 (3.0)	4.0 (3.5)*	4.9 (3.0)	3.8 (3.6)*
Exits	1.1 (1.2)	0.7 (0.9)*	1.0 (1.1)	0.8 (1.0)
Non-independent events, $\bar{x}$: s.d.				
PANI	6.5 (5.7)	5.1 (5.8)	6.5 (5.9)	4.6 (5.3)*
Tennant and Andrews	5.0 (4.2)	3.7 (4.3)*	4.9 (4.4)	3.4 (4.0)*
Paykel *et al*	4.1 (4.0)	3.1 (3.8)	4.0 (3.9)	2.9 (3.8)
Undesirable	3.0 (2.8)	2.2 (2.8)*	2.9 (2.8)	2.0 (2.7)*
Exits	0.3 (0.6)	0.2 (0.5)	0.3 (0.6)	0.2 (0.6)
Independent events, $\bar{x}$: s.d.				
PANI	8.1 (5.6)	7.1 (5.2)	8.1 (5.6)	6.6 (4.9)
Tennant and Andrews	4.2 (3.4)	3.5 (2.7)	4.2 (3.4)	3.2 (2.5)*
Paykel *et al*	3.4 (3.2)	2.9 (2.4)	3.4 (3.1)	2.8 (2.3)
Undesirable events	2.0 (1.8)	1.8 (1.7)	2.0 (1.8)	1.8 (1.6)
Exits	0.8 (0.9)	0.6 (0.8)	0.7 (0.9)	0.6 (0.8)

$^{*}P<0.05$; $^{**}P<0.01$

dichotomisation about a cut-off of 4, whereas the Newcastle-5 scale refers to the traditional diagnosis based on a cut-off of 5.

Demographic and clinical characteristics

Newcastle diagnosis was not significantly associated with sex ratio or level of education. Newcastle-4 neurotic depressives were significantly more likely to be separated or divorced at the time of the index admission (34.2% *vs* 19.0%, $P<0.05$). Newcastle-5 diagnosis was similarly associated with current marital separation or divorce (31.2% *vs* 17.9%, $P<0.10$). Endogenous depressives were significantly older than neurotics according to both scales (Newcastle-4 44.4 ± 17.1 *vs* 34.4 ± 13.2, $P<0.001$; Newcastle-5 46.7 ± 17.7 *vs* 35.5 ± 13.5, $P<0.001$).

Baseline HRS and BDI scores were significantly higher, and GAS scores significantly lower (which indicates greater severity), in endogenous depressives (Newcastle-4 HRS 24.6 ± 5.2 *vs* 20.2 ± 6.8, $P<0.001$; BDI 30.1 ± 10.8 *vs* 25.6 ± 11.3; GAS 35.0 ± 6.2 *vs* 43.1 ± 9.2; Newcastle-5 HRS 25.3 ± 5.0 *vs* 20.9 ± 6.6; BDI 31.6 ± 10.8 *vs* 25.8 ± 10.0; GAS 34.1 ± 6.0 *vs* 41.7 ± 8.9). Serious suicide attempts were equally frequent in endogenous and neurotic depressives; however, non-serious suicide attempts were more frequent in neurotic depressives (Newcastle-4 39.7% *vs* 5.1%, $P<0.001$; Newcastle-5 31.3% *vs* 5.4%, $P<0.001$). Approximately 90% of the 33 non-serious suicide attempers were neurotic depressives.

Psychosocial variables

Life events

The PANI semi-structured life event interview was completed on 128 (84.2%) patients. There was no association between Newcastle diagnosis and interview completion. Neurotic depressives reported significantly more events during the year prior to admission (Table II). In general, the relationship between Newcastle diagnosis and life events was similar for both cut-offs. The relationship between subtyping and stress was stronger for non-independent events than for events independent of the patient's behaviour.

Social support

The rating of confidant support based on Brown & Harris' (1978) method was available for 142 (93.4%) patients. Individuals with an 'A' rating on this scale have a close, intimate, and confiding relationship with their spouse, boyfriend/girlfriend, or other friend with whom they are sharing a residence. Newcastle-4 endogenous depressives were significantly more likely to have an 'A' rating than Newcastle-4 neurotic depressives. The Newcastle-5 scale was not significantly associated with the rating of confidant support (Table III).

Personality disorder

Of 89 patients interviewed with SIDP, half received at least one DSM-III Axis II personality disorder. The frequency

TABLE III

Social support, personality disorder, and dysfunctional attitudes in Newcastle subtypes

	Cut-off of 4		*Cut-off of 5*	
	Neurotic	*Endogenous*	*Neurotic*	*Endogenous*
'A' rating on Brown & Harris' social support scale: %[1]	20.0	38.9**	26.4	35.3
DSM-III personality disorder: %[2]	64.2	30.5**	60.3	26.9**
Dysfunctional attitude scale,[3] $\bar{x}$: s.d.	143.9 (41.1)	144.4 (38.0)	142.4 (39.3)	146.9 (40.0)

1. Based on a cut-off of 4, 95.9% (70/73) neurotic and 91.1% (72/79) endogenous patients were interviewed.
   Based on a cut-off of 5, 94.8% (91/96) neurotic and 91.1% (51/56) endogenous depressives were interviewed.
2. Psychotic patients were not administered the SIDP. In our previous paper we reported that in contrast to neurotic depressives significantly more endogenous patients had delusions and/or hallucinations (Zimmerman *et al*, 1986*c*). Thus it is not surprising that SIDPs were more frequently administered to the neurotic patients (Newcastle-4: 72.6% (53/73) vs 45.6% (36/79), $\chi^2 = 11.4$, $P < 0.001$; Newcastle-5: 65.6% (63/96) vs 46.4% (26/56), $\chi^2 = 5.37$, $P < 0.05$).
3. Based on a cut-off of 4, 41.1% (30/73) neurotic and 35.4% (28/79) endogenous patients completed the DAS.
   Based on a cut-off of 5, 37.5% (36/96) neurotic and 39.3% (22/56) endogenous patients completed the DAS.

of each specific diagnosis is presented elsewhere (Pfohl *et al*, 1984). As diagnosed by both Newcastle scales, neurotic depressives were significantly more likely to have a personality disorder (Table III). The relationship between Newcastle subtyping and personality disorder diagnoses is partially confounded because the adequate personality item on the Newcastle scale was influenced by the SIDP results. We recomputed each patient's Newcastle score, based upon the remaining nine items. Using the same cut-offs as the ten-item scale, we found that the relationship between the corrected Newcastle diagnoses and personality disorder was still significant.

Cognitive distortion

Newcastle subtyping was unrelated to DAS scores (Table III). Several studies have recently suggested that depressives' cognitive style normalises after depressive symptoms resolve (Eaves & Rush, 1984; Simons *et al*, 1984). If the DAS is more of a state characteristic of all depression than a trait marker for certain types of depression, then the lack of association between the Newcastle scale and DAS scores should have been expected.

Discussion

Though the Newcastle diagnostic scale was published almost 20 years ago, its validity has only recently received much attention. Renewed interest in it has paralleled the growing literature on biological markers of endogenous depression. The most frequently examined biochemical correlate of Newcastle subtyping is the dexamethasone suppression test (DST). Similar to our previously reported results, six other studies found significantly higher DST non-suppression rates in endogenous depressives (McIntyre *et al*, 1981; Coppen *et al*, 1983; Kasper & Beckmann, 1983; Holden, 1983; Davidson *et al*, 1984*a*; McKeith, 1984); two negative findings have also been published (Ames *et al*, 1984; Klein *et al*, 1984). Two studies reported that endogenous depressives are older (Kiloh & Garside, 1963; Abou-Saleh & Coppen, 1984), though a third (Davidson *et al*, 1984*b*) found no age differences between the groups. Two studies found that endogenous depressives have higher symptom severity scores (Davidson *et al*, 1984*b*; Klein *et al*, 1984). The only study other than our own which examined the patients' family history of depression found no differences (Abou-Saleh & Coppen, 1984). No previous studies compared familial alcoholism rates, nor are we aware of any studies which examined psychosocial characteristics such as life events and social support.

Our results suggest that the Newcastle diagnostic scale is a valid discriminator of endogenous and neurotic depressives respectively. Consistent with our predictions, endogenous patients were characterised by greater severity of symptoms, older age, better social support, fewer life events, less personality disorder, and a lower familial rate of alcoholism and antisocial personality. In contrast, neurotic depressives were younger, less severely depressed patients who in many cases had been admitted to hospital after a non-serious suicide attempt, and had a more stormy and disruptive lifestyle, as reflected by more life events (particularly the types of events that may have been caused by the individual rather than those occurring outside of the individual's control), poorer social support, and more personality disorder, as well as a higher morbid risk of alcoholism and antisocial personality in their first-degree relatives.

Since the 1920s, European psychiatrists have debated whether endogenous and neurotic depressives are clearly distinct subtypes. These debates have often centered around the form of the frequency distribution of symptom scores – a bimodal distribution of scores being interpreted as support for the two-type thesis. A bimodal distribution of Newcastle scores has been found only by Carney *et al* (1965) and by Carney & Sheffield (1972). The unimodal distribution of scores we found is consistent with the results of other research groups (Abou-Saleh & Coppen, 1984; Bech & Rafaelsen, 1980; Kendell, 1968). Kendell (1982), however, indicated that support for typological classification is also provided by demonstrating a non-linear relationship between symptomatology and an independent variable such as treatment response, family history, or a laboratory measure. We have found two such relationships – one between Newcastle scores and the morbid risk for alcoholism in first-degree relatives, and the other between Newcastle scores and rates of DST non-suppression (Zimmerman *et al*, 1986*b*). Both analyses found that a cut-off of 4 was the optimal point of discrimination between neurotic and endogenous groups. In their study of the DST and the Newcastle scale, Davidson *et al* (1984*a*) also reported that a cut-off of 4 was superior to the conventional point of 5.

Our one major negative finding was the failure to validate Newcastle subtyping with familial depression rates: in fact, the results were contrary to initial expectation, with the neurotic depressives having a significantly higher morbid risk of depression. The conventional wisdom that endogenous depression is more genetic than neurotic depression may be incorrect. Just as there are genetic components to antisocial personality (Crowe, 1974) and anxiety disorders (Torgersen, 1983), the aetiology of neurotic depression probably involves an inherited component. Neurotic and endogenous depression may be equally genetic; but neurotic depression may be more familial. The results of our study suggest that neurotic depression is associated with more environmental chaos and disruption; therefore, perhaps we should expect a higher rate of depression in relatives of neurotic depressives because they are exposed to both genetic and psychosocial insults, whereas the relatives of endogenous depressives are only genetically predisposed. For example, the children of neurotic depressives may be psychosocially vulnerable to (neurotic) depression because their parents separated or divorced. Consistent with this, Brown & Harris (1978) found that early parental loss due to separation was associated with a clinical diagnosis of neurotic rather than endogenous depression. It will be necessary to turn to twin and adoption studies in order to better partial out the relative effects of genetic and psychosocial determinants of depressive subtypes.

References

Abou-Saleh, M. T. & Coppen, A. (1984) Classification of depressive illnesses: Clinical-psychological correlates. *Journal of Affective Disorders*, **46**, 53–66.

Akiskal, H. S., Hirschfeld, R. M. & Yerevanian, B. I. (1983) The relationship of personality to affective disorders: A critical review. *Archives of General Psychiatry*, **40**, 801–810.

Ames, D., Burrows, G., Davies, B., Maguire, K. & Norman, T. (1984) A study of the dexamethasone suppression test in hospitalized depressed patients. *British Journal of Psychiatry*, **144**, 311–313.

Andreasen, N. C. & Grove, W. M. (1982) The classification of depression: Traditional versus mathematical approaches. *American Journal of Psychiatry*, **139**, 45–52.

Bech, P. & Rafaelsen, O. J. (1980) The use of rating scales exemplified by a comparison of the Hamilton and the Bech-Rafaelsen melancholia scale. *Acta Psychiatrica Scandinavica*, **62** (Suppl. 285), 128–132.

Beck, A. T. (1972) *Depression: Causes And Treatment*. Philadelphia: University of Pennsylvania Press.

——, Rush, A. J., Shaw, B. F. & Emery, G. (1979) *Cognitive Therapy for Depression*, New York: Guilford Press.

Breborowicz, G. & Trzebiatowska-Trzeciak, O. (1976) A method for testing differences in morbidity risk for affective psychoses. *Acta Psychiatrica Scandinavica*, **54**, 353–358.

Brown, G. W. & Harris, T. O. (1978) *Social Origins Of Depression*, London: Tavistock.

Carney, M. W. P. & Sheffield, B. F. (1972) Depression and the Newcastle scales: Their relationship to Hamilton's Scale. *British Journal of Psychiatry*, **121**, 35–40.

——, Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal Psychiatry*, **111**, 659–674.

Carroll, B. J., Feinberg, M., Greden, J. F., Tarika, J., Albala, A. A., Haskett, R. F., James, N. M., Kronfol, Z., Lohr, N., Steiner, M., Devigne, J. P. & Young, E. (1981) A specific laboratory test for the diagnosis of melancholia: Standardization, validation, and clinical utility. *Archives of General Psychiatry*, **38**, 15–22.

Coppen, A., Abou-Saleh, M., Milln, P., Metcalfe, M., Harwood, J. & Bailey, J. (1983) Dexamethasone suppression test in depression and other psychiatric illnesses. *British Journal of Psychiatry*, **142**, 498–504.

Crowe, R. R. (1974) An adoption study of antisocial personality. *Archives of General Psychiatry*, **31**, 785–791.

Davidson, J., Lipper, S., Strickland, R., Krishnan, R. & Mahorney, S. (1984*a*) Validation of four definitions of melancholia by the dexamethasone suppression test. *American Journal of Psychiatry*, **141**, 1220–1223.

——, Turnbull, C., Strickland, R. & Belyea, M. (1984*b*) Comparative diagnostic criteria for melancholia and endogenous depression. *Archives of General Psychiatry*, **41**, 506–511.

Eaves, G. & Rush, A. J. (1984) Cognitive patterns in symptomatic and remitted unipolar major depression. *Journal of Abnormal Psychology*, **93**, 31–40.

Endicott, J., Spitzer, R. J., Fleiss, J. L. & Cohen, J. (1976) The Global Assessment Scale: A procedure for measuring overall severity of psychiatric disturbance. *Archives of General Psychiatry*, **33**, 766–771.

——, Andreasen, N. & Spitzer, R. L. (1978) *Family History Research Diagnostic Criteria*, 3rd ed. New York: Biometrics Research, New York State Psychiatric Institute.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

Gershon, E. S., Hamovit, J., Guroff, J. J., Dibble, E., Leckman, J. F., Sceery, W., Targum, S. D., Nurnberger, J. I., Goldin, L. R. & Bunney, W. E. (1982) A family study of schizoaffective, bipolar I, bipolar II, unipolr, and normal control probands. *Archives of General Psychiatry*, **39**, 1157–1167.

Hamilton, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–296.

Holden, N. L. (1983) Depression and the Newcastle scale: Their relationship to the dexamethasone suppression test. *British Journal of Psychiatry*, **142**, 505–507.

Jacobs, S. C., Prusoff, B. A. & Paykel, E. S. (1974) Recent life events in schizophrenia and depression. *Psychological Medicine*, **4**, 444–453.

Kasper, S. & Beckmann, H. (1983) Dexamethasone suppression test in a pluridiagnostic approach: Its relationship to psychopathological and clinical variables. *Acta Psychiatrica Scandinavica*, **68**, 31–37.

Kendell, R. E. (1968) The problem of classification. In *Recent Developments in Affective Disorders* (eds A. Coppen & A. Walk), Ashford: Headley Brothers.

—— (1982) The choice of diagnostic criteria for biological research. *Archives of General Psychiatry*, **39**, 1334–1339.

Kiloh, L. G. & Garside, R. F. (1963) The independence of neurotic depression and endogenous depression. *British Journal of Psychiatry*, **109**, 451–463.

Klein, H. E., Seibold, B., Bender, W., Nedopil, N., Albus, M. & Schmauss, M. (1984) Postdexamethasone prolactin and cortisol: A biological state variable in depression. *Acta Psychiatrica Scandinavica*, **70**, 239–247.

McIntyre, I. M., Norman, T. R., Burrows, G. D., Davies, B. & Maguire, K. P. (1981) The new psychiatry (letter). *British Medical Journal*, **283**, 1609–1610.

McKeith, I. G. (1984) Clinical use of the DST in a psychogeriatric population. *British Journal of Psychiatry*, **145**, 389–393.

Paykel, E. S., Prusoff, B. A. & Uhlenhuth, E. H. (1971) Scaling of life events. *Archives of General Psychiatry*, **25**, 340–347.

——, —— & Myers, J. K. (1975) Suicide attempts and recent life events. *Archives of General Psychiatry*, **32**, 327–333.

Perris, H., Von Knorring, L. & Perris, C. (1982) Genetic vulnerability for depression and life events. *Neuropsychobiology*, **8**, 241–247.

Pfohl, B., Stangl, D. & Zimmerman, M. (1982) *The structured interview for DSM-III personality disorders (SIDP)*, Iowa City, Iowa: Department of Psychiatry, University of Iowa.

——, —— & —— (1984) The implications of DSM-III personality disorders for patients with major depression. *Journal of Affective Disorders*, **7**, 309–318.

Simons, A. D., Garfield, S. L. & Murphy, G. E. (1984) The process of change in cognitive therapy and pharmacotherapy for depression: Changes in mood and cognition. *Archives of General Psychiatry*, **41**, 45–51.

Spitzer, R. L. & Endicott, J. (1978) *Schedule for Affective Disorders and Schizophrenia*. New York: Biometric Research, New York State Psychiatric Institute.

Stangl, D., Pfohl, B., Zimmerman, M., Bowers, W. & Corenthal, C. (1985) A structured interview for the DSM-III personality disorders. *Archives of General Psychiatry*, **45**, 591–596.

Tennant, C. & Andrews, G. (1976) A scale to measure the stress of life events. *Australian and New Zealand Journal of Psychiatry*, **10**, 27–32.

Torgersen, S. (1983) Genetic factors in anxiety disorders. *Archives of General Psychiatry*, **40**, 1085–1089.

Tsuang, M. T., Winokur, G. & Crowe, R. R. (1984) Psychiatric disorders among relatives of surgical controls. *Journal of Clinical Psychiatry*, **45**, 420–422.

Weissman, A. N. (1980) *Assessing Depressogenic Attitudes: A Validation Study*. Paper presented at the 51st annual meeting of the Eastern Psychological Association, Hartford, CT.

Winokur, G. (1983) Controversies in depression, or do clinicians know something after all? *Treatment of Depression: Old Controversies and New Approaches* (eds P. J. Clayton & J. E. Barrett), New York: Raven Press.

—— & Pitts, F. N. (1964) Affective disorder: I. Is reactive depression an entity? *Journal of Nervous and Mental Disorders*, **138**, 541–547.

Zimmerman, M., Coryell, W. & Pfohl, B. M. (1985) The importance of diagnostic thresholds in familial classification: The dexamethasone suppression test and familial subtypes of depression. *Archives of General Psychiatry*, **42**, 300–304.

——, —— & Stangl, D. (1986*a*) The validity of four definitions of endogenous depression: II. Clinical, demographic, familial and psychosocial correlates. *Archives of General Psychiatry*, **43**, 234–244.

——, Pfohl, B. & Stangl, D. (1986*b*) Life event assessment in depressed patients: A comparison of self-report and interview formats. *Journal of Human Stress*, 13–19.

——, ——, —— & Coryell, W. (1986*c*) An American validation study of the Newcastle diagnostic scale. I. Relationship with the dexamethasone suppression test. *British Journal of Psychiatry*, **149**, 627–630.

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An Evaluation of the Performing and Recording of Physical Examinations by Psychiatric Trainees

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The physical examination on admission, as recorded in the psychiatric case notes, was assessed for 100 consecutive admissions under the age of 65. Assessment was made on the presence or absence of reference to 43 items of examination and the recording of the examinations was found to be uniformly poor. One of the authors re-examined 50 patients within 48 h of admission and the findings were then compared with the initial recorded examination. Significant unrecorded positive findings were most frequent in the neurological and locomotor systems.

It has been demonstrated that psychiatric and physical disorders occur together more than by chance combination. Herridge (1960) and Kellner (1966) have shown that physical illness often precedes psychiatric disorder and Lovett Doust (1952) found that psychiatric patients experienced more physical illness than healthy controls, with psychotic patients suffering more than those in whom the diagnosis was a neurosis or personality disorder. In a general practice survey with a cohort in excess of 14 000, Shepherd *et al* (1966) confirmed the high incidence of physical illness in psychiatric populations and Koryani (1972), found almost 50% of a study of 100 consecutive out-patient attenders to have 'significant physical illness', of which it was thought to be the direct cause in half. That this extends to psychiatric inpatients was demonstrated by Maguire & Granville-Grossman (1968) who, in 200 consecutive admissions to a psychiatric hospital found 57 with physical disorder, half of which had not previously been diagnosed.

Thus, psychiatrists often meet physical disorder in their psychiatric population, and psychiatrists in training need, and are expected to, regularly exercise their physical skills. Increasingly however, trainees begin to specialise immediately on registration and therefore have limited opportunity to consolidate these medical skills. The present study assesses how adequately physical examinations are recorded, and whether or not they satisfactorily identify physical disorder.

Method

The psychiatric case notes of 100 consecutive admissions under the age of 65 were obtained and the record of the physical examination on admission was rated on the presence, or absence, of reference to a 43-item check list based on the standard admission document of the professorial medical unit. The maximum score for each recorded physical examination was thus 43. For each individual item, e.g. finger clubbing, carotid bruits, it was then noted in how many of the 100 case notes it was referred to, thus giving a score of 0–100 in each item.

Of the 100 consecutive admissions, alternate patients were re-examined by one of the authors (J.C.R.) within 48 h of admission, and the positive findings compared with the admission record. To avoid any discomfort to the patients concerned, pelvic examination was excluded in all cases, although both external genitalia and perianal areas were examined. Measurement of blood pressure was similarly excluded, as its magnitude may be altered significantly by changing mental state, and it was felt that this would make interpretation of the findings difficult. Although none of the patients refused examination for research purposes, two were judged sufficiently psychiatrically disturbed so as to make meaningful examination impossible without sedation, and in these cases the next two consecutive admissions were examined. Thus 50 patients were examined, 21 males and 29 females.

Results

The 100 recorded physical examinations were scored using the 43-item check list, and the average number of items referred to was 20. The range was 11–37. For each item of examination the number of admission records which referred to it, either positively or negatively, is recorded in Table I. The items are grouped into the bodily systems to which they appertain, and listed in order of the frequency to which they were referred.

For the 50 patients re-examined, 54 abnormalities were found, of which 32 were recorded in the notes. Table II records, for each item of examination, the discrepancies between the number of positive findings made by J.C.R.,

TABLE I

Checklist and frequency of reference to each item in the admission record

General observation		*Cardiovascular system*	
Nodes	74	Pulse rate	100
Anaemia	62	BP	96
Cyanosis	57	Heart sounds	95
Jaundice	53	Murmurs and added sounds	76
Clubbing	41	Jugular venous pressure	63
Breasts	21	Pulse rhythm	63
Goitre	19	Dependent oedema	54
		Apex beat	27
		Carotid bruits	15
		Peripheral pulses	4
Respiratory system		*Alimentary system*	
Breast sounds	74	Scars	65
Additional sounds	69	Tenderness	59
Percussion	68	Masses	57
Trachea	53	Organomegaly	55
Chest movements	42	Rectal examination	22
Respiratory rate	12	Genitals	19
		Hernial orifices	14
		Bowel sounds	5
Neurological system		*Locomotor system*	
Pupils	86		1
Reflexes	85		
Fundi	59		
Power	59		
Sensation	54		
Tone	51		
Cranial Nerves	40		
Co-ordination	34		
Abnormal Movements	22		
Gait	6		
Meningism	0		

and those made by trainees at the initial examination.

Of those patients re-examined, general observation revealed two cases of finger clubbing and one of goitre, all of which had been noted initially, although one case of clinically apparent anaemia (subsequently confirmed by an Hb of 9g) was missed. No cases of lymphadenopathy, jaundice, cyanosis or breast abnormalities were found at either examination.

In the cardiovascular system two findings of atrial fibrillation, two cardiaic murmurs, and one of dependent oedema were made at both examinations, and one case of absent peripheral pulses was found at re-examination but not recorded in the admission record.

In the respiratory system five cases of added sounds were noted at both examinations and no other positive findings made.

In the gastronintestinal system one case of hepatomegaly was noted both on admission and at re-examination, as was one case of inguinal hernia. Another inguinal hernia was missed, and only seven of 13 abdominal scars were noted.

Neurologically, one case of unequal pupils and two of limb weakness were noted at both examinations. Although one case of tardive dyskinesia, presenting as choreoathetotic limb movements, was recorded on admission, two orobucco-lingual tardive dyskinesias were not. Concerning drug-induced parkinsonism, five cases were observed at re-examination. All had increased tone, three had tremor, and there were two with a typical parkinsonian gait. Of these, three cases of rigidity were not recorded, as were one each of tremor and gait abnormality. The diagnosis of drug-induced parkinsonism was made in only two of five cases.

In the locomotor system, re-examination revealed one case each of severely disorganised hand joints (rheumatoid arthritis), marked unilateral lower limb deformity (old trauma), hip movements markedly limited by pain, and frozen shoulder, none of which had been noted initially. Dupuytren's contracture was noted in both examinations on one occasion.

TABLE II

Discrepancies between initial examination and re-examination

Item	*Positive findings made by trainees*	*Positive findings made by J.C.R.*	*Number of discrepancies*
Clubbing	2	2	0
Goitre	1	1	0
Anaemia	0	1	1
Pulse rhythm	2	2	0
Cardiac murmurs	2	2	0
Dependent oedema	1	1	0
Peripheral pulses	0	1	1
Additional chest sounds	5	5	0
Hepatomegaly	1	1	0
Hernial orifices	1	2	1
Abdominal scars	7	13	6
Pupillary abnormalities	1	1	0
Muscle power	2	2	0
Tardive dyskinesias	1	3	2
Muscle tone	2	5	3
Drug induced tremors	2	3	1
Parkinsonian gait abnormalities	1	2	1
Other gait abnormalities	0	2	2
Locomotor abnormalities	1	5	4

Discussion

The records made of physical examinations on admission often seem to have significant deficiencies. On average, reference was made to only 20 items of the 43-item check list, and many items were regularly omitted. Breasts, goitre, carotid auscultation, peripheral pulses, respiratory rate, rectal examination, hernial orifices, bowel sounds, abnormal movements, gait, meningism, and the locomotor system were items particularly seldom referred to.

Where positive findings are not made, it is of some importance to have documented that they have at least been looked for. Only by such documentation may the line be drawn between negligence and clinical error. Not only for such medicolegal reasons is recording of negative findings as important as that of positive findings, but also for purely medical reasons. Should positive findings be made, it is of importance to know whether these are of recent onset, or long-standing and present at previous examinations.

In this study, re-examination was carried out in most circumstances under optimal conditions, with no pressure of time, and on patients who were probably more co-operative than they had been at the time of the initial examination. Although the re-examination was carried out without reviewing the admission record first, i.e. it was blind, no abnormality of any system was found on the first occasion that was not noted on the second.

The discrepancies between the initial examination and the re-examination were most marked in the neurological and locomotor systems. Drug-induced parkinsonism had not been noted in three of five cases, and tardive dyskinesia in two of three cases. On none of these occasions had medication been altered between the time of the initial examination and the re-examination, and the discrepancies are therefore likely to have resulted from a failure to recognise the clinical signs. Of four significant gait abnormalities, (two resulting from parkinsonism, and two from other locomotor abnormalities) found at re-examination, only one was documented at the initial recording. In the examination of the neurological system, trainees clearly need to pay more attention to muscle tone, gait, and the presence of abnormal movements.

The discrepancies in the findings of the locomotor system seem to have resulted from failure to examine joints, to test movements adequately, and again from inadequate observation of gait.

Comparing Table II with Table I, it would seem that, for the neurological and locomotor systems particularly, low rates of recording, e.g. of muscle tone, abnormal movements, and gait, are accompanied by significant discrepancies between the initial examination and the re-examination. This also remained true for the other systems for, with the exception of anaemia and abdominal scarring (both of which were well recorded), discrepancies occurred in those areas least well documented e.g. peripheral pulses and hernial orifices.

It appears therefore, that it is among the more poorly recorded items that positive findings are most often missed. The encouraging of a more complete recording may thus also encourage a more complete and therefore successful examination. Further work is required to investigate whether, for example, the provision of a standard typed record, allowing space for the description of signs in each system, would achieve this end.

References

Herridge, C. G. (1960) Physical disorders in psychiatric illness. *Lancet*, ii, 949–951.

Kellner, R. (1966) Psychiatric ill-health following physical illness. *British Journal of Psychiatry*, 112, 71–73.

Koryani, E. K. (1972) Physical health and illness in a psychiatric department outpatient population. *Canadian Psychiatric Association Journal*, 17, 109–116.

Louvett Doust, J. W. (1952) Psychiatric aspects of somatic immunity: differential incidence of physical disease in the histories of psychiatric patients. *British Journal of Social Medicine*, 6, 49–67.

Maguire, G. P. & Granville-Grossman, K. L. (1968) Physical illness in psychiatric patients. *British Journal of Psychiatry*, 115, 1365–1369.

Shepherd, M., Cooper, B., Brown, A. C., Kalton, G. W. (1966) *Psychiatric Illness in General Practice*. London: Oxford University Press.

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Five Risk Factors for Depression

A. ROY

Significantly more of 300 patients with non-endogenous depression compared with 300 matched controls were unemployed and had a poor marriage before the onset of depression, had a first-degree relative who had been treated for depression, had experienced separation for one year or more from a parent before 17 years of age and had three or more children under 14 years of age at home. However, significantly more of 44 patients with endogenous depression, than their 46 controls, also had a poor marriage before the onset of depression and 43% of them had a first-degree relative who had been treated for depression. Thus a family history of depression and a poor marriage before the onset of depression are associated with both non-endogenous and endogenous depression but unemployment, separation for one year or more from a parent before 17 years of age, and having three or more young children at home may be risk factors for non-endogenous depression.

In a community study, Brown & Harris (1978) described four vulnerability factors which increased the chances of a woman developing depression in the presence of a life event or difficulty. They were: lack of full-time or part-time employment, lack of a good marital relationship, the presence of three or more children under 14 years at home, and parental loss before 17 years of age. In psychiatric patients, when compared to medical patient controls, these four factors along with a fifth – depression in a first-degree relative – have been previously reported to be significantly associated with non-endogenous depression (Roy, 1978, 1981*a*,*b*). However, the specificity of these factors for the non-endogenous subtype of depressive disorder has not been examined. Thus the purpose of this final report is to examine these risk factors in subtypes of depressive disorder by presenting the data as found in (a) this large gradually accumulated series of 300 patients with non-endogenous depression compared to their 300 matched normal controls and in (b) a consecutive series of 44 patients with endogenous depression compared to 46 normal controls.

Method

From July 1976 to June 1982 300 white patients meeting DSM-III criteria for a major depressive episode (MDE) without melancholia were seen (Diagnostic and Statistical Manual of Mental Disorders, 1980). Before DSM-III draft criteria became available the International Classification of Diseases (1965) category 3000.4, depressive neurosis was used. All of these patients would have met DSM-III criteria for MDE without melancholia and, in fact, had to have had a duration of depression of at least one month to be studied – twice the duration criterion in DSM-III. Few patients were seen with bipolar or psychotic depression or melancholia ('endogenous' depression) and thus were excluded as were patients with secondary depression. These 300 patients are the 'non-endogenous' sample.

The first 84 patients were all female patients seen at the Maudsley and Dulwich Hospitals in London. The remaining 216 depressed patients were seen at the Clarke Institute of Psychiatry, Toronto. All 300 patients were matched for age (within 5 years), sex, marital status, social class and country with non-depressed and never-depressed gynaecological and orthopaedic patients seen at Dulwich Hospital or clinics of the Wellesley or Orthopaedic and Arthritic Hospitals, Toronto. The General Health Questionnaire (GHQ) (Goldberg, 1972), followed by a short psychiatric interview, was used to screen out potential controls with depression or other psychiatric disorder.

From July 1982 to June 1985 a consecutive series of 44 white in-patients with endogenous depression (296.2, ICD) who met DSM-III criteria for a major depressive episode with melancholia was collected at the National Institute of Mental Health (NIMH), in Bethesda, Maryland, and are the 'endogenous' sample. They were compared with (a) 46 normal controls recruited through the NIMH normal volunteer office; and (b) with the 27 of these 46 controls with whom they could be matched for age, sex and marital status. Controls were interviewed with the Schedule for Affective Disorders and Schizophrenia (SADS-L) (Endicott & Spitzer, 1979) and had no past, current or family history of psychiatric disorder.

All 690 subjects were asked by the same interviewer about age, marital status, the occupation of the head of the household, whether they were in full- or part-time employment (at the onset of depression), about early parental loss because of death or separation for one year or more between 0 and 17 years of age, whether there was a first-degree relative treated for depression by a psychiatrist, and whether there were three children under 14 years of age at home (earlier papers reported three children 14 years or younger). The duration of depression and the Hamilton depression score (1960) were recorded. For married (or cohabiting) subjects a semi-structured marital interview was performed, using the method of Quinton *et al* (1976), covering the year before the onset of depression (or the year before for controls). Ratings 1–3 were called 'good marriage' and ratings 4–6 'poor marriage'. Two reliability studies of the marital interview were performed (Roy, 1978, 1981*a*). (The marriages of the first 16 depressed patients were not assessed.) Social class was determined using the occupation of the head of the household and the classification of occupations of Goldthorpe and Hope (1974); 1–22 was called middle class and 23–36 working class.

In the statistical analysis the χ^2 and Fisher's exact test were used and possible interactions were examined for using linear model analysis (Grizzle *et al*, 1969).

Results

The demographic data and clinical variables are shown in Table I.

Non-endogenous depression

Unemployment before onset of depression

Significantly more depressed patients than controls were unemployed before the onset of depression (104 vs 57, $P<0.0001$) (Table II). Significantly more of both the total group of Canadian ($n=216$) and British ($n=84$) depressed patients than controls were unemployed (71 vs 39, $P<0.01$; 33 vs 18, $P<0.0006$, respectively). Significantly more of both the total group of working-class ($n=129$) and female working-class ($n=88$) depressed patients were unemployed (63 vs 31, $P<0.0001$; 50 vs 24, $P<0.0001$, respectively).

Poor marriage before onset of depression

Significantly more depressed patients than controls had a poor marriage before the onset of depression (94 vs 13, $P<0.0001$) (Table II). Significantly more of both the total group of working ($n=53$) and middle-class ($n=51$) female depressed patients than controls had a poor marriage (36 vs 6, $P<0.0001$; 33 vs 2, $P<0.0001$, respectively).

Among the married English depressed 14 of 32 (44%) working-class and four of 16 (25%) middle-class patients were unable to confide in their spouse compared with 0 and 0 of controls ($P<0.0001$ and NS). Among the married Canadian depressed 16 of 36 (44%) working-class and 14 of 55 (26%) middle-class patients were unable to confide in their spouse compared with 1 (3%) and three of controls ($P<0.0001$ and $P<0.008$, respectively).

Three or more children under 14 years at home

Significantly more depressed patients than controls had three or more children under 14 years of age living at home (17 vs 8, $P<0.05$) (Table II).

Depression in a first-degree relative

Significantly more depressed patients than controls had a first-degree relative treated for depression (77 vs 30, $P<0.0001$) (Table II).

Early parental loss

The detailed data about early parental loss because of permanent separation or death has been previously

TABLE I
Demographic data and clinical variables

	Non-endogenous depressed patients (n=300)	*Endogenous depressed patients (n=44)*	*Controls for endogenous patients (n=46)*
Female	207 (69%)	37 (84%)	28 (61%)
Male	93 (31%)	7 (16%)	18 (39%)
Married	155 (51.7%)	31 (71%)	29 (63%)
Single	91 (30.3%)	4 (9%)	13 (28%)
Separated/divorced/widowed	54 (18%)	9 (20%)	4 (9%)
Middle-class	171 (57%)	34 (77%)	42 (91%)
Working-class	129 (43%)	10 (23%)	4 (9%)
Out-patients	202 (67.3%)	3 (7%)	46 (100%)
In-patients	98 (32.7%)	41 (93%)	
Mean age in years	33.7 (range 18 to 65)	45.4 (range 26 to 73)	38.2 (range 21 to 78)
Mean duration of depression in months	5.2 (range 1 to 24)	9.2 (range 1/2 to 33)	
Mean Hamilton depression score	16.5 (range 9 to 30)	26.1 (range 15 to 37)	

TABLE II

Risk factors in patients with non-endogenous depression compared to normal controls

	Women			*Men*			*Total*		
	Depressed (n=207)	*Controls (n=207)*	*Sig. P*	*Depressed (n=93)*	*Controls (n=93)*	*Sig. P*	*Depressed (n=300)*	*Controls (n=300)*	*Sig. P*
Unemployed before onset of depression	85	50	<0.0004	19	7	<0.009	104	57	<0.0001
Poor marriage before onset of depression	69/104	8/104	<0.0001	25/35	5/35	<0.0001	94/139	13/139	<0.0001
Three children under 14 years of age	12	7	NS	5	1	NS	17	8	<0.05
Depression in a first-degree relative	57	21	<0.0001	20	9	<0.02	77	30	<0.0001
Parental death before 17 years of age	30	23	NS	9	9	NS	39	32	NS
Permanent separation from parent(s) before 17 years of age	42	14	<0.0001	24	5	<0.0003	66	19	<0.0001
Non-permanent separation from parent(s) for at least one year or more before 17 years of age	33	8	<0.0001	9	2	NS	42	10	<0.0001

TABLE III

Risk factors in patients with endogenous depression compared to normal controls

	Total groups			*Groups matched for age, sex, and marital status*		
	Depressed (n=44)	*Controls (n=46)*	*Sig. P*	*Depressed (n=27)*	*Controls (n=27)*	*Sig. P*
Unemployed before onset of depression	19	7	<0.007[1]	9	3	NS[1]
Poor marriage before onset of depression	15/31	4/29	<0.009	10/20	3/20	<0.04
Three children under 14 years	2	5	NS	2	4	NS
Depression in first-degree relative	19	NA[2]		14	NA[2]	
Parental death before 17 years of age	5	5	NS	3	3	NS
Permanent separation from parent(s) before 17 years of age	2	1	NS	2	1	NS
Non-permanent separation from parent(s) for at least one year or more before 17 years of age	1	2	NS	1	1	NS

Mean age of matched depressed patients, 20 female and 7 male, was 41.3 years and of controls 40.6 years; six patients and four controls were working class.

[1] Fourteen of the total 46 and 9 of the 27 matched controls were hospital employees.

[2] NA is not applicable. Controls were selected for biological studies thus those with a family history of psychiatric disorder were excluded. However, 30 of the 300 non-endogenous controls (10%) had a depressed first-degree relative (Table II) and if 10% of these controls had such a relative there would be significant differences.

published (Roy, 1983, 1985). Significantly more depressed patients than controls had permanent separation from a parent before 17 years of age (66 vs 19, $P<0.0001$) (Table II). In 14 patients (4.7%) this was separation from both parents compared with three controls (1%) ($P<0.01$). There were no significant differences for early parental death.

Significantly more depressed patients than controls had non-permanent separation of or from parents for at least one year or more before 17 years of age (42 vs 10, $P<0.0001$). Sixteen of these 42 depressed patients and three of these control patients also had experienced a permanent loss (either parental death or permanent parental separation). Thus only 26 of the depressives with non-permanent loss had experienced no permanent loss as had seven of these ten controls ($P<0.001$).

The linear model analysis revealed no significant interactions between any of the five factors in any group.

Endogenous depression

Unemployment before onset of depression

There was no significant difference in the matched comparison.

Poor marriage before onset of depression

Significantly more depressed patients than controls in both total and matched groups had a poor marriage before the onset of depression ($P<0.009$ and $P<0.04$, respectively) (Table III).

Three or more children under 14 years at home

There were no significant differences (Table III).

Depression in a first-degree relative

Nineteen of the total 44 (43.2%) and 14 of the 27 (51.9%) matched depressed patients had a first-degree relative treated for depression. However, their controls were selected for biological studies and those with a family history of psychiatric disorder were excluded (Table III).

Early parental loss

There were no significant differences for either parental death or permanent or non-permanent separation before 17 years of age (Table III).

Discussion

This final study reports on 344 depressed patients and 346 controls seen over 9 years by one investigator. Significantly more non-endogenous depressed patients than controls were unemployed and had a poor marriage before the onset of depression, had a family history of depression, had three children under 14 years of age at home, and had experienced permanent separation from a parent before 17 years of age. Only two of these five factors, poor marriage before the onset of depression and a family history of depression, were found significantly more among patients with endogenous depression than controls.

Being unemployed before the onset of depression was significantly associated only with patients with non-endogenous depression. Unemployment was high in both British and Canadian depressed patients (39.3% and 32.9%). Significantly more of both British and Canadian female depressed patients were unemployed as were significantly more male depressed patients. In the total group of patients unemployment was significantly associated only with the working class, and was found in 47.3% of them. Tennant *et al* (1982) found that being employed was highly protective for the mental health of both men and women.

A poor marriage before the onset of depression was found in significantly more of both non-endogenous and endogenous depressed patients. It was found in 67.6% of married non-endogenous and 48.4% of married endogenous depressed patients—percentages similar to the 69.2% of neurotic and 36.0% of endogenous depressed patients reported by Birtchnell & Kennard (1983a,b) to have marital maladjustment preceding the onset of depression. Other studies have also found that a poor marriage was associated with non-endogenous depression (Weissman & Paykel, 1974; Paykel *et al*, 1980; Kumar & Robson, 1984; Watson *et al*, 1984; Hallstrom & Persson, 1984; D'Arcy & Siddique, 1984). Assortative mating and the stresses induced by recurrent episodes of affective disorder may also play a part in causing a poor marriage (Brodie & Leff, 1971; Hoover & Fitzgerald, 1981; Merikangos, 1984).

Quinton *et al* (1976) developed the marital interview assessment technique used here. They, and others, found that marital ratings can be performed reliably. Goering *et al* (1986) examined whether expressed marital dissatisfactions and criticisms were merely reflections of the depressed state but found no correlation between severity of depression and the patient's report of the premorbid marital relationship, but there was a strong correlation between premorbid marital ratings obtained from separate interviews with depressed patients and their spouses. Crowther (1985) also found highly significant correlations between depressed patients and spouses for their scores on the Locke-Wallace Marital Adjustment Test and between depressed patient's scores and their therapists rating of marital adjustment. Similarly Waring & Patton (1984) reported general agreement between spouses and depressed patients about the degree of marital intimacy. Thus these studies suggest that depressed patients are able to give a reasonably undistorted account of their premorbid marital relationship.

Having three or more children under 14 years of age at home was significantly associated only with non-endogenous depressed patients—but was found in only 5.7% of them. Brown *et al* (1975) identified this risk factor in depressed working-class women in London, and Campbell *et al* (1983) reported similarly from Oxford. Tennant *et al* (1982) found that having young children at home was not directly associated with psychiatric disorder but through the effect on unemployment, as having young children prevented married women being employed.

A first-degree relative treated for depression was found in more of both non-endogenous and endogenous depressed patients. Weissman *et al* (1982, 1984) also found a familial aggregation of non-bipolar depression in first-degree relatives of non-bipolar depressed patients when compared with controls. In the present study the percentage of patients with a depressed first-degree relative was higher among endogenous than non-endogenous depressed patients (43.2% vs 25.7%) which conforms with the view that the evidence for genetic transmission is less clear for non-endogenous depression than for endogenous depression (Kidd & Weissman, 1978). However, another interpretation of these data is that families transmit a depression-proneness culturally rather than genetically, and that when, as with non-endogenous depressions, current circumstances as well as past family culture contribute to onset, the effect of the latter will be diluted by the former.

Early parental loss because of permanent separation from a parent before 17 years of age (but not because of death) was significantly associated only with patients with non-endogenous depression (discussed in Roy, 1985). Non-permanent separation for at least one or more years was also significantly associated with the non-endogenous depressed patients, as found by Brown *et al* (1977). However, among endogenous depressed patients there were no significant differences, compared to controls, for either early permanent or non-permanent separation or death.

Social risk factors for depression may best be identified from studies of general population samples rather than from studies of the minority of depressives who are referred to hospital psychiatrists. Thus, it is possible that the differences in the present study between endogenous and non-endogenous depressed patients and their respective controls are not genuine differences but the product of differences in the 'filters' determining who consults a doctor, whether that doctor refers the patient to a psychiatrist and whether the psychiatrist admits the patient to hospital. Also, the distinctions found in the present study between the non-endogenous patients (from London and Toronto) and the endogenous patients (from NIMH in Maryland) may be confounded by national, cultural and temporal differences. However, all the depressed patients were matched with controls from the same country, and DSM-III criteria were used by one investigator which prevented study variables (e.g. job loss, marital disharmony) influencing diagnoses of non-endogenous or endogenous depression. Furthermore, the endogenous sample contains significantly more middle-class patients than the non-endogenous sample and this social class difference may possibly confound interpretations of the endogenous/non-endogenous distinctions reported here.

In summary, all five factors studied were significantly associated with non-endogenous depression: unemployment before onset, a poor marriage before onset, separation for one year or more from a parent before 17 years of age, having a first-degree relative treated for depression, and having three or more children under 14 years of age at home. No significant interactions were found. Patients with endogenous depression were found to have two of these five factors: a poor marriage before onset and depression in a first-degree relative. Thus the present study suggests that three social factors - unemployment before onset, separation for one year or more from a parent before 17 years of age and having three or more children under 14 years at home - are risk factors for non-endogenous depression but that depression in a first-degree relative and a poor marriage before the onset of depression are found in both non-endogenous and endogenous depression.

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References

BIRTCHNELL, J. & KENNARD, J. (1983a) Does marital maladjustment lead to mental illness. *Social Psychiatry*, **18**, 79–88.

—— & —— (1983b) Marriage and mental illness. *British Journal of Psychiatry*, **142**, 193–198.

BRODIE, K. & LEFF, M. (1971) Bipolar depression—a comparative study of patient characteristics. *American Journal of Psychiatry*, **127**, 1086–1090.

BROWN, G. & HARRIS, T. (1978) *The Social Origins of Depression*. London: Tavistock Publications.

——, BHROLCHAIN, M. & HARRIS, T. (1975) Social class and psychiatric disturbance among women in an urban population. *Sociology*, **9**, 225–254.

——, Harris, T. & Copeland, J. (1977) Depression and loss. *British Journal of Psychiatry*, **130**, 1–18.

Campbell, E., Cope, S. & Teasdale, J. (1983) Social factors and affective disorder: an investigation of Brown and Harris' model. *British Journal of Psychiatry*, **143**, 548–553.

Crowther, J. (1985) The relationship between depression and marital adjustment. *Journal of Nervous and Mental Disease*, **173**, 227–231.

D'Arcy, C. & Siddique, C. (1984) Social support and mental health among mothers of preschool and school age children. *Social Psychiatry*, **19**, 155–162.

Diagnostic and Statistical Manual of Mental Disorders, 3rd edn. (1980). Washington DC: American Psychiatric Association.

Endicott, J. & Spitzer, R. (1979) A diagnostic interview: the schedule for affective disorders and schizophrenia. *Archives of General Psychiatry*, **35**, 837–844.

Goering, P., Lancee, W. & Freeman, S. (1986) Expressed emotion and recovery from depression. *Canadian Journal of Psychiatry*, Submitted.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. London: Oxford University Press.

Goldthorpe, J. H. & Hope, K. (1974) *The Social Grading of Occupations—A New Approach and Scale*. London: Oxford University Press.

Grizzle, J., Starmer, C. & Koch, G. (1969) Analysis of categorical data by linear models. *Biometrics*, **25**, 489–504.

Hallstrom, T. & Persson, G. (1984) The relationship of social setting to major depression. *Acta Psychiatrica Scandinavica*, **70**, 327–336.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

Hoover, C. & Fitzgerald, R. (1981) Marital conflict in manic-depressive patients. *Archives of General Psychiatry*, **38**, 65–67.

International Classification Of Diseases (1965) Eighth Revision. Geneva: World Health Organization.

Kidd, K. & Weissman, M. (1978) Why we do not yet understand the genetics of affective disorders. In *Depression: Biology, Psychodynamics, and Treatment* (J. Cole, A. Shatzberg, & S. Frazier, eds). New York: Plenum Press.

Kumar, R. & Robson, K. (1984) A prospective study of emotional disorders in childbearing women. *British Journal of Psychiatry*, **144**, 35–47.

Merikangos, K. (1984) Divorce and assortative mating among depressed inpatients. *American Journal of Psychiatry*, **141**, 74–76.

Paykel, E., Emms, E., Fletcher, J. & Rassaby, E. (1980) Life events and social supports in puerperal depression. *British Journal of Psychiatry*, **136**, 339–346.

Quinton, D., Rutter, M. & Rowlands, O. (1976) An evaluation of an interview assessment of marriage. *Psychological Medicine*, **6**, 577–586.

Roy, A. (1978) Vulnerability factors and depression in women. *British Journal of Psychiatry*, **133**, 106–110.

—— (1981*a*) Vulnerability factors and depression in men. *British Journal of Psychiatry*, **138**, 75–77.

—— (1981*b*) Risk factors for depression in Canadian women. *Journal of Affective Disorders*, **3**, 65–70.

—— (1983) Early parental death and adult depression. *Psychological Medicine*, **13**, 861–865.

—— (1985) Early permanent separation and adult depression. *Archives of General Psychiatry*, **42**, 987–991.

Tenant, C., Bebbington, P. & Hurry, J. (1982) Female vulnerability to neurosis: the influence of social roles. *Australian and New Zealand Journal of Psychiatry*, **161**, 135–140.

Waring, E. & Patton, E. (1984) Marital intimacy and depression. *British Journal of Psychiatry*, **145**, 641–644.

Watson, J., Elliott, S., Rugg, A. & Brough, D. (1984) Psychiatric disorder in pregnancy and the first postnatal year. *British Journal of Psychiatry*, **144**, 453–462.

Weissman, M. & Paykel, E. (1974). *The Depressed Woman: A Study of Social Relationships*. Chicago: University of Chicago Press.

——, Kidd, K. & Prusoff, B. (1982) Variability in rates of affective disorders in relatives of depressed and normal probands. *Archives of General Psychiatry*, **39**, 1397–1403.

——, Gershon, E., Kidd, K., Prusoff, B., Leckman, J., Dibble, E., Hamovit, J., Thompson, D., Pauls, D. & Guroff, J. (1984) Psychiatric disorders in the relatives of probands with affective disorders. *Archives of General Psychiatry*, **41**, 13–21.

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The Effect of Sodium Valproate on Tardive Dyskinesia – Revisited

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As a treatment for tardive dyskinesia, sodium valproate was tested in a double-blind placebo-controlled parallel group trial, with 6-week base-line observation period followed by 6 weeks of treatment. Sodium valproate was not found to be an effective treatment for either tardive dyskinesia or drug-induced Parkinsonism, and did not affect mental state or behaviour.

At present, there is no satisfactory or uniformly effective treatment for tardive dyskinesia (TD) (Mackay & Shepherd, 1979; Jeste & Wyatt, 1982); the initial success of many treatment strategies has not been maintained on replication (Jeste & Wyatt, 1979). There are difficulties in defining TD and in assessing it with respect to both reliability and validity (Gardos *et al*, 1977); Barnes & Trauer, 1982). This and the possibility that there are a number of neuropharmacological subtypes of TD (Casey, 1976; Fann *et al*, 1977; (Moore & Bowers, 1980) complicate an already confusing array of trial data.

Nevertheless, there has been growing evidence for the usefulness of drugs facilitating γ-aminobutyric acid (GABA) in the treatment of TD (Jeste & Wyatt, 1979, 1982). Although the GABAergic system is not fully determined in neuroanatomical terms, it is thought that GABAergic striato-nigral neurones modulate the activity of dopaminergic nigro-striatal neurones (Kelly & Beart, 1975), and interplay with extra-pyramidal and limbic cholinergic systems (McGeer & McGeer, 1976). It has been found that GABA facilitators inhibit dopamine (DA) turnover and impulse flow (Andén & Stock, 1973; Walters *et al*, 1973; Andén & Wachtel, 1977). The findings of reduced concentrations of GABA and of the synthesising enzyme glutamic acid decarboxylase (GAD) in the caudate nucleus, putamen, globus pallidus and substantia nigra in Huntington's chorea and in Parkinson's disease (Lakke & Teelken, 1976; McGeer & McGeer, 1976; Enna *et al*, 1977), together with reports of reduced GABA receptor binding in the former (Lloyd *et al*, 1977), have helped inspire research into the effects of GABA facilitators in TD. Attempts have been made to correct a postulated relatively hypo-GABAergic balance between DA and GABA systems.

Sodium valproate (SV), considered to be a GABA facilitator, has been the subject of a number of clinical trials, but its mechanism of action is still the subject of research. The evidence that SV enhances the concentration of GABA in the brain, inhibiting the breakdown of GABA by inhibition of GABA transaminase, has recently been updated by reports that this drug enhances the sensitivity of GABA post-synaptic membrane receptors (Hammond *et al*, 1981; Meldrum & Chapman, 1983).

However, the evidence for the efficacy of SV provided by past clinical trials is equivocal: the first trial that claimed it to be of value fell short of critical standards of significance (Linnoila *et al*, 1976). Gibson's (1978) single-blind trial, using unskilled raters with no valid or reliable instrument for measuring TD, obtained negative findings. A further single-blind trial used a cross-over design comparing three drugs (Chien *et al*, 1978): the findings were equivocal and interpretation was complicated by the small number of patients. Nair *et al* (1980) found SV ineffective when used on its own, but effective when combined with small (and, by themselves, ineffective) doses of haloperidol, but the small numbers and single-blind design made this trial also unsatisfactory. Casey & Hammerstad (1979) obtained a positive result in a single-patient study. More recently, SV has been found to exert an antidyskinetic effect in eight patients considered to have TD, in a single-blind study attempting to look at the drug's effect on akathisia (Friis *et al*, 1983), but the result was not significant. The methodological short-comings of these trials to date has left the question of the efficacy of SV still inadequately answered.

We have aimed to overcome some of these methodological inadequacies. The particular focus of attention in this trial was whether SV is an effective treatment for TD in a chronically psychotic population maintained on antipsychotic drugs. Discontinuation of antipsychotic medication before the trial was not considered, since it would not have been a desirable clinical step for most of the patients. We were particularly interested in an antidyskinetic treatment which could be effective when used alongside existing antipsychotic medication, in the manner that antiParkinsonian agents are used for drug-induced Parkinsonism. We also wanted to avoid the methodological problem of discontinuation of antipsychotic medication prior to the trial effecting *per se* (Gardos *et al*, 1978).

Method

Patients

This was a double-blind placebo-controlled parallel group study. Patients were drawn for three mental hospitals: Pastures and Kingsway in Derby, and Mapperley in Nottingham. Patients with chronic functional psychotic illness and diagnosis of at least 6 months of TD, defined by a score of two or more on the central portion of the Barnes & Kidger (1979) rating scale, were included in the study if they were under the age of 70, had been maintained for at least 1 year on antipsychotic drugs, were physically fit, without evidence of liver disease, and with absence of acute or chronic organic brain syndromes (including dementias, tumours, epilepsy, cerebrovascular lesions, demyelinating disorders). Informed consent was obtained.

Design

Trial entrants were randomised between SV and placebo treatment groups, with stratification for age, sex, hospital, in-patient or community-based status, and high or low pretrial TD, using a computer program giving these factors differential weightings. (High TD was defined as a trial score of five or more on the Barnes & Kidger scale.) Double-blind procedures were used throughout.

Medication was prepacked and labelled with the patient's name. There were two dose regimes: either a stepwise build-up over 6 days to a fixed dose of 1500 mg/day, or for patients weighing less than 50 kg, a similar build-up to 1200 mg/day. Adjustments to this final dosage were made for some patients because of side-effects. Maintenance antipsychotic medication, together with any other existing maintenance psychotropic or antiparkinsonian medication, was continued and kept constant throughout the trial.

Hospital pharmacists acted as coordinators of treatment dispensation with emergency access to treatment allocation. Compliance was monitored by records of tablet returns.

The trial lasted for 12 weeks, consisting of a 6-week base-line period, followed by 6 weeks of treatment. The base-line period was included because it was considered appropriate to control for the effect of attention *per se* in this group of patients, which invariably receives comparatively little attention. Assessment were carried out at 0, 6, 9 and 12 weeks.

Assessment

Patients were assessed, using the Barnes & Kidger rating scale (Kidger *et al*, 1980), the Simpson & Angus (1970) scale for extrapyramidal side-effects and the Krawiecka *et al* (1977) assessment scale for rating mental state and behaviour in chronic psychotic patients. The Barnes & Kidger scale gives a total TD score and also a central and peripheral score. In addition to a 'live' TD rating, each patient was videotaped simultaneously, as described by Barnes & Kidger (1979) and by Kidger *et al* (1980), the only modification being the replacement of a portion of standard prose by that of the daily newspaper. Thus, each patient was videotaped in a standard environment and was given a standard task. The videotapes were used to provide two further blind TD ratings by experienced independent raters.

In addition to the rating scales, a standardised question concerning side-effects was used, and physical examination performed, as appropriate. At the first assessment, venepuncture was performed for liver function tests, full blood count, urea and electrolytes, and was repeated after 3 weeks of, and at the end of, the treatment phase together with serum SV levels. All medication administered was monitored.

Analysis

Comparisons between groups made use of the Mann–Whitney *U* test on either actual scores or change scores; within-group comparisons used the Wilcoxon matched pairs signed ranks test. Contingency tables based on operational definitions of clinical change were evaluated using χ^2 and the Fisher exact probability test. Clinically significant change in TD was defined, using the Barnes & Kidger scale, as a change of two or more on the central subscale or two or more on the peripheral subscale with one on the central. Clinically significant change in extrapyramidal symptoms was defined as a change in score of six or more on the Simpson & Angus scale. Clinically significant change in mental state was defined as a mental state score of three or more using the Krawiecka scale. Change in each case was measured from the base-line score average to the final assessment. For inter-rater reliability, the Kendall coefficient of concordance was employed.

Results

Patients

Sixty-two patients entered the trial (Table I). They had an average age of 54 years, with ten under the age of 50, 21 between 50 and 60, and 31 between 60 and 70. A total of 49 were maintained on anticholinergic antiparkinsonian medication and 14 on benzodiazepines. A check on randomisation showed no significant difference between SV and placebo treatment groups for age, sex, in-patient *vs* community-patient status, diagnosis, duration of antipsychotic treatment, maintenance medication level and type of medication.

During the base-line period, three patients were withdrawn because of ill-health, and a further patient on SV for 2 weeks was withdrawn because of pneumonia. Nine patients defaulted or failed to comply (five on SV, four on placebo) and in two patients, background medication was unstable. Therefore, 15 patients failed to complete the trial.

The profile of the remaining 47 patients is shown in Table I. There were no significant differences between this group and trial entrants for age, sex, placement, diagnostic distribution, duration of pre-trial antipsychotic medication, or current medication; 22 of these patients were allocated to SV, 25 to placebo, and a further randomisation check revealed no significant profile differences.

Inter-rater reliability

Concordance between two 'live' ratings, carried out on a sample of 25 patients, for TD (total, central and peripheral scores), extrapyramidal symptoms and the mental state was significant. (Kendall coefficient of concordance: $P<0.001$, respectively.)

TABLE I
Profile of the population studied

	Trial entrants (n=62)	*Trial completers (n=47)*	*Sodium valproate (n=33)*	*Placebo (n=29)*
Age: years				
Mean	54	58	58.1	57.6
s.d.	8.86	8.27	8.83	9.05
Sex: *n*				
M	42	34	21	21
F	20	13	12	8
Status: *n*				
In-patient	31	27	17	14
Community	31	20	16	15
Diagnosis: *n*				
Schizophrenia	56	43	32	24
Manic-depressive	3	3	1	2
Recurrent depressive	3	1	0	3
Duration of antipsychotic medication: years				
Mean	15.75	15.88	17.4	14.6
s.d.	7.95	8.1	7.51	7.76
Antipsychotic dosage: *n*				
<300 mg/day (Chlorpromazine equivalents)	21	18	12	9
300–900 mg/day (Chlorpromazine equivalents)	29	18	17	12
>900 mg/day (Chlorpromazine equivalents)	12	11	4	8

Concordance between 'live' and the two video-ratings for TD (total, central and peripheral scores), based on the trial population, was significant (Kendall coefficient of concordance: $P<0.005$, respectively). This allowed these three ratings to be reduced to the one 'modal' score as described by Barnes & Kidger (1979).

Results of treatment

Sodium valproate and placebo groups were compared; first, using all scores available for comparison at each assessment point and, secondly, confining the analysis to the scores of all 47 patients completing the trial.

Tardive dyskinesia

All scores

There were no significant differences between the assessments at the beginning and at 6 weeks, within either the SV or placebo group. On the first assessment, the SV group had significantly higher scores than had the placebo group for central scores (Mann–Whitney: $P=0.02$), with a similar trend for total and peripheral scores failing to reach significance. Although there were no significant differences between treatment groups after 6 weeks, because of the disparity found initially, the remaining analyses were carried out on change scores.

Between the 6-week assessment, at which point the different treatments were introduced, and the 9-week assessment, both the SV and placebo groups improved non-significantly, with no significant difference in the score changes between them. There were no significant differences in score changes between the 6- and 12-week assessments (i.e. the complete period of treatment), but there was a trend towards improvement in placebo relative to SV for total TD scores, nearly reaching significance (Mann–Whitney: $P=0.06$). From the half-way treatment point, at 9 weeks, to 12 weeks, the score changes between treatment groups differed significantly for total scores (Mann–Whitney: $P=0.05$) and central scores (Mann–Whitney: $P=0.01$). This reflected the SV group's greater tendency to return to base-line scores.

Completers (Fig. 1)

There were no significant differences between SV and placebo initially or at 6 weeks, except for significantly lower central scores for the placebo initially (Mann–Whitney: $P=0.02$). There were no differences at 9 weeks (i.e. 3 weeks after treatment commenced). At 12 weeks, placebo had significantly lower total scores (Mann–Whitney: $P=0.03$) and central scores (Mann–Whitney: $P=0.05$). Score changes were used again because of the slight base-line disparity. Between 6 and 12 weeks, the period of treatment, change scores for SV and placebo groups were significantly different for central scores (Mann–Whitney: $P=0.02$), but this just failed to achieve significance for total scores (Mann–Whitney: $P=0.06$); this reflected a relative improvement in the placebo group. While score changes between 6 and 9 weeks did not differ significantly, between 9 and 12 weeks, a greater tendency to return to base-line scores in the SV group resulted in significant differences for total scores (Mann–Whitney: $P=0.03$) and central scores (Mann–Whitney: $P=0.01$).

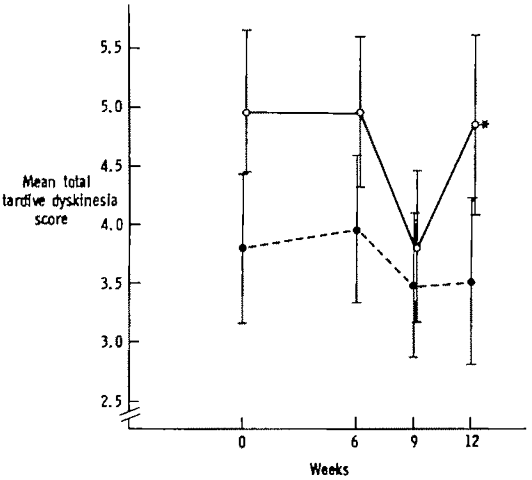

FIG 1. Total tardive dyskinesia scores for those completing the trial on sodium valproate (O——O) and placebo (●——●). The asterisk indicates that the score change was significantly different from that for placebo between weeks 9 and 12 ($P=0.03$).

Seven patients showed clinically significant improvements (four on SV, two on placebo) and eight patients showed deterioration (six on SV and two on placebo). There was no significant trend, but a trend towards no change, whether improvement or deterioration, being associated with placebo; this failed to reach significance. ($\chi^2=3.77$; $P>0.05$).

Extrapyramidal symptoms

There were no significant differences within treatment groups during the base-line period, and no differences between SV and placebo groups during the base-line period. At the 9- and 12-week assessments, placebo had significantly lower scores than had SV (Mann–Whitney: $P=0.03$ and 0.012, respectively). This held for all scores available for comparison at each assessment point and for the 47 completers. Within the SV group, there was no evidence of deterioration during treatment.

During the trial, three patients improved clinically, two of them being on SV, while one patient on SV deteriorated. These trends did not reach significance.

Mental state

There were no differences between base-line assessments within each treatment group, or between treatment groups. There were no significant differences between SV and placebo during treatment. Three patients improved clinically, all of whom were on SV; three patients deteriorated, two on SV and one on placebo. These trends did not achieve significance.

Serum sodium valproate

There was no significant association between clinical outcome for TD and serum SV levels.

Side-effects

Twenty-one patients reported side-effects. Of these, 11 complained of sedative side-effects; seven were on SV and four on placebo. The sedation was mild, and not clinically noticeable in all but one 69-year old chronic schizophrenic in-patient on SV, who experienced heavy sedation, ataxia and faecal incontinence after 10 days of SV. All side-effects disappeared when the dose was cut to 500 mg daily. In four patients, three of whom were on SV, reduction of dosage to 500 mg *b.i.d.* successfully removed mild sedative side-effects.

Ten patients reported mild non-sedative side-effects, from headache to irritability; six of these were on SV. One patient self-administered SV erratically and became restless and socially disruptive. He was found to have a serum SV level of 1074 μmol/litre and was withdrawn from the trial.

There was no overall trend towards side-effects, whether sedative or non-sedative, being significantly associated with SV. There was no association between serum levels of SV and side-effects. There was no trend towards abnormal blood tests with SV.

Subcomponents of tardive dyskinesia

Finally, we used our data to investigate whether central and peripheral TD score changes within patients on SV were significantly different. Using score changes in the treatment period, we found no significant difference.

Discussion

After 6 weeks of SV treatment, there was no evidence of any improvement in TD: the non-significant improvement found after 3 weeks of SV was not significantly different from that found for placebo and was not maintained, unlike that for placebo. This effect after 3 weeks of SV is of interest, because a similar trend was noted by Chien *et al* (1978) in their single-blind cross-over trial. The findings of Linnoila *et al* (1976) of a non-statistically significant improvement in TD after 2 weeks in a double-blind cross-over trial may also be due to this effect. This suggests that the positive results from their trials are a transient placebo effect.

The findings at the end of the trial – that placebo did significantly better than SV did, both in terms of direct comparison and in terms of relative change from the pretreatment base-line – underline the ineffectiveness of SV; TD was relieved more successfully by placebo.

The improvement in Parkinsonian-type extrapyramidal symptoms, found after 3 and 6 weeks of placebo, was not produced with SV. Although Parkinonism was relieved more successfully by placebo, there was no tendency for SV to exacerbate Parkinsonian symptoms, which is broadly in line with the trial carried out by Friis *et al* (1983).

The suggestion has been made by Lautin *et al* (1980) that SV can cause a deterioration in chronic schizophrenic symptoms; however, their patient numbers were small, the trend could not be disentangled from the effect of neuroleptic discontinuance and the results did not achieve significance. We used a similar dose range, and found no evidence of improvement or deterioration in mental state. There have been reports of GABA facilitators being sedative, particularly in the elderly (Gerlach, 1977; Gerlach *et al*, 1978; Casey *et al*, 1980), and even the suggestion that their efficacy in TD is secondary to such sedation, but we have not confirmed this in the population we studied. However, there is the possibility that this reflects our younger age group, and that the elderly may be more sensitive.

This trial provided additional evidence for the usefulness of the Barnes & Kidger rating scale for TD. The levels of concordance between 'live' ratings, and between 'live' ratings and two independent videoratings, both for total scores and for central and peripheral scores, support its reliability. The significant association between score changes for central and peripheral TD during treatment adds weight to the view that oro-facial and peripheral subsyndromes of tardive dyskinesia, while separable, are related phenomena.

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References

Andén, N. E. & Stock, G. (1973) Inhibitory effect of gamma-hydroxybutyric acid and gamma aminobutyric acid on dopamine cells in the substantia nigra. *Naanyn — Schmiedeberg's Archives of Pharmacology*, **297**, 89–92.

—— & Wachtel, H. (1977) Biochemical effect of baclofen on the dopamine and the noradrenaline in the rat brain. *Acta Pharmacologia et Toxicologia*, **40**, 310–320.

Barnes, T. R. E. & Kidger, T. (1979) Tardive dyskinesia and the problems of assessment. In *Current Themes in Psychiatry* (eds R. N. Gaind & B. L. Hudson) Vol II. London: Macmillan.

—— & Trauer, T. (1982) Reliability and validity of a tardive dyskinesia videotape rating technique. *British Journal of Psychiatry*, **140**, 508–515.

Casey, D. E. (1976) Tardive dyskinesia: are there subtypes? *New England Journal of Medicine*, **295**, 1078.

—— & Hammerstad, J. P. (1979) Sodium valproate in tardive dyskinesia. *Journal of Clinical Psychiatry*, **40**, 483–485.

——, Gerlach, J. Magelund, G. & Rosted Christensen, T. (1980) Gamma-acetylenic GABA in tardive dyskinesia. *Archives of General Psychiatry*, **37**, 1376–1379.

Chien, C., Jung, K. & Ross-Townsend, A. (1978) Efficacies of agents related to GABA, dopamine and acetylcholine in the treatment of tardive dyskinesia. *Psychopharmacology Bulletin*, **14**, 20–22.

Enna, S. J., Stern, L. Z., Wastek, G. J. & Yammura, H. J. (1977) Neurobiology and pharmacology of Huntington's disease. *Life Sciences*, **20**, 205–212.

Fann, W. E., Stafford, J. R., Malone, R. L., Frost, J. D. & Richman, B. W. (1977) Clinical research techniques in tardive dyskinesia. *American Journal of Psychiatry*, **134**, 759–762.

Friis, T., Rosted Christensen, T. & Gerlach, J. (1983) Sodium valproate and biperiden in neuroleptic-induced akathisia, Parkinsonism and hyperkinesia *Acta Psychiatrica Scandinavica*, **67**, 178–187.

Gardos, G., Cole, J. O. & La Brie, R. (1977) The assessment of tardive dyskinesia. *Archives of General Psychiatry*, 34, 1206–1212.

——, —— & Tansy, D. (1978) Withdrawal syndromes associated with antipsychotic drugs. *American Journal of Psychiatry*, **135**, 1321–1324.

Gerlach, J. (1977) The relationship between Parkinsonism and tardive dyskinesia. *American Journal of Psychiatry*, **134**, 781–784.

——, Rye, T. & Kristjansen, P. (1978) Effect of baclofen on tardive dyskinesia. *Psychopharmacology*, **56**, 145–151.

Gibson, A. C. (1978) Sodium valproate and tardive dyskinesia. *British Journal of Psychiatry*, 133, 82.

Hammond, E. J., Wilder, B. J. & Bruni, J. (1981) Central actions of valproic acid in man and in experimental models of epilepsy. *Life Sciences*, **29**, 2961–2574.

Jeste, D. V. & Wyatt, R. J. (1979) In search of treatment for tardive dyskinesia: review of the literature. *Schizophrenia Bulletin*, 5, 251–293.

—— & —— (1982) Therapeutic strategies against tardive dyskinesia. *Archives of General Psychiatry*, 39, 803–816.

Kelly, J. S. & Beart, P. M. (1975) Amino acid receptors in the CNS. II: GABA in supraspinal regions. In *Handbook of Psychopharmacology* (eds. L. L. Iverson & S. H. Synder) Vol. 4. New York: Plenum Press.

Kidger, T., Barnes, T. R. E., Trauer, T. & Taylor, P. J. (1980) Subsyndromes of tardive dyskinesia. *Psychological Medicine*, **10**, 513–520.

Krawiecka, M., Goldberg, D. & Vaughan, M. (1977) A standardized psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

Lakke, J. P. W. & Teelken, A. W. (1976) Amino acid abnormalities in the cerebrospinal fluid of patients with parkinsonism and extrapyramidal disorders. *Neurology*, **26**, 489–493.

Lautin, A., Angrist, B., Stanley, M., Gershon, S., Heckl, K. & Karabath, M. (1980) Sodium valproate in schizophrenia: some biological correlates. *British Journal of Psychiatry*, **137**, 240–244.

Linnoila, M., Viuka, M., Hietala, O. (1976) Effect of sodium valproate on tardive dyskinesia. *British Journal of Psychiatry*, **129**, 114–119.

Lloyd, K., Dreksler, S. & Bird, E. (1977) Alterations in H-GABA binding in Huntington's chorea. *Life Sciences*, **21**, 747–754.

Mackay, A. V. P. & Shepherd, G. P. (1979) Pharmacotherapeutic trials in tardive dyskinesia. *British Journal of Psychiatry*, **135**, 489–499.

McGeer, P. L. & McGeer, E. G. (1976) Enzymes associated with the metabolism of catecholamines, acetylcholine and GABA in human controls and patients with Parkinson's disease and Huntington's chorea. *Journal of Neurochemistry*, **26**, 65–76.

Meldrum, B. S. & Chapman, A. G. (1983) The mechanism of action of sodium valproate: recent advances. *British Journal of Clinical Practice*, Supplement 27, 136–141.

Moore, D. C. & Bowers, M. B. (1980) Identification of subgroup of tardive dyskinesia patients by pharmacologic probes. *American Journal of Psychiatry*, 137, 1202–1205.

Nair, N. V. P., Lal, S. & Guyda, H. (1980) Effects of GABAergic drugs on nigrostriatal and hypothalamic pituitary dopaminergic systems. *Brain Research Bulletin*, **5** (Supplement 2), 427–431.

Simpson, G. M. & Angus, J. W. S. (1970) A rating scale for extrapyramidal side effects. *Acta Psychiatrica Scandinavica*, **212**, 11–19.

Walters, J. R., Roth, H. H. & Aghajanian, G. K. (1973) Dopaminergic neurones: similar biochemical and histological effects of gammahydroxytbutyrate and acute lesions of the nigrostriatal pathways. *Journal of Pharmacology and Experimental Therapeutics*, **186**, 630–639.

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Brief Reports

Cousin Marriages and Schizophrenia in Saudi Arabia

The rate and degree of consanguinity in the parents of 143 schizophrenics who satisfied the DSM-III diagnostic criteria, was compared in the same number of controls matched for age, sex and socioeconomic class. A family history of disorders suggestive of schizophrenia in the offspring of consanguineous parents who were schizophrenic, was compared with the incidence of a similar history in the schizophrenic offspring of non-consanguineous parents. There was no statistically significant difference in the former, but there was in the latter. This finding supports the theory of a familial tendency towards schizophrenia and the possibility of recessive or a multigene pattern of inheritance.

The study of genetic theories of schizophrenia can be divided into three categories: the incidence of schizophrenia relative to the degree of consanguinity in relatives of known patients; the concordance rate in twins and studies on adopted twins.

Since Rüdin's report in 1916, there have been numerous similar studies but using increasingly more sophisticated methods: Schultz in 1932, Kallman in 1938, Zerbin-Rüdin in 1967 and Slater in 1968. All reported a significantly higher prevalence of schizophrenia in the families of schizophrenic patients than in the families of non-schizophrenics, but their studies only show that schizophrenia is a familial trait and not that it is necessarily genetic.

Twin studies by Tienari in 1963, Gottesman & Shields in 1966, Kringlen in 1967, Pollin *et al* in 1969, and Fischer in 1973, reveal a three-fold increase in the monozygotic concordance rate as compared with the dizygotic rate. This is not unexpected because when the lifestyles of twins are examined closely, there is frequently found a more intense bond between monozygotic twins than between non-twin siblings or dizygotic twins (Jackson, 1960). This argument, however, has been countered by schizophrenic twins reared apart.

The Danish adoption studies by Kety *et al* in 1975, which reported a significantly greater prevalence of schizophrenic spectrum disorders among the relatives of schizophrenic adoptees than among those of non-schizophrenic adoptees, certainly support the genetic theory of schizophrenia. However, Benjamin (1976) criticised the statistical analysis used in that study, and indicated that an alternative method of analysis leads to a conclusion that violates the principle of genetic effect increasing with greater consanguinity. So Kety's studies may be flawed, but the sequel to them, the current Danish adoption study of Jacobsen, is finding the same results.

The Arabian culture provides the opportunity to investigate this matter from a different angle, that of the rate of consanguinity of parents of schizophrenics compared with that of controls. Only three similar studies are found in the literature. The Sudanese study (Ahmed, 1979) used a large number of subjects but was very brief, did not include data from family histories, did not differentiate between acute, remitting cases and chronic cases, and there were no well-defined diagnostic criteria. Another study was done in Azerbaijan, Russia (Skoblo, 1975) and indicated an increased frequency of sporadic schizophrenic patients in comparison with the average; the abstract did not give the statistical significance of this difference. The study is in Russian and not available for comment. In the third study, by Prasad in 1985, only 15 schizophrenic patients were compared with patients with affective disorders. The author said that the inference of the results should be accepted guardedly, and that more controlled studies are needed.

The nature of the study presented here is such that it will not resolve the argument of familial versus inherited nature of schizophrenia, but it will add to the data available and help to correct a deficiency in the medical literature.

Method

During one year, 143 randomly selected patients who satisfied the DSM-III diagnostic criteria for schizophrenia, were interviewed individually by the two authors. Patients with a diagnosis of schizophreniform psychosis, schizo-affective disorder, or affective disorder were not included in the study. Complete demographic data were taken; also data were obtained on type and duration of illness, number of stays in hospital and out-patient visits, dosage of neuroleptic drug used to maintain control; and history of schizophrenia in siblings, parents, and siblings of parents identified by the patients or family by similarity of signs and symptoms and the requirement of long-term psychiatric care. The degree of parental consanguinity was accurately tabulated into marriage of first cousins, second cousins, and distant relatives; the later included marriage between members of the same tribe and was called third-degree consanguinity.

TABLE I
Occurrence of schizophrenia in families of cousin-marriages and non-consanguineous marriages

Degree of consanguinity in patients' parents	*No family member affected*	*One family member affected*	*Two family members affected*	*Three or more family members affected*	*Total*	*Control*
First cousins	12	7	3	1	23	17
Second cousins	6	4	2	1	13	10
Distant cousins	26	3	2	1	32	15
Non-consanguineous	56	10	7	2	75	101
Total	100	24	14	5	143	143

Saudi Arabia maintains no public records of birth, marriages and deaths, therefore there are no official data available on the rate of consanguinity in the general population. Therefore, the companions of the patients, who were unrelated but of the same socioeconomic class and who attended the clinic with the patients, were asked the same questions as the patients and thus comprised the control group in this study.

Results

Of the 143 patients studied, 105 were males and 38 were females; their ages ranged from 16 to 70 years, with a mean of 31.6 years and standard deviation of 11.0 years. There was the same number of controls and they were matched for age, sex and socioeconomic class. Twenty-three patients were the offspring of first-cousin marriages, 13 were the offspring of second-cousin marriages, 32 were of distant-cousin marriages and 75 reported no parental consanguinity. The respective numbers for the controls were 17, 10, 15 and 101 (Table I). Although these figures indicate a higher rate of parent–consanguinity among the patients than among the controls, a statistical significance was not demonstrated ($\chi^2=1.65$, $P=0.20$). Of the 75 patients with no parental consanguinity, 56 had no family history suggesting a schizophrenic disorder, 10 had one family member (family member for this purpose is defined as sibling, parent or sibling of parent only) who was affected, seven had two family members who were affected, and two had three or more family members who were affected. Of the 32 patients who were the offspring of distant cousins, the distribution of family history suggestive of schizophrenia was 26, 3, 2 and 1 respectively, and for the 13 who were the offspring of second cousins was 6, 4, 2 and 1 respectively and for the 23 offspring of first cousins was 12, 7, 3 and 1 respectively. The scatter of these figures was too wide to give a statistically significant conclusion.

However, combining the patients from distantly related parents with those of no consanguinity, and combining patients who were the offspring of first- and second-cousin marriages, an interesting, statistically significant, conclusion is reached (Table II). The offspring of first and second cousins are far more likely to have a positive family history suggestive of schizophrenia than similar patients who are the offspring of distantly related and non-consanguineous parents ($P=0.005$).

TABLE II
Occurrence of family history of schizophrenia in families of close and distant (including non-cousin) cousin marriages

	With negative family history	*With positive family history*	*Total*
Distant cousin or non-consanguineous marriages	82	25	107
First and second cousin marriages	18	18	36

$\chi^2=7.866$, d.f. $=1$, $P=0.0059$

Several attempts were made to identify the demographic differences of these two subgroups; the type of schizophrenia e.g. paranoia, undifferentiated or simple; the chronicity and severity of illness identified by unremitting status, duration of more than 10 years, more than three stays in hospital and unemployment. In none of these parameters was there a statistically different distribution in either of the subgroups.

Discussion

Intermarriage was once a matter of pride and honour among the Bedouin tribes (Hasanien, 1984) but it has declined considerably in inverse proportion to their rising socioeconomic status in recent years. Even today, however, there are families who would in no circumstances let their women marry out of the family; this would be demeaning and show a disregard for their blood line. Hence it was important to have a carefully matched control for this study.

We found there was no statistically different rate of intermarriage between the parents of the patient group and the parents of the control group, although the rate was relatively higher in the former. It is possible that the result is affected by the small size of the sample groups; however, our results are in agreement with the Sudanese study (Ahmed, 1979), but not with the Russian study (Skoblo, 1975).

The finding of a significantly higher rate of positive family history in patients of consanguineous parents than in the other groups of patients, indicates a definite familial tendency of schizophrenia and possibly an inherent pattern of a multiple gene type, since it is well known that in-breeding will promote the appearance of a multi-gene trait of recessive character. Our data support the genetic theory of schizophrenia. The fact that neither the type nor the clinical course of schizophrenia have taken any specific pattern in this study, conforms with the concept that schizophrenia is single disorder with various clinical courses and differing presentation.

In conclusion, although this study does not resolve the argument of familial versus hereditary nature of schizophrenia, it does support the later and provide an evidence to suggest association with recessive genetic disorders, where two copies of any aberrant gene are required for expression of the disorder, or a polygene nature of the disease.

References

AHMED, A. H. (1979) Consanguinity and schizophrenia in Sudan. *British Journal of Psychiatry*, **134**, 635–636.

BENJAMIN, L. S. (1976) A reconstruction of the Kety and associates' study of genetic factors in the transmission of schizophrenia. *American Journal of Psychiatry*, 133, 1129–1133.

FISCHER, M. (1973) Genetic and environmental factors in schizophrenia. *Acta Psychiatrica Scandinavica*, (suppl.), **238**, 9–142.

GOTTESMAN, I. I. & SHIELDS, J. (1966) Schizophrenia in twins: 16 years' consecutive admissions to a psychiatric clinic. *British Journal of Psychiatry*, **112**, 809–814.

HASANIEN, M. M. (1984) *Beduin Sociology*. Riyadh, Kingdom of Saudi Arabia: Okadh Publishing.

JACKSON, D. D. (1960) A critique of the literature on the genetics of schizophrenics. In *The Etiology of Schizophrenia*, (ed. D. D. Jackson). New York: Basic Books.

KALLMAN, F. J. (1938) *The Genetics of Schizophrenia*. New York: Augustin.

KETY, S. S., ROSENTHAL, D., WENDER, P. H., SCHULSINGER, F. & JACOBSEN, B. (1975) Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic: a preliminary report based on psychiatric interviews. In *Genetic Research in Psychiatry*, (ed. R. R. Fieve, D. Rosenthal & H. Brill). Baltimore: Johns Hopkins University Press.

KRINGLEN, E. (1967) *Heredity and Environment in the Functional Psychoses*. London: Heinemann.

POLLIN, W., ALLEN, M. G., HOFFER, A., STABENAU, J. R. & HRUBEC, Z. (1969) Psychopathology in 15,909 pairs of veteran twins. *American Journal of Psychiatry*, **126**, 597–602.

PRASAD, A. J. (1985) First cousin marriages and psychiatric morbidity. *Canadian Journal of Psychiatry*, **30**, 69–70.

RUDIN, E. (1916) *Zur Vererbung und Neuentstehung der Dementia Praecox*. Berlin: Springer.

SCHULTZ, B. (1932) Zurt Erbpathologie der Schizophrenia. *Zeitschrift fuer die Gesamte Neurologie und Psychiatrie*, **143**, 175–179.

SKOBLO, G. V. (1975) Inbreeding and the distribution of schizophrenia in a rural area of Azerbaijan (Russia). *Genetika*, **11**, 149–152.

SLATER, E. (1968) A review of earlier evidence on genetic factors in schizophrenia. In *The Transmission of Schizophrenia* (ed. D. Rosenthal & S. S. Kety). Oxford: Pergamon Press.

TIENARI, P. (1963) Psychiatric illnesses in identical twins. *Acta Psychiatrica Scandinavicam* (Suppl. 171), 1–195.

ZERBIN-RUDIN, E. (1967) Endogene Psychosen. In *Humangenetik, ein kurzes Handbuch*, (ed. P. E. Becker). Stuttgart: Thiéme.

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Electrodermal Response as a Monitor in Electroconvulsive Therapy

A study comparing the use of electrodermal response with the cuff technique and clinical judgment as monitors in electroconvulsive therapy is reported. A sensitivity of 90.5% and a specificity of 41.7% were found and it is, therefore, suggested that this method is not used routinely.

The administration of electroconvulsive therapy (ECT) in the UK was reported on in 1980 (Pippard & Ellam, 1980) with some disturbing findings. It is generally accepted that a convulsion is required for the therapeutic benefit (Kendell, 1982), but it is not always obvious to those administering ECT that a convulsion has, in fact, occurred. Various methods have been used to determine this although observation for convulsive muscular movements seems the most common. The use of EEG monitoring is the surest way to measure seizures, but both EMG and the cuff technique have also been widely used (Fink, 1983). In the cuff technique (Addersley & Hamilton, 1953) a sphygmomanometer is inflated to above systolic blood pressure before the administration of suxamethonium. This is left on during the ECT and that arm subsequently has an unmodified seizure. This method of monitoring has the advantage of being simple, safe and universally available. Other measures used have been dilatation of pupils (Addersley & Hamilton, 1953), ECG (Goffen, 1984) and electrodermal response (EDR). The latter was reported in a small study (Watkins-Pitchford & Reynolds, 1984) comparing EDR on 15 administrations of ECT with clinical twitching of the muscles, which is the unreliable measure under question. Therefore a study was designed to compare the sensitivity and specificity of the EDR with the cuff technique whose validity has been demonstrated (Fink & Johnson, 1982).

Method

The EDR was measured using the simple apparatus described by Watkins-Pitchford & Reynolds (1984). Two electrodes on non-adjacent fingers were connected to the apparatus. A light-emitting diode (LED) was then illuminated by adjusting a pre-set resistor. This was turned down until the light was just extinguished to give the pre-ECT baseline. A successful convulsion increased the skin resistance and re-illuminated the LED. The cuff technique was performed on the other arm with the cuff kept at 260 mmHg from before the suxamethonium was administered to after the seizure had finished.

Forty-eight patients consented and 117 administrations of ECT were monitored. All were given bilateral ECT using an Ectron Duopulse constant current apparatus. Anaesthesia was induced using methohexitone; suxamethonium and atropine were also given. After preoxygenation and insertion of a mouthpiece, ECT was given. The administering psychiatrist noted whether or not a tonic-clonic convulsion had occurred. The convulsive response of the isolated arm and the EDR change were recorded. The psychiatrist was working blind regarding this last measure.

Results

Interestingly we found that the EDR was positive in nearly all patients during initial current application. A positive response was, therefore, only recorded if a subsequent EDR occurred sufficient to illuminate the LED after the current had ceased. This coincided with the expected convulsion.

On three occasions the cuff technique was negative when there was an obvious bilateral convulsion in the rest of the body. This must have been due to poor technique and shows limitations of this method. Since the results of the cuff technique are highly correlated with the EEG method of monitoring (Fink & Johnson, 1982), we decided to use the cuff technique, and clinical observation to be the measure of convulsion occurrence.

The results using this method are displayed in Table I. Of 105 patients who had a convulsion, a positive EDR showed in 95 of them. This gives a sensitivity of 90.5%. Of the 12 patients who did not have a convulsion, seven showed a positive EDR. This gives a specificity of 41.7%.

TABLE I

EDR compared with a combination of the cuff technique and clinical judgment on the occurrence of convulsions

	Cuff and clinical	
EDR	*Convulsion occurred*	*Convulsion not occurred*
Convulsion occurred	95	7
Convulsion not occurred	10	5

Discussion

The EDR is not a simple measure as it is determined by many variables (Venables & Christie, 1980). In this study a simple apparatus has been used as a gross indicator of change in skin resistance (illumination of LED). Skin resistance is determined by the sympathetic nervous system's stimulation of sweat glands which have cholinergic post-ganglionic innervation. As atropine is given intravenously it will presumably block this effect as it has been shown to do when given by iontophoresis (Lader, 1970). It is, therefore, not surprising that the use of EDR has been shown in this study to have poor validity. The cuff technique, in contrast, is simpler and has been shown previously to correlate well with the EEG (Fink & Johnson, 1982). In this study only three patients (2.6%) showed a discrepancy between the clinical and cuff technique and this must have been due to the patients' blood pressure rising above 260 mmHg during the convulsion. The clinical opinion in this study took the cuff technique into consideration as the psychiatrists were not working blind and this is the reason for the high level of agreement between them.

It is suggested from this study that the EDR is not yet a useful technique to monitor the occurrence of convulsions during the ECT and that the cuff technique should, therefore, be used until further development has taken place.

References

ADDERSLEY, D. J. & HAMILTON, M. (1953) Use of Succinylcholine in ECT. *British Medical Journal*, 1, 195–197.

FINK, M. (1983) Missed seizures and the bilateral-unilateral electroconvulsive therapy controversy. *American Journal of Psychiatry*, 140, 198–199.

—— & JOHNSON, L. (1982) Monitoring the duration of Electroconvulsive Therapy Seizures. 'Cuff' and EEG Methods Compared. *Archives of General Psychiatry*, 39, 1189–1191.

GOFFEN, B. S. (1984) ECT Seizure Monitoring. *Archives of General Psychiatry*, 41, 106.

KENDELL, R. E. (1982) The Present Status of electroconvulsive therapy. *British Journal of Psychiatry*, 139, 265–283.

LADER, M. H. (1970) The unit of quantification of the GSR. *Journal of Psychosomatic Research*, 14, 109–110.

PIPPARD, J. & ELLAM, L. (1980) *Electroconvulsive Treatment in Great Britain*. London: Headley Brothers.

VENABLES, P. H. & CHRISTIE, M. J. (1980) Electrodermal Activity. In *Techniques in Psychophysiology* (ed. P. H. Venables & M. J. Christie). Chichester: John Wiley & Sons.

WATKINS-PITCHFORD, J. M. & REYNOLDS, F. (1984) Autonomic Monitor for Electroconvulsive Therapy. *British Journal of Psychiatry*, 145, 551–554.

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A Schoolgirl with Onset of Anorexia Nervosa during a Concealed Pregnancy

In anorexia nervosa, amenorrhoea and endocrine disturbance make pregnancy unlikely while the patient's weight is low. Bulimics may conceive at quite a low weight, and anorexics who have regained weight may become pregnant but typically relapse following delivery (Crisp, 1980). Anorexia has also been reported following termination of pregnancy (Thomas & Harris, 1982). Cases of onset of anorexia nervosa with weight loss during pregnancy are rare in the literature, although there are isolated reports (e.g. Weinfeld *et al*, 1977; Strimling, 1984). Strimling described a treated anorexic who restarted dieting during pregnancy in response to weight gain before she realised that she was pregnant. We report on a schoolgirl who became pregnant at 14 years, whose anorexia began when she was 5 months pregnant and whose pregnancy was not discovered until she was in labour.

Young schoolgirls tend to delay seeking advice about pregnancy (Russell, 1983). In a series of patients requesting late termination of pregnancy in Tower Hamlets, Savage (1985) found that the major reason for late presentation was the youth of the patients, often associated with denial of pregnancy. Denial of pregnancy is described (Finnegan *et al*, 1982) as sometimes extending to (and beyond) delivery. It may involve the patient's relatives and doctors. It may prejudice the mother's acceptance of the baby after birth.

Case history

The patient 'Gill' was an only child of a shop assistant. Her birth weight was 2.33 kg at 37 weeks gestation. Her mother developed a post partum embolus resulting in a 2-week separation from Gill and advice against further pregnancy.

Gill's early years were unremarkable, but her later friendships were restricted by her parents; she remembers being locked in a garage to prevent her playing with neighbourhood children. She and her friend were known as 'Laurel and Hardy' because Gill was plump. The menarche occurred before she left primary school. At 11 years 7 months her weight was 50.5 kg (over the 97th Percentile) and her height was 146 cm (over the 50th Percentile).

She failed the 11-plus examination and was unhappy at her secondary school. She was seen by a child psychiatrist at 14 years, giving a history of depressive symptoms for 1 year and weighing 63.5 kg. She was treated with dothiepin for a month but this was discontinued when she developed 'fits' which were not thought to be epileptic. They rapidly ceased and her mood improved.

When she returned to school, parents and head-teacher agreed that a girlfriend was 'unsuitable' and discouraged the friendship. Gill formed a friendship with a boyfriend 'John' and when she was 14 years 9 months they had sexual intercourse once. Two further periods occurred before menstruation ceased. At 15 years 1 month (4 months pregnant) she was taken by mother to the GP complaining of headaches and vomiting. Gill estimates that at this time she weighed between 57 and 64 kg. She wondered if she was pregnant, but thought this impossible because her periods had continued after intercourse. She concealed the amenorrhoea from her GP who diagnosed migraine. After being teased at school, she decided to diet, weighed herself twice daily and lost 0.45 kg daily whilst vomiting but later up to 1.82 kg per week when the vomiting stopped. She has said 'I put the possibility of pregnancy to the back of my mind', and was convinced that if she lost weight she could not be pregnant. When she was 6 months pregnant her parents felt that her mood had improved. She cooked for others but only ate one small kipper daily and drank orange juice. She pretended to eat her meals but secretly fed them to the dog. A month later the school became concerned at her loss of weight and tried to persuade her parents to take her to their GP, but father claimed she was eating well. At a school medical inspection when she was 8 months pregnant, the doctor suspected anorexia nervosa and thought Gill was depressed and anxious. The pregnancy was not noted. She weighed 49 kg, said that her periods were regular and refused to see her GP.

She was upset because she had gained 0.45 kg and took no more solid food until she presented in Casualty 3 days later with abdominal pain. She weighed 46.3 kg (height 154.9 cm). She was in the second stage of labour and was delivered by forceps of a healthy female child, weighing 2.4 kg, estimated maturity 37 weeks. Gill and her parents denied all knowledge of the pregnancy and the baby was fostered at the parents' request.

Taking the lowest estimate of original weight, 57 kg and allowing for an average expected weight gain in pregnancy of 10.9 kg, Gill had probably lost about 32% of expected body weight prior to her delivery. She was transferred to an Adolescent Psychiatric Unit. She weighed 43.64 kg, was refusing carbohydrate and fat, and considered herself too fat, especially around the hips and thighs. She remained resistant to a feeding programme, was weight phobic and took exercise whenever possible. While Gill was an in-patient, the relationship with John continued and he spent most of his free time in her parents' house. After treatment, with individual and family therapy, Gill was discharged after being prescribed oral contraceptives, at 15 years 7 months weighing 46.8 kg.

Two months after discharge, when the adoption of the baby was completed, Gill weighed 50 kg. Six months later she started to diet, made herself vomit and took laxatives. She denied bingeing and weighed 47.73 kg on readmission. She was grieving the loss of her child and hostile towards her parents for insisting on adoption. She was unable to accept her own ambivalence about keeping the baby. She became engaged to John on the baby's first birthday, took CSE exams, obtained work as a shop assistant and was discharged at 16 years 8 months weighing 50.45 kg. Three months later her Eating Attitudes Test score (EAT 40, Garner & Garfinkel, 1979) was 91 (anorexic range more than 30) and her weight was 54.09 kg. At 17 years 2 months, her parents accused John of disrupting their family and Gill left home to live with John's parents. Gill's mother became depressed and sought psychiatric help.

Two months later Gill presented to an ante-natal clinic weighing 65.4 kg. The couple moved to a rented flat and Gill gained weight steadily during pregnancy to 81.3 kg. Her EAT 40 score fell to 8 in late pregnancy. She says that she 'wanted to tell everybody' about this pregnancy, unlike the previous one. She was delivered of a male child, weighing 4.06 kg, estimated maturity 40 weeks, and bottle fed the baby. She was noted to be eating all meals provided in hospital and contacted her parents to show them the child before her discharge.

At her post-natal examination Gill weighed 70.01 kg and the baby weighed 5.17 kg. Her EAT 40 score had risen to 50 and she was unhappy about her weight. She lost 2.73 kg in 2 weeks. She is now refusing to be weighed.

Discussion

At the end of her first pregnancy this patient fulfilled the DSM-III criteria for anorexia nervosa.

Why does anorexia nervosa rarely present for the first time during a pregnancy? Maternal protective feelings for the unborn child perhaps prevent pregnant women from dieting excessively. We note that in a case reported by Strimling (1984) the anorexic patient tried to resume more regular eating habits after the discovery of her pregnancy. Also, in pregnancy, weight gain is clearly anticipated and a pregnant woman may feel temporarily absolved from the pursuit of thinness. However, if the pregnancy is denied this may make anorexia nervosa psychologically possible.

The aetiology of anorexia nervosa is discussed by Hsu (1983). Individual and family theories both involve the concept of failure to complete adolescent developmental tasks. As the only child of over-controlling parents our patient had never established independence in her relationships. In common with many anorexics she was a plump child whose puberty came early, but she was not from the upper social classes, was of average intelligence and non-competitive and her parental expectations of academic achievement were not high. She first presented with depression, but the stress of her first sexual experience followed by pregnancy at a young age precipitated anorexia nervosa. The development of anorexic symptoms may have been assisted by the vomiting of pregnancy. The onset of anorexia with weight loss fostered the conviction that she was not pregnant and the weight gain of pregnancy disguised the anorexia.

Pregnancy normally necessitates maturational progress for a patient, and her family. The patient adopts a clearly defined adult role which involves some degree of separation from parents, who are themselves offered a new role as grandparents. Pregnancy in a young teenager results in the sudden, unprepared precipitation of the teenager into an adult role and of her parents into a different relationship with their child.

As this girl's first pregnancy was not acknowledged until delivery, these adjustments did not occur, and could have been unacceptable. The subsequent removal of the baby returned the family to their previous situation without evident maturational progress. The parents' resistance to separation involved incorporating the patient's boyfriend into the family as a second child when they failed to divide him from the patient. A second pregnancy, 2 years later, was conceived deliberately and the patient left home. Increasing maturity with age and therapeutic interventions may have contributed to the patient's ability to deal more normally with her second pregnancy but anorexic symptoms returned after delivery and neither her separation nor her eating problems are fully resolved.

Acknowledgement

We are grateful to Dr G. J. R. Richardson, Consultant Psychiatrist, Southfield Adolescent Unit, York.

References

Crisp, A. H. (1980) *Anorexia Nervosa: Let Me Be.* London: Academic Press.

Finnegan, P., McInstry, E. & Erlick Robinson, G. (1982) Denial of pregnancy and childbirth. *Canadian Journal of Psychiatry,* **27,** 672–674.

Garner, D. M. & Garfinkel, P. E. (1979) The eating attitudes test: an index of the symptoms of anorexia nervosa. *Psychological Medicine,* **9,** 273–279.

Hsu, L. K. G. (1983) Editorial, The aetiology of anorexia nervosa. *Psychological Medicine,* **13,** 231–238.

Russell, J. K. (1983) School pregnancies – medical, social and educational considerations. *British Journal of Hospital Medicine,* Feb. 159–166.

Savage, W. (1985) Requests for late termination of pregnancy: Tower Hamlets, 1983. *British Medical Journal,* **290,** 621–623.

Strimling, B. S. (1984) Infant of a pregnancy complicated by anorexia nervosa. *American Journal of Diseases of Children (Chicago),* **138,** 68–69.

Thomas, C. S. & Harris, B. (1982) Anorexia nervosa following termination of pregnancy (letter). *British Journal of Psychiatry,* **141,** 428.

Weinfeld, R. H., Dubay, M., Clay Burchell, R., Millerick, J. & Kennedy, A. (1977) Pregnancy associated with anorexia and starvation. *American Journal of Obstetrics and Gynaecology* Nov. 15, 698–699.

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'Imposed Psychosis'

A Case Variant of the Gaslight Phenomenon

In 1969 Barton & Whithead described the Gaslight Phenomenon. In this condition a presentation of mental illness is found, on further enquiry, to have been induced or imposed on the patient by some other person for that other person's gain. Amongst these gains is removal of the patient to a hospital. Previous reports have highlighted the malicious intent of the exploiter (Barton & Whitehead, 1969; Smith & Sinanan, 1972; Tyndal, 1973; Lund & Gardiner, 1977).

The phrase 'Gaslight Phenomenon' was derived by Barton & Whitehead (1969) from the play *Gaslight* (Hamilton, 1939). In that play the victim's husband manipulated the gaslight in a way which made his wife's complaint about it seem as if she was going insane. His aim in doing this was to remove her from the scene and gain for himself her property.

We wish to report on a case of imposed illness, but one in which the intent of the inducer was not primarily malicious.

Case descriptions

Mrs A is a 52-year-old widow who first presented in the Accident and Emergency Department. She expressed distress about the sudden disappearance, 3 months previously, of her 22-year-old daughter. On further enquiry she stated that over a period of 6 years her late husband had returned in spirit to the house. He had brought flowers for her and her daughter. He often rearranged the contents of the cupboards, at times criticising Mrs A for her unsatisfactory housework. On occasion he had struck Mrs A on the head and pushed her by the shoulder. He took her daughter's clothes out of the cupboard and laid them on the bed for her. He had daily engaged her and her daughter in conversations. At times he created a great deal of noise in the house by banging on doors and cupboards. Mrs A claimed that these events had also been witnessed by her daughter. However, since her daughter's disappearance her husband's spirit had not appeared again.

In her history Mrs A claimed that her daughter worked for famous private doctors. She stated that her daughter had spoken, on the doctors' car radios, with Prince Charles and Princess Diana, and that her daughter and Princess Diana were good friends. She claimed that her daughter had met Prince William when he was 6 weeks old.

Mrs A was presented at a psychiatric case conference 1 week after her admission. The general view expressed at the meeting was that her symptoms were suggestive of a psychotic illness, possibly paraphrenia. The day of her admission into hospital Mrs A commenced chlorpromazine in a dose of 50 mg 8-hourly, both in view of her mental state presentation and her level of distress.

In view of Mrs A's concern about her daughter an attempt was made to contact the daughter by letter through the DHSS. Concern by the staff had also been expressed about the daughter's mental state.

Two weeks after the DHSS enquiry the daughter, Miss B telephoned the hospital to enquire about Mrs A's health. She insisted, however, that Mrs A was not told of her whereabouts as she wanted to build up her own life away from home. She claimed that Mrs A had prevented her from leaving home and had stopped her from claiming unemployment benefit. In the year before she left home Miss B had begun to claim her unemployment pay, informing Mrs A that this money was from work she did for private doctors. To direct questioning, Miss B stated that her father's spirit had been in the house and that he had spoken to both Mrs A and herself. She also confirmed her mother's contention that he had rearranged various articles in the house.

During a further telephone conversation with Miss B she was asked about Mrs A's reference to Prince Charles, Princess Diana and Prince William. Miss B stated that she herself had never met them, but that she had told Mrs A that the doctor she worked for knew them. Miss B agreed that she may have conveyed the impression to Mrs A that she knew them personally as well. Miss B admitted that in fact she had never worked for private doctors.

Miss B was invited to attend the out-patient department for a fuller discussion of the situation, to which she agreed. At that meeting a detailed psychiatric history was obtained from Miss B and the opportunity was taken to elicit further information about Mrs A. Toward the end of the meeting Miss B was asked specifically about her father's spirit. Initially she reiterated that her father's spirit was there, and also added that on occasion he left notes of instruction to Mrs A. However, after being asked to confide in us and after being assured that what she told us would be held in the strictest confidence, Miss B suddenly became distressed and tearful: she then admitted that she had, over the years, been speaking in her father's voice to make it appear as if her father was talking to Mrs A and to herself. She also admitted rearranging things in the house and to leaving notes for Mrs A, ostensibly written by father. The following reasons were given by Miss B as to why she did these things: she was afraid of Mrs A and felt she could not cope with Mrs A by herself; she needed more freedom for herself from Mrs A and in the notes supposedly written by her father explanations were given for Miss B's absence from home during the day time.

Formal assessment of Miss B, using the Research Diagnostic Criteria (Spitzer *et al*, 1978) revealed no formal mental illness. She was of average intelligence, had an appropriate affect, and a good rapport was established with her.

An outline of Miss B's developmental history showed this to have been a highly stressful one. Her earliest childhood memories were of her parents shouting at each other, not infrequently this culminating in violence. This continued until her parents separated when Miss B was 8 or 9 years old. Her father died 2 years later of a brain tumour. Miss B stated that she cried after her father died, although before he died she had experienced feelings of anger toward him for the way he had treated Mrs A. However, her father had never mistreated her and after his death she would remember the holidays she used to spend with him. With regard to Mrs A, Miss B stated that after her parents separated Mrs A would often blame her for having prevented her from obtaining a job, despite many applications. When Miss B was 11 or 12 years old Mrs A began threatening to put her into a Children's Home because then she would be able to get a job and sell the house and move away if necessary.

With regard to Mrs A's own developmental history, she recalled that starting school and separating from her own mother was extremely difficult. Mrs A, as a child, was only able to remain at school with the reassuring knowledge that her mother would visit the school gates to see her at both break and lunch-time. She recalled how her father would do some of the housework at weekends because her mother was extremely tired. Her mother's fatigue was seen as further evidence of tireless dedication to attending and caring for her. At the age of 11 Mrs A refused to sit her 11-plus examination which both her elder sibs had passed. She recalled being terrified of failing, although she was unable to connect this with her mother's stated hopeful desire and hopeful expectation that her children should do well in life.

When Mrs A's daughter, Miss B, was born she was nursed in an incubator for the first 2 weeks of life, during which time Mrs A was constantly afraid that Miss B would die. Mrs A went to visit Miss B but was unable to hold her and recalled returning to her hospital room in tears. She never really understood what was wrong with her baby and has held a grudging resentment toward the medical profession for the way she was treated at that time.

Mrs A stated that when Miss B wished to leave home in her late teens she was totally unable to understand why she would want to do so, other than to marry or to find employment.

It is of particular importance that Mrs A's mother was a spiritualist who attended seances. During her late teens Mrs A would go with her mother to the spiritualist meetings where attempts were made to contact the dead.

Within a few days of her admission into hospital, Mrs A began to wonder whether what she had been told by her daughter was true, and she started to have doubts about the return of her husband's spirit. She was puzzled by the disappearance of her husband's spirit the moment her daughter left home. Mrs A's subsequent history has shown her to be free of all psychotic phenomena, but she remains angry and deeply embittered to the point of tears about Miss B not wanting to establish contact with her, though she had been informed that Miss B had telephoned the hospital on a number of occasions to ask the staff to reassure Mrs A that she was well.

Discussion

Mrs A's life history shows that she has had severe difficulties in the process of individuation and separation (Mahler, 1972). She experienced marked difficulty in starting school, and separating from her mother, who was idealised as being totally devoted to her, while her teachers were seen as frightening authority figures. Mrs A continues to idealise the mother/daughter relationship as one in which there is little or no room for separateness. Mrs A's object relations are typified by a rigid idealised self/object representation maintained by excessive splitting and projective identification (Klein, 1975*a*,*b*; Jacobsen, 1964). She only seems associated with idealised relationships. Feelings associated with the unsatisfactory side of her relationships are warded off, denied, or projected into others who are attacked as bad – with loss of any good or loving feelings for them. This is illustrated by her apparent absence of feeling when talking about her husband's behaviour prior to their separation; her husband apparently left her for another woman but Mrs A did not reveal this, such information being revealed by Miss B. Mrs A was totally unable to accept anything about her late husband which did not fit into her idealised view of him. She similarly described an absence of feeling for her mother who remarried late in life, but feelings of anger, resentment and sadness are denied.

Mrs A seems to have retained an ambitiousness throughout her life, something which she connected with her mother's desire that she should get on in life. She did not want to start a family as this would interfere with her own ambitions and financial goals in life. Following her husband's death she had wanted to start a new life by going out to work, but felt unable to do so because of Miss B. Here can be seen the nature of her envious rivalry with Miss B who she saw as potentially having a life denied herself. Currently she enviously attacks Miss B's independent life, with the resultant loss of any good feelings for her, though her attitude to Miss B throughout life has always been extremely ambivalent and the envious feelings have been denied (Klein, 1975*a*,*b*).

With regard to the belief that her husband was visiting the house from the grave, this would seem to serve the function of attempting to provide a solution to a complex dilemma for both Mrs A and Miss B. The 'voices' appear to provide both mother and daughter with comfort, serving as a defence for both of them against depressive anxieties to do with loss and separateness.

Two further factors seem to contribute toward understanding why Miss B invoked the idea of a return of her father's spirit. When Miss B was 14 years old Mrs A had mentioned that a spirit must be in the house because an article of clothing was in a place she had not placed it. Mrs A had thought that this spirit was either that of her own late mother (who had died the year before) or of her husband (who had died 4 years previously). The second factor was that Miss B had heard from a schoolfriend of that friend's experience of her own late mother's spirit rearranging things in the house and of the comfort which this spirit had brought to her.

References

BARTON, R. & WHITEHEAD, J. A. (1969) The gaslight phenomenon. *Lancet*, 1, 1258–1264.

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Consensus Statement: Panic Disorder

SIR: We wish to draw the attention of readers to a recent initiative concerning the topic of panic. In November 1986 an informal meeting was held in the UK to discuss anxiety and panic, with particular reference to diagnosis and classification. The participants, listed below, came from several academic and clinical centres in the UK and represented a wide range of expertise and specialist interests within this area of psychiatry. Various topics were outlined in order to focus the subsequent discussions.

The concept of anxiety neurosis was established nearly 100 years ago, and has emphasised at several times since the somatic, particularly cardiovascular, symptoms. 'Panic' originally implied collective and excessive fear reactions, but the term has come to have somewhat different psychiatric implications, stressing individual physiological response. Cardinal features are the rapid rate of onset of the attack and the feeling of loss of personal control. Recently, attempts have been made to separate panic and anxiety as diagnostic entities, both in the proposed ICD–10 and in the DSM–III classification system of the American Psychiatric Association.

The epidemiology of panic attacks and disorders varies according to the sample studied and the criteria used: population studies, surveys of general practice attenders, and out-patient studies provide very different prevalences of panic. In one American study, prevalences were: Generalised Anxiety Disorder 2.5%; Phobic Disorders 1.5%; and Panic Disorders 0.5% of the general population. One study suggests a low use of psychiatric services; others, a higher use of general practice services. Distinct diagnostic categories probably give arbitrary estimates of prevalence. An approach regarding severity of panic attacks as an important dimension gives more realistic estimates: for example, in a recent UK study occasional panics were present in 1 in 10, whereas frequent disabling panic attacks were reported by only 1 in 50 of the adult population. Alcohol can be involved in a vicious circle of panic leading to problem drinking leading to withdrawal leading to panic, although this is less common in the community than in out-patient psychiatric practice. The natural history of panic either as a cluster of symptoms occurring alone or forming part of another psychiatric illness has not been established.

Family studies show that anxiety neuroses are familial, and the data from twin studies suggest a genetic contribution. This is more obvious in Panic than in Generalised Anxiety Disorder, and this has led some to suggest a specific genetic contribution to the former but not the latter. However, more general genetic predispositions are likley to be important: personality, neuroticism, autonomic lability, and biochemical factors. Multifactorial or polygenic explanations may, therefore, fit the data better.

In the study of anxiety and panic, reliable measures are important, and self-reports must remain a key feature; several dimensions should be taken into account. These include severity, duration, and frequency of attacks as well as type, e.g. 'spontaneous' as opposed to 'situation-related' panic attacks. The diary method is very useful. Intensity is best assessed by the disruptive effects of the attack on normal on-going activities. Other aspects of the psychiatric condition should be rated, such as phobias and levels of generalised anxiety and depression. The natural history of the panic attacks should be documented, for example whether the attacks follow an episodic, cyclic, or clustered pattern.

Views on the psychological aspects of panic attacks currently focus on the cognitive hypothesis. This proposes a cycle in which a trigger stimulus, internal or external, gives rise to perceived threat, apprehension, and in particular to bodily sensations which are misinterpreted in a catastrophic fashion and thereby reinforce the perceived threat. These misinterpretations involve perceiving these sensations as indicating immediate and severe physical or mental harm. In cognitive therapy these misinterpreted bodily sensations are identified, and by suggesting and testing alternative non-catastrophic interpretations panic and anxiety levels are reduced. There is some evidence, stemming from studies on cognitive abnormalities, that panic can be differentiated from generalised anxiety disorders.

The bulk of morbidity in anxiety disorders is encountered in general practice, but there are problems with current diagnostic schemes. Patients may present with anxiety, phobic, panic, and depressive symptoms in various combinations. As symptom-clusters are not stable over time, there are often changes in appropriate diagnostic labels; somatisation is common, so that patients who experience panic attacks often complain of bodily rather than psychological symptoms. This may lead either to failure of recognition or inappropriate referral.

The treatment of panic disorders is varied. Most of the evidence comes from studies which included agoraphobic patients with panic attacks, and concurrent behavioural treatments were usually given. Tricyclic antidepressants, such as imipramine and clomipramine, have an established place in the prevention of panic attacks. Where panic occurs without significant depression some workers have evidence to support the use of low doses of antidepressant drugs, but others have suggested the use of high doses. There is also disagreement about the time of onset of the therapeutic effect, some workers reporting prompt onset, others noting substantial delays. Some panic patients appear very sensitive to unwanted effects of these drugs. Reponse at low doses may suggest a mechanism of action distinct from the antidepressant action seen at higher doses. Although improvement may be maintained for some time after stopping the drugs, over the longer term the outcome is more uncertain. A few benzodiazepines used in doses above the usual anxiolytic ones (e.g. alprazolam, clonazepam, diazepam) have been shown to prevent panic attacks; the evidence for this is most clear for alprazolam, and further studies are in progress. Such response to benzodiazepines is, however, less convincing than that to the tricyclic antidepressants, and there is a danger of dependence. Alternative drugs include the MAOIs and β-adrenoceptor blockers; the latter are of limited usefulness. All of these treatments can be used in conjunction with behavioural and cognitive therapies. Indeed, preliminary evidence suggests the efficacy of cognitive therapy alone in patients with panics. High consultation rates and limited time in general practice emphasise the need for simple, pragmatic therapies.

Panic attacks can be regarded as a cluster of symptoms which can occur alone or as part of another disorder which has its own place in the classificatory scheme. Controversy attends the nosological status of disorders in which the symptoms of panic occur in conjunction with other disorders (agoraphobia, generalised anxiety, or depression). In the American classification (Revised DSM–III) panic attacks are given primacy and are thought to delineate a separate diagnostic entity (called Panic Disorder). The proposed revision of the ICD uses the term 'Panic Disorder (episodic anxiety)' to describe a disorder in which panic attacks occur as the major feature in the absence of significant anxiety between attacks and a depressive disorder. The meeting preferred the latter convention, as it is unclear whether panic attacks which occur in association with significant generalised anxiety are best considered as a separate disorder or as a more severe form of generalised anxiety disorder.

The meeting also recognised that for research purposes the individual symptom clusters should be considered independently, and it urged longitudinal studies to clarify their natural history and outcome.

The consensus of the meeting was that it is useful descriptively to recognise the symptom cluster of panic attacks which can be defined operationally in terms of features and content. Frequency is a useful, albeit arbitrary, indicator of severity. It was recognised that patients are encountered in whom panic attacks occur in the absence of other significant symptoms. The status of panic disorder as a separate entity was not strongly supported by available clinical and scientific evidence. Finally, although some evidence suggested that panic attacks often represented a severe form of more generalised anxiety, it was premature to attempt to describe the relationships between the various symptom clusters.

Participants: G. W. Ashcroft *(Aberdeen)*; G. Beaumont *(Stockport)*; J. Bonn *(London)*; S. Brandon *(Leicester)*; A. Briggs *(Leicester)*; D. Clark *(Oxford)*; K. Davison *(Newcastle-upon-Tyne)*; M. G. Gelder *(Oxford)*; D. Goldberg *(Manchester)*; R. Herrington *(Glasgow)*; M. C. Khan *(Hartlepool)*; M. Lader, *Chairman (London)*; M. S. Lipsedge *(London)*; A. Macdonald *(London)*; P. Maguire *(Manchester)*; P. T. S. Milln *(Southampton)*; R. M. Murray *(London)*; R. F. Stirton *(Leicester)*; A. C. P. Sims *(Leeds)*; R. P. Snaith *(Leeds)*; D. Wheatley *(Hinchley Wood)*.

(See also pp 563–564)

Senile Dementia and Parietal Lobe Dysfunction

Sir: Gilleard *et al* (*Journal*, January 1987, **150**, 114–117) miss the point of the paper by McDonald (1969), whose findings they claim to have disproved. Since the latter's hypothesis was essentially a predictive one which was vindicated by follow-up and confirmed by others (Naguib & Levy, 1982) it is difficult to see how a purely cross-sectional study with no follow-up can be strictly relevant to the issue. Furthermore, Gilleard *et al* claim that McDonald did not exclude cases suggestive of a vascular aetiology, whereas the 1969 paper states that such exclusion did occur: "If there was a history of strokes or epileptic seizures or if there were focal neurological signs the patients were excluded as being suspect arteriosclerotic dementias".

It is true that age and age of onset may no longer be regarded as reliable predictive factors in view of the somewhat contradictory results reported by different authors (Huff *et al*, 1987), but the sinister prognostic significance of visuo-spatial dysfunction must be considered as one of the most robust findings in the clinical literature on Alzheimer's disease. Its relationship to dysphasia and other focal features on the one hand and to overall severity on the other is a complex one which my group is trying to unravel in the course of a prospective study.

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References

HUFF, F. J., GROWDON, J. H., CORKIN, C. & ROSEN, R. J. (1987) Age of onset and rate of progression of Alzheimer's disease. *Journal of the American Geriatrics Association*, **35**, 27–30.

MCDONALD, C. (1969) Clinical heterogeneity in senile dementia. *British Journal of Psychiatry*, **115**, 267–271.

NAGUIB, M. & LEVY, R. (1982) Prediction of outcome in senile dementia – a computed tomography study. *British Journal of Psychiatry*, **140**, 263–267.

Gluten Sensitivity in Schizophrenia

SIR: Singh & Kay (*Journal*, January 1987, **150**, 130–131) make telling points in their criticism of our study of gluten sensitivity in schizophrenia, most of which we have already made and accept. We hardly perceived ours as the critical experiment; such experiments are difficult to perform in any science and very difficult in clinical psychiatry. They are usually misreported simplifications of the history of science. Indeed, we experienced, as others will, considerable problems in getting psychotic people to co-operate convincingly in accepting dietary controls, and so the attempt, which is what we report, was of short duration and in a special hospital. We certainly don't wish to deter others from performing more adequate work, but in our limited study the major changes occurred in the patients before they had a gluten-free period. We were pleased that, fortuitously perhaps, the *Journal* published Singh & Kay's letter after one by Wing suggesting "very impaired patients" were "not impervious to social stimulation", as seemed obvious in our study.

In addition, though, we are blamed for not considering the heterogeneity of the syndrome, while in fact we discussed it and concluded that gluten-free diets may be of value to some schizophrenics. We could go on at length over details of interpretations, but perhaps it would be wisest to concede that Singh and Kay have done an immense amount of work in this field, which we took seriously enough to examine and to try to confirm. Hence we are sorry if our inability to support them in our short-term and necessarily imperfect study can be read as wishing to dismiss their efforts and their approach completely. Far from it – we want, with them, to see what other studies can demonstrate, but we needed to report what we found.

We note, though, that Singh & Kay have written several similar letters criticising other peoples' failures to confirm their hypothesis (Singh, 1979; Singh & Kay, 1983).

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References

SINGH, M. M. (1979) Celiac-type diets in schizophrenia. *American Journal of Psychiatry*, **136**, 733.

SINGH, M. M. & KAY, R. S. (1983) Study of gluten effects in schizophrenia. *Archives of General Psychiatry*, **40**, 345.

Sub-cortical Dementia

SIR: In his review of sub-cortical dementia (*Journal*, December 1986, **149**, 682–697) Cummings makes the statement: "Sub-cortical dementias are characterised by psychomotor retardation, whereas the cortical dementias" (among which he includes Alzheimer's disease) "manifest a normal psychomotor speed through most of the clinical course", and supports this with reference to Cummings & Benson (1986). Although it is true that this view has become something of an orthodoxy in neurological texts, it flies in the face of much evidence from other sources. For instance, it is clear that on the Digit Copying Test (DCT) component of the Kendrick Battery (Kendrick *et al*, 1979) groups of patients with dementia predominantly of the Alzheimer type were significantly slower than non-demented subjects. Evidence of slowing also comes from other sources using quite different techniques. Thus slowing is also found in Alzheimer patients when they are asked to identify pictures presented tachistoscopically (Neville & Folstein, 1979) or when they carry out tasks using a peg-board (Miller, 1977). This issue is discussed by Woods (1982), who suggests that a critical point may be the degree of cognitive load in terms of choices available. Another important variable which may be seen as a form of cognitive load is the delay involved before the subject is expected to respond. In a study using a computerised visual matching to sample task with variable delay

(Morris *et al*, 1987; Sahakian *et al*, 1987) we found that patients with relatively early Alzheimer disease only reacted as rapidly as controls when an immediate response was required (i.e. with a zero second delay). The data show that when the test stimulus is delayed the Alzheimer patients respond a good deal more slowly than the age-matched controls, and that in this respect they perform far worse than recently diagnosed patients with Parkinson's disease who showed much smaller differences from their own controls on this task.

We suggest that psychomotor speed is a complex variable, and that contrary to belief it is not a useful way of differentiating between cortical and subcortical dementia.

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References

CUMMINGS, J. L. & BENSON, D. F. (1986) Dementia of the Alzheimer type: an inventory of diagnostic features. *Journal of the American Geriatrics Society*, **34**, 12–19.

KENDRICK, D. L., GIBSON, A. J. & MOYES, I. L. A. (1979) The revised Kendrick Battery: clinical studies. *British Journal of Social and Clinical Psychology*, **18**, 329–340.

MILLER, E. (1977) *Abnormal Ageing: The Psychology of Senile and Presenile Dementia*. Wiley: New York.

MORRIS, R. G., EVENDEN, J. L., SAHAKIAN, B. J. & ROBBINS, T. W. (1987) Computer-aided assessment of dementia: comparative studies of neuropsychological deficits in Alzheimer-type dementia and Parkinson's disease. In *Cognitive Neurochemistry* (eds S. M. Stahl, S. D. Iversen & E. C. Goodman). Oxford: Oxford University Press. (In press).

NEVILLE, H. J. & FOLSTEIN, M. F. (1979) Performance on three cognitive tasks by patients with dementia, depression or Korsakov's syndrome. *Gerontology*, **25**, 285–290.

SAHAKIAN, B. J., MORRIS, R. G., EVENDEN, J., ROBBINS, T. W., HEALD, A. & LEVY, R. (1987) A comparative study of visuospatial memory and learning deficits in Alzheimer-type dementia and Parkinson's disease (in preparation).

WOODS, R. (1982) The psychology of ageing. In *The Psychiatry of Late Life* (eds R. Levy & F. Post). Oxford: Blackwell Scientific Publications.

Dementia in Parkinson's Disease

SIR: Oyebode *et al* (*Journal*, December 1986, **149**, 720–725) present data about dementia in 43 patients with Parkinson's disease, and draw conclusions concerning the prevalence of this. Their population was drawn from patients attending an illustrious neurological centre. Perhaps the area served contains patients with Parkinson's disease who were not referred to the centre, but were looked after by general practitioners, geriatricians, or psychiatrists. There might have been a tendency to fail to refer the bedridden, the incontinent, and the demented.

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Anorexia Nervosa or Dysmorphophobia?

SIR: An alternative diagnostic formulation is possible (*Journal*, December 1986, **149**, 780–782). As Hay (1970) points out, dysmorphophobia is a non-specific symptom often indicative of a 'sensitive personality development', or occasionally early schizophrenia. The phenomenological distinction between the overvalued idea and the delusion depends in part on external evidence of physical abnormality and adverse environmental experience, often in the form of teasing in childhood. This girl was clearly obese at the age of 12, was instructed to lose weight by two professionals, and had previously been teased by family members. The positive family history of a transient psychotic episode in her brother with no further deterioration would support the formulation of a constitutionally-based sensitive personality prone to neurotic elaboration. While the patient meets the criteria for both agoraphobia and social phobia, she apparently never lost enough weight or persisted long enough in her dieting to qualify as a case of anorexia nervosa by DSM–III criteria, but would be more appropriately labelled as having atypical eating disorder.

These diagnostic distinctions may have significant clinical relevance for treatment. Slade and others have documented the variability in body size estimations of anorexics who, as a group, have a tendency to over-estimate certain body part dimensions. Unfortunately, no body image measurements are reported in this case. In contrast, body image measurements have been described in dysmorphophobic patients which might help differentiate them from the anorexic group. Jerome (1980) described a group of patients with dysmorphophobia requesting cosmetic rhinoplasty who were *more* accurate than normal controls in estimating their own nose size, and spent more time looking at this particular feature of their appearance in mirrors. The data suggested that dysmorphophobics may be utilising a 'perceptual defence' of over-focusing on specific aspects of their body image. This contrasts with the clinical findings in anorexia nervosa, which suggest an avoidance of observation of their own bodies with parallel mirror avoidance and a relative over-estimation of specific body parts in comparison with controls. Furthermore, a recent article by Norris (1984) suggests that anorexics might show clinical improvement following mirror confrontation.

In contrast, preferred treatment for dysmorphophobia might involve the reverse behavioural approach. Support for this hypothesis comes from clinical accounts of patients who report themselves much improved after cosmetic surgery when objective ratings of their appearance indicate no significant change.

The patients who do well have a supportive confidant, an optimistic attitude, and have been prevented from looking at the particular body part, which is shrouded in dressings after surgery. This arguably may allow the dissolution of the distorted body image, which in part may be a manifestation of heightened cognitive awareness for, and the preferential visual scanning of, the body part in question. A similar cognitive-behavioural approach could be envisaged for over-focused dysmorphophobic patients.

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References

HAY, G. G. (1970) Dysmorphophobia. *British Journal of Psychiatry*, **116**, 399–406.

JEROME, L. (1980) *A Study of the Perception of Body Image in Patients Requesting Cosmetic Rhinoplasty*. MSc thesis, University of Manchester.

NORRIS, D. L. (1984) The effects of mirror confrontation on self-estimation of body dimensions in anorexia nervosa, bulimia, and two control groups. *Psychological Medicine*, **14**, 835–842.

Organic Factors in Catatonia

SIR: Though Wilcox & Nasrallah (*Journal*, December 1986, **149**, 782–784) note that catatonia has been reported in non-schizophrenic individuals, they fail to mention that in a study of 55 consecutively admitted catatonic patients Abrams & Taylor (1976) found a preponderance of cases with manic (34) and depressive (5) disorders but only four with schizophrenia. Similarly, of the 25 cases studied by Barnes *et al* (1986), ten were idiopathic and nine had a depressive illness. There was a single case of schizophrenia. Both the idiopathic and the affective groups had a high incidence of recurrent episodes and a family history of catatonia. These findings confirm Kahlbaum's original description of catatonia as a non-specific syndrome, rather than a disease entity, with a wide range of causes, of which affective disorders appear to be the most common.

The undue emphasis on the association of catatonia with schizophrenia is also pertinent to the tragic patient reported by Ainsworth (*Journal*, January 1987, **150**, 110–112). In view of the striking affective changes in her patient's mental state (as well as the family history of depression and mutism), an affective disorder, possibly mania, seems a more appropriate diagnosis than catatonic schizophrenia. Though reported as a case of Stauder's lethal catatonia, no evidence is presented of its characteristic features, i.e. mounting fever and extreme hyperactivity progressing to stuporous exhaustion and death. Clinically it resembles the more familiar benign variety of Kahlbaum (Mann *et al*, 1986).

Ainsworth raises several questions which have an important bearing on the treatment of such patients. Though amylobarbitone sodium interview is reportedly helpful in distinguishing catatonia of psychiatric origin from that with a toxic-metabolic or neurological basis, its value in the lethal variety is uncertain. Whether a putative affective disorder developed subsequent to a sub-clinical viral encephalitis or predisposed her patient to increased infection is a moot point (Wilson, 1976), but the brief return to normality after i.v. diazepam would argue against the usefulness of such techniques rather than render a diagnosis of encephalitis less probable in light of the clinical and pathological findings. Orthostatic hypotension due to the high doses of chlorpromazine could have caused the patient to fall, and while the cause of death seems unrelated to the catatonia, a viral encephalitis may well have compromised her gag reflex, resulting in fatal aspiration. The outcome for patients with benign catatonia, whether of psychiatric or other origins, is described as excellent and most recover spontaneously or rapidly after ECT, although a high rate of recurrence has been reported, particularly among those with a family history of catatonia (Barnes *et al*, 1986). In patients with lethal catatonia ECT may also be dramatically effective, and perhaps lifesaving (Mann *et al*, 1986).

Finally, several authors have commented on the similarities between lethal catatonia and the neuroleptic malignant syndrome, which may well be a drug-induced variety of the former. Because of the dangers posed by neuroleptic treatment in iatrogenic and other forms of lethal catatonia, as well as evidence that some patients develop stupor and fever only after the initiation of such treatment, a prudent approach would be to discontinue its use in benign catatonia too. In view of its efficacy, ECT would appear to be the treatment of choice in catatonia, although in some cases of the lethal variety other agents, e.g. dopamine agonists such as bromocriptine or dantrolene, may be necessary initially.

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References

ABRAMS, R. & TAYLOR, M. A. (1976) Catatonia: a prospective clinical study. *Archives of General Psychiatry*, **33**, 579–581.

BARNES, M. P., SAUNDERS, M., WALLS, T. J., SAUNDERS, I. & KIRK, C. A. (1986) The syndrome of Karl Ludwig Kahlbaum. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 991–996.

MANN, S. C., CAROFF, S. N., BLEIER, H. R., WELZ, W. K. R., KLING, M. A. & HAYASHIDA, M. (1986) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1381.

WILSON, L. G. (1976) Viral encephalopathy mimicking functional psychosis. *American Journal of Psychiatry*, **133**, 165–170.

SIR: Wilcox & Nasrallah (*Journal*, December 1986, **149**, 782–784) suggest that the reason why catatonia has become something of a rarity is that brain injury in childhood, which the authors postulate as predisposing to catatonia, has declined in frequency.

I work in the field of mental handicap, in which brain damage is considered, in many instances, to be at the root of the handicap, particularly in profoundly affected patients. I have seen hardly any mentally handicapped individuals suffering from catatonia, although only recently I have been treating someone with this condition. If my experience is shared by other psychiatrists practising mental handicap, this would be an argument against the hypothesis that an organic brain condition is a forerunner of catatonia, unless one were to say that in the mentally handicapped catatonia, for reasons to do with the level of intelligence, is not a feature.

A survey of the incidence of catatonia in the mentally handicapped would throw light on the attractive theory put forward by Wilcox & Nasrallah.

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A Case of Resistant Schizophrenia

SIR: I wish to comment on the treatment of this unfortunate young man (Mr A) (*Journal*, December 1986, **149**, 789–793). The record of therapeutic failure includes a trial of electroconvulsive therapy: "He was treated with six ECT for a possible affective component to his disorder, but with little benefit." Such a course of electroconvulsive therapy was probably inadequate.

Prior to the introduction of neuroleptic drugs, ECT was commonly used in the treatment of patients with schizophrenia. The comparative studies of the 1960s did not demonstrate a failure of ECT; rather, they demonstrated the equivalence of group results between those patients treated with ECT and those with neuroleptic drugs. The ease of administration, lesser expense, and assumed greater safety of psychotropic drugs, however, led to their replacement of ECT. Recent concerns about tardive dyskinesia, neuroleptic malignant syndrome, and other hazards of neuroleptic drug use (as well as therapeutic failures) have led some clinicians to re-examine the application of ECT in the treatment of schizophrenia.

Friedel (1986) recently reported the successful use of the combination of thiothixene and ECT in eight of nine patients who were non-responders to extended courses of neuroleptic drugs. We have treated nine schizophrenic patients who were neuroleptic and multi-drug treatment failures with the combination of ECT and fluphenazine. Of these, seven have been functioning well in the community for at least one year, and we are encouraged enough to undertake a random assignment trial. However, these patients required an average of 15 ECT in their treatment course, a number greater than is ordinarily given to our depressed patients.

The report by Brandon *et al* (1985) of the results of schizophrenic patients treated in the Leicestershire study and that of Taylor & Fleminger (1980) also encourage the use of ECT in schizophrenic patients. These findings were recently endorsed by the NIH Consensus Conference (1985) and by van Valkenberg & Clayton (1985).

If Mr A is still psychotic, a repeat trial of ECT should be considered with a minimum of 12 induced seizures, preferably with bilateral electrode placement, and with minimum durations of 25 s for each peripheral seizure (30 s central seizure). These treatments should be given while the usual dosages of neuroleptic drug therapy are continued.

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References

FRIEDEL (1986) The combined use of neuroleptics and ECT in drug-resistant schizophrenic patients. *Psychopharmacology Bulletin*, **22**, 928–930.

BRANDON, S., COWLEY, P., MCDONALD, C., NEVILLE, P., PALMER, R.

& Wellstood-Eason, S. (1985) Leicester ECT trial: results in schizophrenia. *British Journal of Psychiatry*, **146**, 177–183.

Taylor, P. & Fleminger, J. J. (1980) ECT for schizophrenia. *The Lancet*, *i*, 1380–1382.

NIMH Consensus Conference (1985) Electroconvulsive therapy. *Journal of the American Medical Association*, **254**, 2103–2108.

van Valkenberg, C. & Clayton, P. J. (1985) Electroconvulsive therapy and schizophrenia. *Biological Psychiatry*, **20**, 699–700.

Panic Attacks and Hyperventilation

Sir: Snaith (*Journal*, November 1986, **149**, 794) takes issue with Gelder's suggestion (*Journal*, September 1986, **149**, 346–352) that lowering of arterial pCO_2 may be a contributory factor in the development of a panic attack, and cites four pieces of evidence that fail to support such a view. However, Snaith has misrepresented Gelder, who did *not* state that hyperventilation *per se* causes panic. The psychological model proposed by Gelder is that hyperventilation induces panic *only* when the bodily sensations which it induces are (a) perceived as unpleasant, and (b) interpreted in a catastrophic fashion (Clark, 1986). In this respect hyperventilation is but one of a number of various pharmacological and physiological agents (such as caffeine, yohimbine, and CO_2 inhalation) which have been shown to provoke panic in susceptible individuals (Margraf *et al*, 1986). Thus the "overwhelming fear of dying or of becoming insane", which Snaith regards as the pathognomonic feature of a panic attack, is simply (in the cognitive model) a consequence of the misattribution of hyperventilation-induced somatic sensations to a life-threatening illness. Ley (1985) provided support for this view when he found that panic patients frequently report that the *first* thing they notice during an episode of anxiety is a physical feeling, most commonly breathlessness.

It is important to note that hyperventilation is neither a necessary nor sufficient prerequisite for panic anxiety. However, it is important to identify the sub-group in whom it may be a necessary condition, i.e. those in whom the symptoms of hyperventilation lead to panic, for the following reasons: firstly, the symptoms which cause panic can usually be reproduced by asking the patient to overbreathe; and secondly, the patients' fears and beliefs about the symptoms can be modified using a therapeutic technique that has been shown to be effective in reducing panic attack frequency (Salkovskis *et al*, 1986). We therefore need to know how this sub-group differs from those patients in whom hyperventilation does not play a role in panic.

Snaith remarks that not all patients who hyperventilate become anxious. This is certainly the case, as we have shown, but such a finding is not unexpected, since it is known that correlation between measures of physiological arousal and subjective reports of anxiety is not robust. It is also consistent with the cognitive model of anxiety. Although it has not been demonstrated that non-anxious hyperventilators do not report anxiety precisely because they do not appraise their somatic symptoms as frightening, this proposition is testable.

Snaith also points out that the administration of CO_2 to patients *induces* anxiety rather than the reverse, and argues that such a finding is not consistent with the hyperventilation theory. But whether CO_2 inhalations are anxiolytic or anxiogenic depends on the individual's cognitive set, i.e. on the instructions that are given before the inhalation. This observation emphasises the importance of cognitive factors in determining the affective state produced by manipulation of CO_2 (Clark, 1986).

I agree with Snaith that there is a lack of consensus about the definition of panic. It is not clear whether panic is quantitatively or qualitatively distinct from other forms of morbid anxiety, and the word has been used to describe both a psychological and a physiological experience. Some authors have introduced additional behavioural criteria such as fear of death, fear of losing control, fear of going crazy, or an urgent need to run, in addition to DSM-III symptoms.

It should be noted that the 'hyperventilation theory' has gained ground not because of the ideology of those committed to a psychogenic rather than a biogenic explanatory model, but because of studies that have revealed the importance of not only cognitive processes, but also specific physiological abnormalities such as hypocapnia in patients with anxiety disorders (Bass, 1985). Laboratory studies using phobic imagery and other provocations may help to further clarify the relationships among cognitive, physiological, and behavioural variables in patients with panic anxiety. Finally, it is worth recalling that the impetus for splitting panic disorder from the body of anxiety disorders arose from the observation that tricyclic drugs had a specific anti-panic effect. The evidence for this 'biogenic' theory of panic is not compelling (Margraf *et al*, 1986).

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References

Bass, C. (1985) Physical symptoms of anxiety. *British Journal of Clinical Practice*, **39** (Suppl. 38), 34–38.

Clark, D. M. (1986) A cognitive approach to panic. *Behaviour Research and Therapy*, **24**, 461–470.

Ley, R. (1985) Agoraphobia, the panic attack, and the hyperventilation syndrome. *Behaviour Research and Therapy*, **23**, 79–81.

Margraf, J., Ehlers, A. & Roth, W. (1986) Biological models of panic disorder and agoraphobia—a review. *Behaviour Research and Therapy*, **24**, 553–567.

Salkovskis, P. M., Jones, D. R. & Clark, D. M. (1986) Respiratory control in the treatment of panic attacks: replication and extension with concurrent measurement of behaviour and pCO_2. *British Journal of Psychiatry*, **148**, 526–532.

Sir: I am grateful for the opportunity to respond to the letters from Snaith (*Journal*, November 1986, **149**, 794) and Bourne (*Journal*, February 1986, **150**, 265–266) about my article on panic attack. In his letter in the December issue, Snaith argues that panic attacks are not caused by hyperventilation, and implies that I take the opposite view. He has misunderstood my position, for I did not conclude that hyperventilation is the most important cause of panic. The misunderstanding centres round two statements in Snaith's letter and these require a response.

Firstly, Snaith writes that the effect of cognitive treatment "supposedly supports the argument that lowering arterial CO_2 is an essential component in the development of a panic attack". This is not the case: the effects of cognitive therapy support a different argument, namely that patients who experience panic attacks have a particular tendency to misinterpret bodily sensations, ascribing them to causes that are more serious than the real ones. For example, palpitations may be interpreted as evidence of heart disease. This view of panic attacks is consistent with the idea that on some occasions the misinterpreted sensations have been produced by hyperventilation, but it is not suggested that they always arise in this way. Equally, the cognitive hypothesis does not suggest that all those who hyperventilate will experience panic – only those people who have the particular tendency to misinterpret bodily sensations.

Secondly, Snaith writes that "the evidence points to the conclusion that raising, *not lowering*, arterial CO_2 induces anxiety". In fact, raising as well as lowering CO_2 can induce anxiety in some people (Van den Hout & Griez, 1984). At first this finding seems paradoxical, but it is easily explained by the cognitive hypothesis. Thus, either an increase or a decrease in pCO_2 can – through different mechanisms – cause unpleasant bodily sensations, and people who misinterpret these bodily sensations will be made anxious by either kind of change.

Returning to my own article, I did not single out hyperventilation as a frequent or major cause of panic attacks. Indeed, I concluded that it is unlikely that all panic attacks will turn out to have a single cause. I also suggested that 'biological' factors (including hyperventilation) have been overemphasised in the recent literature, and that more attention should be given to the investigation of psychological factors in these patients.

Bourne criticises me for failing to give a fuller account of Freud's views. However, he writes as though the subject of my paper were the aetiology of anxiety neurosis; in fact, it was the more specific problem of why some patients with anxiety neurosis develop panic attacks while others do not. My selective quotation from Freud was chosen to show how carefully he had described the clinical phenomena of anxiety disorders including panic attacks. I included this quotation because I agree with Bourne that some of Freud's early papers contain penetrating observations about clinical phenomena. I also agree that these observations are of great interest to present day practitioners of cognitive therapy. It is in their interpretation of the phenomena that analysts and cognitive therapists differ and, more importantly, in the kind of treatment that they have developed. Time will decide which approach is more fruitful: cognitive treatment for panic disorder is now being tested in controlled trials, but we still await reports of a comparable evaluation in which psychoanalytic treatment is compared with other methods.

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Van den Hout, M. A. & Griez, E. (1984) Panic symptoms after inhalation of carbon dioxide. *British Journal of Psychiatry*, **144**, 503–507.

Use of Paraldehyde

Sir: The generally unfavourable discussion on the modern use of paraldehyde by Linter & Linter (*Journal*, November 1986, **149**, 650–651) cannot go unchallenged. The patient reportedly received 40 ml of paraldehyde intramuscularly over 12 hours. This is more than double the recommended maximum i.m. dose of 30 ml per 24 hour period (McEvoy & McQuarrie, 1986). Paraldehyde is a drug with a disagreeable smell and special storage requirements, but over 100 years of anecdotally safe use is precisely why "Its current use is limited to psychiatric units, particularly those without resident medical cover" (Linter & Linter, 1986): an important factor in those parts of the world that are short of doctors. Intermittent claims that paraldehyde is outmoded usually include a pharmacologically erroneous comparison with modern sedative/anxiolytics such as the benzodiazepines and phenothiazines and their derivatives. Paraldehyde belongs to the class of drugs including ethinate, clomethiazole, methyprylon, and ethanol (Goodman & Gilman, 1985) that act by membrane transport inhibition due to high lipophilicity and produce dose-related sedation devoid of the autonomic side-effects common with those drugs that act putatively via neurotransmitter mechanisms. While it is probably correct to state that the high potency benzodiazepines such as clonazepam and lorazepam have superceded paraldehyde in the parenteral treatment of status epilepticus, it remains useful as a sedative/hypnotic adjunct in hyperaroused states such as established delirium tremens, psychotomimetic 'street-drug' toxicity, and extreme mania, complementing the actions of neuroleptics of high anti-psychotic potency (but low sedative rating) that are usually administered concurrently. In these cases paraldehyde in normal doses does not produce the marked hypotension common with equi-sedative doses of the low potency sedating phenothiazines such as chlorpromazine which have marked anticholinergic and alpha-adrenergic blocking actions (Hollister, 1977). The patient reported by Linter & Linter arguably received an overdose of a drug no longer indicated for the condition concerned. These two errors should not be extrapolated to adversely influence the continued use of a drug still regarded by many as the standard against which other agents must be measured (McEvoy & McQuarrie, 1986), which has limited but highly specific indications and which currently has no exact pharmacological equivalent.

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References

Goodman, L. S. & Gilman, A. (eds) (1985) *The Pharmacological Basis of Therapeutics*. London: Balliere Tindall.

Hollister, L. E. (1977) Anti-psychotic medications and the treatment of schizophrenia. In *Psychopharmacology* (eds J. D. Barchas *et al*). New York: Oxford University Press.

McEvoy, G. K. & McQuarrie, G. M. (eds) (1986) Anxiolytics, sedatives and hypnotics. In *American Hospital Formulary Service/Drug Information 1986*. Bethesda, MD.

Autocastration in Ontario Federal Penitentiary Inmates

Sir: With regard to the report by Waugh (*Journal*, November 1986, **149**, 656–658), the summary asserts that autocastration usually occurs in men with chronic paranoid schizophrenia. The evidence for this is not clear in the text, and it seems to contradict our experience, with an admittedly selected population, at the Regional Treatment Centre (Ontario). The Treatment Centre provides in-patient psychiatric care for the approximately 3000 federal penitentiary inmates of the region. The following brief case studies cover seven cases of attempted or successful autocastration on record since 1980.

Case reports: (1) A 23-year-old inmate was incarcerated for life for the second degree murder of his mother, which occurred during an argument about his transsexual behaviour. In 1980 he inserted foreign bodies into his genitalia as an attempt to force surgical castration. During reparative surgery his physique caught the attention of the urologist, and subsequent tests confirmed an XYY karyotype. In 1983 he lacerated his scrotum, intending castration, as part of a suicide attempt in protest at prison authorities' refusal to finance sex reassignment surgery. Final psychiatric diagnoses were of transsexualism and schizoid personality disorder.

(2) A 50-year-old Hungarian immigrant, also serving life for murder, in 1981 cut his scrotum, removed his testicles, and flushed them down the toilet, then swallowed the razor blade. He revealed that he did so to pre-empt a Mafia inspired scheme for other inmates to crush his testicles in punishment. Final diagnosis was of paranoid schizophrenia.

(3) A 38-year-old inmate, serving two years for theft, with earlier convictions for prostitution, in 1984 lacerated his scrotum, removed the right testicle, and by stuffing the cavity with cigarette ends, forced a complete surgical orchidectomy. Diagnoses were of transsexualism and antisocial personality disorder.

(4) A 41-year-old man lacerated his scrotum as part of a suicide attempt after revocation of his parole. He had served ten years of a composite 24 year sentence for a series of rapes. While out on parole and because of impotence he had refused continued provera treatment, which in turn lead to the revocation. Diagnoses were of adjustment disorder with depressive features, and antisocial personality disorder.

(5) A 40-year-old transsexual, who had castrated himself in 1979, slashed at his penis in 1980 and again in 1983 in protest at being placed in a male, rather than female, institution after his conviction for a series of armed robberies. Diagnoses were of gender dysphoria and personality disorder, unspecified.

(6) A 34-year-old serving ten years for six counts of robbery in 1984 almost completely severed his genitalia and slashed his wrists, intending suicide. He did it, he said, to punish himself. His sexual orientation was normal; diagnoses were of substance abuse and borderline personality disorder.

(7) A 27-year-old transsexual serving a 15-year composite sentence for assault and attempted murder in 1983, under the influence of "brew" and illicit drugs, attempted castration to force sex reassignment surgery. Diagnoses were of transsexualism, substance abuse, and personality disorder, unspecified.

Of these seven patients, three of them successful, only one was diagnosed as having a psychosis. The others all showed personality disorder of various kinds. Almost all had a previous history of self-mutilation, slashing of the wrists, or previous suicide attempts. The largest group, four patients, consisted of long-standing transsexuals whose disturbed personalities had possibly suggested a poor prognosis for sex reassignment surgery, and who consequently had been refused such surgery by the various specialists they had encountered. Of interest in a generally young population is the higher than average age of the group. While the high proportion of personality disorders may simply reflect the catchment population, which may also account in part for the low number of psychotic patients, this report serves to emphasise that such behaviour is not necessarily, nor particularly often, associated with paranoid schizophrenia. The most common associations seem to be a disturbance of sexual identity, a previous history of self-mutilation, and personality disorder.

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Ethnic labels in South Africa

SIR: We refer to the correspondence regarding the use of South African Population Registration Act categories as a basis for psychiatric research (Sashidharan & Lipsedge, *Journal*, April 1986, **148**, 484; Teggin *et al*, *Journal*, November 1986, **149**, 667–668; Graham, *Journal*, November 1986, **149**, 669). During the time that we were working at the MRC/University of Cape Town Clinical Psychiatry Research Unit this was a major issue of debate. The labels "Black", "Coloured", "Indian" and "White" as used in South Africa are fundamentally political, and do not refer in any scientific sense to discrete ethnic or cultural groups (Sharp, 1980). The important way in which these labels are "real" is that they dictate vastly differing access to resources of all kinds, including housing, education, employment, and health care.

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The Continuum of Psychosis and the Gene

SIR: Crow attempts to replace the Kraepelinian dichotomy between manic depressive psychosis and schizophrenia with a continuum of psychosis (*Journal*, October 1986, **149**, 419–429). We agree that data such as those obtained by Kendell *et al* support the conclusion that a symptomatic continuum exists. However, the evidence for a genetic continuum of the form envisaged by Crow is less persuasive.

Crow's model is based on several studies from the older literature reporting an excess of individuals with schizophrenia among the offspring of patients with affective disorder while failing to observe the converse. None of these studies employed modern diagnostic criteria, and it is possible that misclassification occurred. Indeed, studies (quoted by Crow) using modern methods have failed to show an increase in schizophrenia among the relatives of probands with affective disorder. Crow also argues that similarities in season of birth effects point to an underlying commonality of genetic mechanisms. However, the season of birth effect in schizophrenia is associated with an absence of a manifest genetic predisposition, suggesting a relationship with environmental factors (Boyd *et al*, 1986).

In contrast, evidence that schizophrenia and affective disorder are based on two independent genetic diatheses is more compelling. Firstly, as Crow acknowledges, the two major functional psychoses by and large breed true. Secondly, bipolar affective disorder appears to have a stronger genetic component than schizophrenia. Crow's hypothesis would appear to predict the converse. Thirdly, Elsässer (1952) found an equal prevalence of the two psychoses in the children of marriages between an affective and a schizophrenic individual. A greater prevalence of schizophrenia than affective disorder is predicted by Crow's hypothesis. Finally, Crow's model also fails to take into account the existence of "schizophrenia spectrum disorders", or the evidence that minor depression and depressive and cyclothymic personality disorder are genetically related to affective disorder. These findings suggest that there are separate phenotypic spectra related to the schizophrenic and the affective genotypes which are orthogonal to the phenomenological continuum that exists between the two disorders.

Given this evidence for independent genotypes, where does this leave the nosological status of schizoaffective disorder which occupies the intermediate position in the symptomatic continuum? One possibility is that it consists of both affective and schizophrenic types of illness. However, as Crow reminds us, the concept arose out of the failure to demonstrate such a division. Secondly, it might be a genuine interform between schizophrenia and affective disorder (Kendell, 1983). This implies that both illnesses are polygenic, with the presence of some elements from each genotype predisposing to schizoaffective disorder. A third possibility, which does not imply polygenic inheritance of both Kraepelinian psychoses, is that it represents the presence of both the schizophrenic and the affective genotypes in the same patient: it occurs in individuals who, in genetic terms, have both illnesses. It has been argued by Kendell (1983) that this is unlikely because schizoaffective disorder is more common than would be expected given that "the chance coincidence of two illnesses each affecting around one person in a hundred is one in ten thousand".

We disagree with such a conclusion on several grounds. Firstly, there is no reason to suppose, as Kendell does, that for schizoaffective disorder to occur a genetic diathesis to schizophrenia would have to exist with one for bipolar rather than unipolar affective disorder. The morbid risk for all types of affective disorder combined is much greater than 1%, and therefore Kendell's expected figure would be larger. Secondly, assortative mating might take place to increase the likelihood of both genotypes being present in combination. Thirdly, Kendell assumes that possession of one genotype will not affect expression of the other. On the contrary, it seems likely that schizophrenic symptoms will trigger the onset of affective illness in a predisposed individual and vice versa. Moreover, it is quite possible that *subclinical* expression of one genotype increases the likelihood of the other being expressed in a predisposed individual. For example, possession of an affective diathesis might render an individual with a schizophrenic genotype more likely to develop a psychotic reaction to stressful life events. Such a psychosis might be expected to show both schizophrenic and affective features.

In summary, we suggest that the symptomatic continuum reflects phenotypic interaction rather than the relationship proposed by Crow.

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References

Boyd, J. H., Pulver, A. E. & Stewart, W. (1986) Season of birth: Schizophrenia and bipolar disorder. *Schizophrenia Bulletin*, **12**, 173–186.

Elsässer, G. (1952) *Die Nachkommen geisteskranken Ellernpaare*. New York: Stechert & Hoffner.

Kendell, R. E. (1983) Other functional psychoses. In *Companion to Psychiatric Studies* (3rd ed.) (eds R. E. Kendell & A. K. Zeally). Edinburgh: Churchill Livingstone.

Weissman, M. M., Gershon, E. S. & Kidd, K. K. *et al* (1984) Psychiatric disorders in the relatives of probands with affective disorders. *Archives of General Psychiatry*, **41**, 13–21.

Procyclidine Abuse

Sir: In reporting five more cases of procyclidine abuse, Fenech & Khoosal (*Journal*, October 1986, **149**, 524) pointed out that the latest edition of the *British National Formulary* (BNF) did not mention the potential for abuse of that drug. Pullen *et al* (1984*a*) drew attention to the fact that the BNF omitted reference to the abuse of any anticholinergic.

At last the BNF (Number 11, 1986) does include one sentence on abuse in its section on the use of antipsychotic drugs (p. 139), although there is still no mention of this danger in the main section on anticholinergics (pp. 182–184). These preparations continue to be freely prescribed, and there still seems to be continuing ignorance about these significant drugs of abuse (Pullen *et al*, 1984*b*).

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References

Pullen, G. P., Best, N. R. & Maguire, J. (1984*a*) Anticholinergic drug abuse: a common problem? *British Medical Journal*, **289**, 612–613.

Pullen, G. P., Best, N. R. & Maguire, J. (1984*b*) Anticholinergic abuse. *British Journal of Psychiatry*, **145**, 671–672.

Neurological Factors in Obsessive-Compulsive Disorder

Sir: In their article on obsessive-compulsive disorder (OCD) (*Journal*, September 1986, **149**, 315–319), Kettl & Marks emphasise the case for organic precipitants, probably operating 'downstream' from the primary mechanisms initiating the condition. Whether cognitive abnormalities in OCD attributed to these organic precipitants could be intrinsic or secondary features of the condition is not assessed. This remains unclear in the recent studies quoted (Behar *et al*, 1984; Flament & Rapoport, 1984), which showed computerised tomogram and cognitive abnormalities in adolescents with OCD. Brain injury was not used as an exclusion criterion in the group concerned. Some patients had histories of head trauma and birth injury, and one is mentioned as having tardive dyskinesia.

I have studied 19 DSM-III-diagnosed obsessive-compulsive patients, none of whom had neurological impairment, psychosis, alcoholism, or a history of head injury (Harvey, 1986). They were given tests of frontal lobe functions and sub-tests of the Weschler Adult Intelligence Scale (WAIS). Using matched normative data for Nelson's Modified Wisconsin Card Sorting Test (MWCST), the obsessionals were shown to perseverate significantly more than normals ($t=2.80$, $P=0.01$). Their mean percentage perseveration (50%, s.d. = 31) was greater than for patients with gross frontal lobe damage (42%, s.d. = 25). Obsessionals with or without significant perseveration were comparable on age-scaled sub-tests of the WAIS. Perseveration correlated with degree of obsessionality on the Leyton Obsessional Inventory (Spearman's $r=0.50$, $P=0.01$), which also correlated, negatively, with alternating category verbal fluency (ACVF) ($r=-0.62$, $P=0.002$). This latter test and the MWCST assess cognitive set-shifting ability, a specific frontal lobe function.

Although OCD could affect cognitive tasks, such as the ACVF test, via impaired performance efficiency, it seems unlikely that a qualitatively distinctive error, such as perseveration, would occur on this basis. An overall impairment of performance would seem more likely, although this was not evident from the WAIS sub-tests. Whether or not idiopathic OCD is associated with frontal impairment therefore needs to be looked at as a specific issue. It should not be assumed that such impairment necessarily implies the presence of a known brain injury.

References

BEHAR, D., RAPOPORT, J. L., BERG, C. J., DENCKLA, M. B., MANN, L., COX, C., FEDIO, P., ZAHN, T. & WOLFMAN, M. G. (1984) Computerised tomography and neuropsychological test measures in adolescents with obsessive-compulsive disorder. *American Journal of Psychiatry*, **141**, 363–369.

FLAMENT, M & RAPOPORT, J. L. (1984) Childhood obsessive-compulsive disorder. In *New Findings in Obsessive-Compulsive Disorder* (ed. T. R. Insel). Washington DC: American Psychiatric Press Inc.

HARVEY, N. S. (1986) Impaired cognitive set-shifting in obsessive compulsive neurosis. *IRCS Medical Science*, **14**, 936–937.

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Gamma Glutamyl Transpeptidase and Mean Cell Volume in Alcoholics

SIR: Latcham (*Journal*, September 1986, **149**, 353–356) suggests that measurement of serum gamma glutamyl transpeptidase (GGT) and red blood cell (RBC) mean corpuscular volume (MCV) are of "little value in the assessment of patients admitted to psychiatric beds for problem drinking".

Macrocytosis in alcoholics has at least four different causes, some of which are independent of the duration of drinking habit. Lindenbaum (1980) comments that alcoholics' enlarged RBC can be secondary to liver dysfunction, reticulocytosis in response to blood loss, or folate vitamin deficiency, as well as "the macrocytosis of alcoholism".

Nearly a quarter of Latcham's male subjects and a sixth of the females were not clinically diagnosed as alcohol-dependent. What differences were there, in the correlations reported, between those diagnosed as alcohol-dependent and those diagnosed as suffering from alcohol abuse?

If only 143 male subjects had GGT assays performed then the maximum number of subjects in his Table III should be the same.

His data suggest that a considerable proportion of the male subjects had been abstinent for two or more weeks. This is long enough for mildly elevated GGT levels to settle to 'normal'.

The upper limit of normality for GGT of 50 i.u./litre is probably excessively high. We have recently shown (Hambridge & Jones, in preparation), that an upper limit of 40 i.u./litre is probably more clinically valid. Also, many clinicians will have experience of assessing younger, fitter alcoholics with high consumption and minimal damage – however defined.

I suggest that Latcham has not proven his case and that the measurement of GGT, RBC, and MCV remain of considerable value in assessing and managing alcoholics, when considered with the full clinical picture.

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References

LINDENBAUM, J. (1980) Folate and Vitamin B12 deficiencies in alcoholism. *Seminars in Haematology*, **17**, 119–124.

Neuroleptic Malignant Syndrome or Lithium Neurotoxicity

SIR: I refer to the comments made by Lowe & Batchelor (*Journal*, September 1986, **149**, 385–386) on the review of the neuroleptic malignant syndrome (NMS) by Abbott & Loizou (*Journal*, January 1986, **148**, 47–51).

Their criticism regarding omission of the original report by Cohen & Cohen (1974) seems to be founded on inconclusive evidence. The descriptive picture in the four patients has been found to be similar to NMS (Frankel & Spring, 1982), but there have been divergent opinions regarding the interpretation of the cause of the clinical picture. It has been suggested that these patients have persistent neurological after-effects of acute lithium neurotoxicity (Donaldson & Cuningham, 1983; Schou, 1984). A detailed analysis of the patients described by Cohen & Cohen reveals that all were on high doses of lithium carbonate (1165 mg/day to 1800 mg/day). The maximum serum lithium ranged from 1.48 mmol/litre to 2.45 mmol/litre during the acute phase of lithium toxicity. All four patients were female. A preponderance of females has been reported in patients with persistent neurological sequelae of lithium (Donaldson & Cuningham, 1983; Schou, 1984), unlike NMS (Shalev & Munitz, 1986).

Moreover, each of the four patients was left with permanent brain damage (two became grossly demented, two had persistent dyskinesias). NMS, despite a mortality rate of 20%, is an acute condition and generally regresses without sequelae. Shalev & Munitz (1986) reviewed 120 patients with NMS and could identify only four with permanent sequelae. In one of these, permanent brain damage was probably due to brain anoxia during ECT given in the course of NMS and not due to the condition itself. In contrast to this, I have identified 48 patients with long-lasting sequelae of lithium intoxication (either alone or in combination with other drugs) in the literature.

Although there is controversy about lithium/haloperidol interaction (Cohen & Cohen, 1974; Frankel & Spring, 1982) it is well established that lithium intoxication at times resolves leaving persistent neurological sequelae (Donaldson & Cuningham, 1983; Schou, 1984). Since NMS is only a descriptive term, superficial resemblance may be seen with any other descriptive syndrome, e.g. lethal catatonia and some cases of lithium neurotoxicity.

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References

COHEN, W. J. & COHEN, N. H. (1974) Lithium carbonate, haloperidol and irreversible brain damage. *Journal of the American Medical Association*, **230**, 1283–1287.

DONALDSON, I. M. & CUNINGHAM, J. (1983) Persisting neurologic sequelae of lithium carbonate therapy. *Archives of Neurology*, **40**, 747–751.

FRANKEL, M. H. & SPRING, G. K. (1982) Questions about combined lithium and haloperidol treatment. *American Journal of Psychiatry*, **139**, 537–538.

SCHOU, M. (1984) Long-lasting neurological sequelae after lithium intoxication. *Acta Psychiatrica Scandinavica*, **70**, 594–602.

SHALEV, A. & MUNITZ, H. (1986) The neuroleptic malignant syndrome: agent and host interaction. *Acta Psychiatrica Scandinavica*, **73**, 337–347.

Gilles de la Tourette's Syndrome in Down's Syndrome

SIR: Karlinsky *et al* (*Journal*, May 1986, **148**, 601–604) speculate on a possible causal link between these two conditions. They appear to have neglected a more straightforward aetiology for the Tourette's syndrome.

It is reported that the patient developed seizures at the age of 24 years. Age-related epilepsy, with an onset in adult life, is a well recognised complication of Down's syndrome (Veall, 1974; Tange, 1979). The patient was being treated with carbamazepine two years later, at the onset of the Tourette's syndrome. Carbamazepine has recently been recognised as one of a number of pharmacological compounds (including dextroamphetamine, methylphenidate, and other adrenergic agents) which may precipitate tics, (Gualtieri & Evans, 1984). This tends to fit in with the dopaminergic theory of Tourette's syndrome, mentioned by the authors of the paper. Although, as they also point out, tics may be missed in people with mental retardation, there is as yet no clearly established link with any particular syndrome.

Precipitation of the tics by carbamazepine may account for the late onset of Tourette's syndrome in this patient, and seems a more likely explanation than those proposed by the authors. It is an iatrogenic complication of which psychiatrists should be aware.

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References

GUALTIERI, C. T. & EVANS, R. W. (1984) Carbamazepine-induced tics. *Developmental Medicine & Child Neurology*, **26**, 546–548.

TANGYE, S. R. (1979) The EEG and incidence of epilepsy in Down's syndrome. *Journal of Mental Deficiency Research*, **23**, 17–24.

VEALL, R. M. (1974) The prevalence of epilepsy among mongols related to age. *Journal of Mental Deficiency Research*, **18**, 43–48.

Mianserin and Blood Dyscrasias

SIR: It is with some concern that I note that manufacturers of mianserin (Norval, Bencard and Bolvidon, Organon) have written to the profession in the UK advising of changes in the Data Sheet concerning bone marrow depression and blood dyscrasias, the changes having been instituted at the insistence of the Committee on Safety of Medicines. The essential change is that a full blood count is recommended every four weeks during the first three months of treatment. This action follows concerns highlighted in a recent CSM update (1985).

There appears to be confusion between a drug-related depression in the white cell count and drug-induced agranulocytosis. Many psychotropic drugs are known to be able to produce changes in the white cell count. Tricyclic antidepressants, phenothiazines, and carbamazepine are all recognised as being able to depress the white cell count. Lithium, on the other hand, is associated with a leucocytosis. On the whole these findings are benign, usually incidental results with no clinical manifestations, and reverse on cessation of the drug (Kaplan & Sadock, 1985). Agranulocytosis with the features of opportunistic infection, however, is a more serious condition, commoner in older females and arising 4–6 weeks after initiation of therapy, which in the case of chlorpromazine can carry a mortality of 30% (Kaplan & Sadock, 1985). This condition arises very quickly and appears unpredictable; thus many authorities believe that routine blood counts are unhelpful and that the doctor should rely on the associated symptoms and signs (Kaplan & Sadock, 1985; Gregory & Smeltzer, 1983).

Actual numbers of patients with these drug-related adverse events are extremely difficult to ascertain. The criteria for reporting a finding as a particular reaction are themselves usually idiosyncratic, depending on the physician's expertise, and there are few guidelines provided to standardise such reports. These problems are compounded by a methodology that is essentially anecdotal, with no control groups. Thus comparisons are difficult and often misleading, with doctors being more likely to report events with newer drugs than with older established compounds. Perhaps the most clinically iniquitous practice, however, is that the reports appear to be acted upon in isolation and out of the context of the overall risk-benefit ratio of the particular drug, and it is the overall mortality from exposure to the drug that should be considered.

Combining data from the Office of Population Censuses and Survey for England and Wales for 1982–84 with similar data from the General Register Office for Scotland over the same period, there were 1029 deaths in which tricyclics either alone or in combination were implicated. This compares with 55 in which mianserin either alone or in combination were implicated. Even allowing for the difference in rates of prescription, the overall mortality associated with mianserin is substantially less than with the tricyclics. Considering deaths resulting from antidepressant drug overdoses alone, the differences are especially striking, with mianserin accounting for only 15 such fatalities per million patients compared with 388 per million for the tricyclics and bridged-tricyclics (Leonard, 1986).

Practising geriatric psychiatry in Canada, I am restricted to using toxic and problematical antidepressants in the elderly which themselves can produce serious blood dyscrasias. For example, with amitriptyline 83 cases were reported in Britain between 1964 and 1981, with 18 deaths (Clink, 1983).

Therefore, it is far from clear that amendments to the Data Sheets for mianserin will achieve an improvement in the treatment or outcome of depressed patients. Routine blood monitoring is not likely to detect the uncommon event of a blood dyscrasia, and may well confuse the picture further as there are no controlled data with which to compare results of such testing. The likely outcome is that many doctors will be put off using mianserin, especially in patients who are particularly at risk from the tricyclic antidepressants such as the elderly. Ironically, the CSM may be contributing to increasing the risks of treatment.

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References

Clink, H. M. (1983) Mianserin and blood dyscrasias. *British Journal of Clinical Pharmacy*, **15**, 291S–293S.

CSM update (1985) Adverse reactions to antidepressants. *British Medical Journal*, **291**, 1638.

Gregory, I. & Smeltzer, D. J. (1983) *Psychiatry; Essentials of Clinical Practice* (2nd ed.). Toronto: Little, Brown and Company.

Kaplan, H. I. & Sadock, B. J. (1985) *Modern Synopsis of Comprehensive Textbook of Psychiatry* (4th ed.). Baltimore: Williams and Wilkins.

Leonard, B. E. (1986) Toxicity of antidepressants. *The Lancet*, *ii*, 1105.

Psychological Complications Following a Mis-diagnosis of Deafness

Sir: The differential diagnosis between hysteria and organic illness is important and fraught with pitfalls. Emphasis has been placed on ensuring that organic conditions are not mis-diagnosed as hysterical with consequent delay in treatment (Slater, 1965); however, it has also been recognised that protracted physical investigations and delay in psychiatric referral may worsen the prognosis for true hysterical symptoms (Goodyer, 1981).

Hysteria in childhood is uncommon, and is reported as not occurring in children under five years of age (Goodyer, 1981). We report a case of chronic hysterical deafness presenting at the age of two years with delay in the exclusion of any organic pathology until the age of fourteen years.

Case report: A fourteen-year-old boy presented to the adolescent psychiatric unit with a six-month history of increasingly poor peer and family relationships. He displayed a marked speech impediment, and communication was complicated by an apparently variable and selective deafness. Otherwise, physical examination was found to be normal. Although of average intelligence, his educational attainments were assessed as being at the eight-year level, as were his behavioural, emotional, and social abilities.

The history revealed that at the age of two years he was referred to a child psychiatrist with disturbed behaviour. His language was delayed and he appeared not to respond to noises or speech. At ENT referral, examination of the ear drums was normal but hearing tests suggested he was profoundly deaf. The diagnosis was supported by a history of otosclerosis in the immediate maternal family. He was given post-aural aids, and at follow-up there was improvement in his language and behaviour. Repeated audiometry between the ages of two and fourteen confirmed the diagnosis, although the degree of deafness was inconsistent. Doubt about the diagnosis was expressed, but dismissed when response to spoken sounds improved with more powerful hearing aids.

He attended educational establishments for the deaf until at 13 years of age he was integrated into an ordinary high school. Although he has always displayed various behavioural difficulties at home and at school, it was not until at high school that an educational psychologist noticed that the degree of deafness appeared to vary with his psychological state; at times his hearing appeared normal. Auditory evoked brain stem potentials were performed which showed normal hearing in the right ear with only a 40 dB loss in the left ear.

In the six months following the revised diagnosis his behaviour deteriorated dramatically. It has since been revealed that at eighteen months old he witnessed his father severely assault his mother, following which he hid in a cupboard for two days with his hands over his ears. The boy's mother thinks that the deafness dated from that time. Despite having been an in-patient in an adolescent psychiatric unit for five months both he and his family are still finding it difficult to adjust to the change in diagnosis.

This case illustrates the danger of mis-diagnosing psychological symptoms as being due to organic illness, and shows that when the correct diagnosis is delayed the sequelae can be severe with significant resultant morbidity. Psychological symptoms are commonly found among the deaf population; the rates appear to be 3–10 times higher than for comparable groups of children with normal hearing (Meadow, 1980). Presumably this was one reason why our patient's behaviour was accepted for so many years. The importance of considering deafness in a young child with behaviour disorders or language delay is well documented (Illingworth, 1983; Williams, 1968). We suggest that where there is any doubt as to the validity of the diagnosis of deafness, a diagnosis of hysterical conversion disorder should be considered.

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References

Goodyer, I. (1981) Hysterical conversion reactions in childhood. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, **22**, 179–188.

Illingworth, R. S. (1983) *The Normal Child.* Edinburgh: Churchill Livingstone.

Meadow, K. P. (1980) *Deafness and Child Development.* London: Edward Arnold.

Slater, E. (1965) Diagnosis of hysteria. *British Medical Journal, i,* 1395–1399.

Williams, C. E. (1968) Behaviour disorders in handicapped children. *Developmental Medicine and Child Neurology*, **10**, 736.

CORRIGENDA

Journal, January 1987, **150,** 43–48 (M. W. P. Carney *et al*). The following co-authors names were omitted from the title page: James Edeh, B. K. Toone and C. Thomas.

Journal, February 1987, **150**, 161–163 (J. A. O. Besson *et al*). In Fig. 1 (page 162) the labels "Schizophrenics ($n=6$) without TD" and "Schizophrenics ($n=15$) with TD" should be interchanged.

A HUNDRED YEARS AGO

French Lunatic Asylums

The supervision of French lunatic asylums appears, if recent accounts be true, to be exceedingly lax. It will be remembered that a short time ago a man was released from an asylum, having been forty years previously confined by his relatives in order to secure his wealth. The escape of another man is now reported, who, although sane, was confined for a year in an ill-lit room, bound hand and foot. Such treatment is strictly forbidden by law, and the matron and two keepers of the establishment have been fined, and the medical superintendent condemned to a fortnight's imprisonment. Such regrettable and scandalous incidents would be impossible if any control were exercised by the authorities; and although our Gallic friends may manage things in general better than we do in England, their most ardent admirers will be compelled to admit that the protection of lunatics and the supervision of asylums do not redound to their credit.

(*The Lancet*, 8 January, 1887, 109.)

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

British Journal of Psychiatry (1987), **150**, 572–579

Book Reviews

Editor: Sidney Crown

Divided Consciousness: Multiple Controls in Human Thoughts and Actions (expanded edition). By ERNEST R. HILGARD. Chichester: John Wiley & Sons. 1986. Pp 313. £18.50.

This book, by one of the most distinguished figures in modern psychology, first appeared in 1977. The current edition contains a ten-page addendum which brings the reader up-to-date with more recent developments in research and theory. The book is a wide-ranging and thorough review of the phenomenon of dissociation. The author adopts a broad definition, including instances of human activity in which some thought, experience, and behaviour, normally performed consciously, takes place largely or exclusively outside of the person's awareness. Defined thus, dissociation is something which can be understood from everyday experience and which has been extensively studied by psychologists working in the field of attention. However, the author concentrates mainly on the more striking manifestations of dissociation which may be observed in the clinical field, and in demonstrations of the capacities of virtuoso hypnotic subjects.

The first chapter is a short history of the study of dissociation, from Janet in the late nineteenth century, through its demise in the 1930s with the influence of behaviourism, to a revival of interest among psychologists from the 1960s onwards. The second chapter examines possession states, fugues, and multiple personalities; this is followed by chapters on hypnotic age regression and amnesia. Subsequent chapters deal with dreams, hallucinations, imagination, creativity, dissociation of motor activity, and automatic writing. The remaining chapters are concerned with the nature of hypnosis and suggestion, and in the final chapters the author expands on his neodissociation theory.

The nature of the material makes this a fascinating book, and the reader's interest is sustained throughout by the author's clear and honest style. His approach is eclectic and undogmatic, but he insists at all times on impeccable standards of logic and scientific rigour. The text is well referenced and illustrates the work of experimental psychologists at its best – not least the contributions of the author himself, which span over half a century.

Hilgard has set the definitive standard for scientific writings on the nature of human consciousness. For any serious student of the human mind this book is obligatory reading.

M. HEAP, *Principal Psychologist and Senior Lecturer, University of Sheffield*

Alzheimer's and Parkinson's Diseases: Strategies for Research and Development. Edited by ABRAHAM FISHER, ISRAEL HANIN and CHAIM LACHMAN. New York: Plenum. 1986. Pp 710. $95.00.

This volume contains papers presented at an international conference held in Israel under the auspices of the Israel Institute for Biological Research in March 1985. It seems to have been an unusually busy meeting, for the contributions number 73 and cover almost 700 pages of small print. This reflects the current clinical and research interest in these major degenerative CNS disorders.

The material is arranged in six sections, covering anatomy, pathology, and biochemistry; clinical and diagnostic aspects; aetiology and genetics; biological markers; experimental models; and new drug development and innovative experimental approaches. As might be expected, there is wide variation in standard and general interest. Many papers report highly specialised animal experimental work, but there are also some valuable review articles of wider interest. For example, Wisniewski provides a useful summary of the clinical and neuropathological diagnostic criteria for Alzheimer's disease and argues in favour of an infective aetiology in genetically susceptible subjects. Edwardson *et al* review neurotransmitter changes and senile plaque morphology in Alzheimer's disease, with particular reference to the recent identification of alumino-silicates at the core of the plaques. Bowen *et al* remind us that, as well as the cholinergic deficit, serotonin receptors are also consistently reduced in Alzheimer's disease, and Groner *et al* report on chromosomal investigations into the fascinating association between Down's syndrome and early onset Alzheimer's disease. The recently identified Parkinsonian syndrome induced by MPTP, a toxic derivative of meperidine, is covered thoroughly by Kopin *et al.*

Accounts of the investigation of Alzheimer's and Parkinson's diseases by magnetic resonance imaging, computerised tomography, positron emission tomography, and regional cerebral blood flow estimations are exciting, if rather technical.

Basic research produced an effective therapy for Parkinson's disease, so one can look forward, with some degree of confidence, to a similar outcome to the massive research effort by various disciplines in Alzheimer's disease, exemplified by this volume. It is a useful sourcebook for researchers into Alzheimer's or Parkinson's disease, but of limited value to clinicians who can obtain the same information elsewhere at less expense.

KENNETH DAVISON, *Consultant Psychiatrist, General Hospital, Newcastle upon Tyne*

Somatization and Hypochondriasis. By ROBERT KELLNER. London: Praeger. 1986. Pp 401. £60.00.

Hypochondriasis has been greatly neglected by psychiatrists, whose writings have been largely based on highly atypical patients seen in hospital or private practice. It is only recently that they have extended their interest to attenders at general hospitals and in primary care. An additional and major obstacle to understanding is classification. 'Hypochondriasis' is used loosely as a comprehensive term, and also as a specific category within what DSM–III christened 'somatoform disorders'. There are advantages to using *somatisation,* another word with a long history, as the general category covering all the psychological mechanisms that cause somatic symptoms, including anxiety, depressive, and adjustment disorders as well as somatoform disorders.

In this major review, Kellner prefers the term 'functional somatic symptoms' for somatic symptoms which do not have a physical cause. This enables him to take a broad view which draws on evidence from general hospitals as well as specialist psychiatry. He emphasises that most symptoms are transient and minor, and that few persist following investigation and reassurance.

The organisation of the book is straightforward. An initial chapter on history is followed by reviews of prevalence, aetiology, the many specific syndromes, principles of diagnosis, psychological tests, and treatments. Psychotherapy is particularly well described. However, even though each chapter is clearly divided by sub-headings there is much repetition, and it becomes difficult to follow consistent themes. It is therefore a book for the already knowledgeable rather than for psychologists or psychiatrists who are unfamiliar with the literature.

Kellner may have taken on an impossible task. Certainly, most previous reviews are unhelpful, much of the evidence unsatisfactory, and the best classification (DSM–IIIR) is arbitrary and tentative. I found his book useful and irritating at the same time. It is copiously referenced, has told me many things I did not know and contains valuable clinical insights, but I had to work hard turning from chapter to chapter and making the maximum use of the index. There are also a number of gaps, too little on anxiety disorders and on behavioural management and rather perfunctory reviews of important syndromes such as hyperventilation and atypical chest pain.

It is a pity that Kellner did not have the nerve to impose a clearer structure on his book and to take a more ruthlessly critical view of his subject. This is a useful, but very expensive, reference work which is best seen as a sourcebook of the background rather than a guide to new ideas.

RICHARD MAYOU, *Clinical Reader in Psychiatry, University of Oxford*

The Reality of Mental Illness. By MARTIN ROTH and JEROME KROLL. Cambridge: Cambridge University Press. 1986. Pp 128. £22.50 (hb), £7.95 (pb).

That such a book as this seems necessary should give psychiatrists serious cause for thought. Is our professional standing and function still, after the hefty buffeting of the 1960s and 1970s, in serious doubt? Do the views disseminated by Szasz over the past 25 years continue to persuade people that mental illness is a myth and not a reality? Do contemporary social and political opinions require a re-examination and re-statement of the nature and relevance of psychiatry? This book makes it clear that answers to these questions are indeed needed and that it is a debate in which each psychiatrist should have his views.

The polemical writings of Szasz, Laing, Foucault, and Basaglia are trenchantly taken to task for their simplistic responses to highly complex issues and the destructive consequences their application may bring. Their views are also criticised, in the context of a valuable and informative historical perspective in which psychiatry is firmly established as part of medicine, for the selectiveness of their choice of evidence.

The eternal problem of the proper boundaries of mental disorder is examined, in particular the uncertainty of the relationship of neurosis and personality disorders to normal feelings and behaviour. However, the reliability of psychiatric diagnosis is found to be comparable to that of electrocardiographic and radiological assessments.

Minor reservations relate to the lack of an index, the absence of biographical information about the authors, and the absence of a clear indication of which readership the book is aimed at.

This reviewer would hope that members of the Mental Health Act Commission involved in re-drafting the Draft Code of Practice will read and carefully consider the final chapter, in which involuntary hospitalisation and the plea of insanity defence are discussed with erudition and elegance. For psychiatrists, the book provides a rousing and reassuring *raison d'etre.*

ALAN KERR, *Consultant Psychiatrist, St Nicholas Hospital, Newcastle upon Tyne*

Alcoholism: Development, Consequences, and Interventions (3rd edition). Edited by NADA J. ESTES and EDITH M. HEINEMANN. Oxford: Blackwell Scientific Publications. 1986. Pp 466. £16.00.

This multi-authored textbook is designed principally for nurses and other health professionals working with alcoholics. The book illustrates two of the major differences between Britain and the USA in their approach to alcohol problems. The first is the high level of seriousness and investment devoted in the USA to professional training in the management of alcohol-related problems;

the second is the primacy given to the concept of 'alcoholism' as a disease entity, which in contrast to the UK incorporates a whole range of health and social consequences that arise out of injudicious as well as addictive drinking.

Despite the title, which suggest a rather narrow focus, the book in fact addresses a wide range of other alcohol-related topics such as epidemiology and the effects of alcohol on the body systems. A further section of the book is devoted to alcohol problems in 'special groups' such as young people, women, the elderly, and the American Indians. An interesting chapter is concerned with drinking problems within the nursing profession, a much-neglected topic.

Counselling a person with alcohol problems is perceptively described in a chapter which contains much good sense and immediately reveals that whatever the rhetoric which surrounds issues such as diagnosis on either side of the Atlantic, the skills required of the sensitive and experienced counsellor are universal. Group and family therapy receive considerable attention, as do the perspectives of AA and Al-Anon, but there is little space devoted to the organisation and co-ordination of services. Chapters on cognitive and behavioural approaches describe more recent developments. Work and research from outside the United States receives scant mention, and the book is designed principally and rightly for its own culture. This will make it of limited usefulness to readers in the UK, although there are undoubtedly chapters which will stimulate and inform. Perhaps its principal value is as a reminder to health professionals in this country that we should take postgraduate training in the management of alcohol problems much more seriously than we do at present.

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Investigating Abnormal Behaviour. By EDGAR MILLER and STEPHEN MORLEY. London: Weidenfeld & Nicolson. 1986. Pp 336. £9.95.

Miller and Morley have set out to produce a volume on abnormal psychology which is radically different in concept and content from texts designed for American undergraduate courses. While most books in this field examine each clinical disorder in turn, or evaluate the various models of abnormal behaviour, these authors have set themselves a more ambitious task: their aims are to present this branch of psychology as a coherent scientific discipline and to illustrate how its investigative techniques can be used to provide answers to at least some of the major questions relating to the nature, aetiology, and treatment of psychiatric disorders. Consequently, they have departed from the rigid structure of a conventional textbook on abnormal psychology and have selected certain topics which serve to demonstrate the width and scope of this discipline.

This strategy is both the strength and weakness of the book. An advantage of the approach is that it has enabled the authors to confine themselves to specific issues which they feel highlight the contributions made by abnormal psychology, while disregarding those areas where psychological research has not proved so fruitful. Not surprisingly, therefore, models of depression are cited throughout, while references to the genesis of schizophrenia are relatively sparse. Similarly, the chapters on process and outcome research in psychotherapy deal exclusively with well-established behavioural techniques, while the newer family therapies receive no mention. Thus, although the authors may have achieved their principal aim of demonstrating the special contributions which abnormal psychology can make to the field of psychiatry, many readers will find the topic selection somewhat arbitrary and the content rather disjointed. However, in view of the thoroughness with which each of the selected issues is examined, postgraduate students will find it a useful complement to the standard textbooks in this field.

DOUGAL MACKAY, *District Psychologist, Bristol and Weston Health Authority*

Tranquillisers: Social, Psychological, and Clinical Perspectives. Edited by JONATHAN GABE and PAUL WILLIAMS. London: Tavistock. 1986. Pp 311. £25.00.

There is a difference of opinion between profession and public about tranquillisers. This work, concerned largely with minor tranquillisers, concentrates on personal and social aspects rather than on pharmacology. There is a rich variety of facets, and after a historical account of the use of tranquillisers the remaining topics fall loosely into four groups.

Three studies on the long-term use of tranquillisers cover the process and effects of withdrawal, the duration of treatment with psychotropic drugs, and a pharmacological view. The second part, which deals with factors concerned in obtaining and using tranquillisers, looks at sex differences and the effect of family environment. A more peripheral paper of general practitioner's methods of enquiry and management concludes that "... the observable behaviour of the general practitioners is best understood as the implementation of familiar and trusted techniques by means of which they seek to impose order in the consulting room". The section on alternatives is particularly topical, including papers on withdrawing tranquillisers, not prescribing them, and self-help groups. A further section deals with social and symbolic aspects of the problem, and the social meanings: tranquillisers as a resource and as social control. The concluding chapter, by the editors, discusses future trends in research on tranquilliser use and draws attention to the need for better international communication.

There is much to be learned from this book, but as 12 chapters are reprinted and these are, on average, over five years old, it is an expensive message. The breadth of topic and approach could suggest difficulty in integration, but this is fully overcome by the introductions to each section and by the forewords which give a comforting perspective to this rapidly changing chemical and social scene. It is a work worthy of priority reading.

JOHN POLLITT, *Medical Director, Hayes Grove Priory Hospital, Kent*

Systematic Family Therapy. By LUCIANO L'ABATE. New York: Brunner/Mazel. 1986. Pp 319. $30.

What is systematic family therapy? Family therapy, like any recently introduced therapeutic form, demands that each expert introduces a brand, or different mixture of the product. There are many forms of family therapy described, including structural family therapy, systemic family therapy, strategic family therapy, contextual family therapy, behavioural family therapy, and now systematic family therapy. There are overlaps and unique features to each. L'Abate is convinced that the field of family therapy is characterised by gurus who have produced schools "based on little or no empirical evidence". He points out that evidence of the gurus' "sensitivity, ingenuity and cunning is not founded on external criteria but on some vague impression and conviction". L'Abate presents an approach that is in his own words "systematically eclectic, systematically empirical, and systematically sequential". "Systematic" therefore refers to a rationale based on classifiable ordered plans and methods.

L'Abate is concerned about two very different world views which have caused controversy in the family therapy field. One he described as represented by the researcher/investigator/empiricist/pragmatist, and the other by the artist/dialectician/aesthetician/impressionist. L'Abate is keen to show that both these activities can be performed in parallel with each other, not distinct from the other. He describes four stages of therapy, the first to do with crisis intervention and stress reduction, the second with skills training and education with the use of homework assignments. Issues of termination are the third stage, and the fourth phase is the follow-up.

In going through the various stages he describes his approach to dealing with crises by the use of paradoxical intervention and, by contrast, the use of confrontation support and control. He introduces a variety of familiar issues to the field, to do with life roles, triangulations, positive reframing negotiation and aspects of family emotional life, competence, feelings, awareness, issues of intimacy, and aspects of various therapeutic approaches. L'Abate is well read, and appears able to find ways of linking many current ideas in the family therapy field. However, his volume is surprisingly thin on case histories in comparison with other works. The evidence he gives of particular interventions seem limited and unconvincing. There is something in L'Abate's thesis about gurus who appear convincing by demonstrating therapeutic work that appeals to the intuitive sense of clinicians. L'Abate's work, while constructing a set of theories and ideas, does not have the same power to convince the clinician, which is a pity since some of his work on the use of paradoxical approaches to therapy is interesting – if he is able to present some research to show that the pragmatist and the impressionist can together produce better outcomes he will have helped the field. There is certainly a need to integrate and make sense of the bewildering varieties of wares on offer to the neophyte family therapist. This in my view is not the definitive conceptual answer.

ARNON BENTOVIM, *Consultant Psychiatrist, Hospital for Sick Children, London*

Essential Papers on Depression. Edited by JAMES C. COYNE. New York: New York University Press. 1968. Pp 507. US$50.00 (hb), US$22.50 (pb).

These papers form part of a series collectively titled "Essential papers in psychoanalysis", a title which belies the much wider aim of the editor of this volume. The opening chapter, a careful and workmanlike review by the editor of the different theoretical approaches to the problems of classification and aetiology of depressive illness, is followed by a section on psychodynamic approaches to depression, including original papers by Freud, Abraham, and Fromm-Reichmann. However, the scope of the book then widens beyond the psychodynamic. The section on behavioural and cognitive approaches features, among other authors, Seligman's model of learned helplessness and Beck's views on maladaptive cognitive structures. Interpersonal and social approaches are well covered, including chapters by George Brown and Ernest Becker. Nor are biomedical approaches ignored, with contributions from Winokur, Dunner, and Baldessarini. The value of this book lies in the wide range of topics covered, mostly in original contributions from key workers in their respective fields. The emphasis is not on integrating differing and often opposing views, but on providing a richly differentiated overview of the theoretical perspectives currently being developed in the search for understanding of depression. The wide range of significant writing included could almost be said to justify its title. The major objection one might have is that all the contributions are available elsewhere, and some (for example Freud's *Mourning and Melancholia*) very widely. Specialists in the field will be familiar with the writings of the contributors to this volume, if not with these particular contributions, and the more general reader may well not have sufficient interest to pursue this volume through the reference library. If this were a reasonably priced, freely available paperback I would recommend that anyone working in the field of mental health might benefit from owning a copy, but doubt from the American prices whether many copies would be sold outside libraries. Nonetheless, this book is a valuable collection of thoughtful and thought-provoking material, as well as an excellent source of references.

GRAEME MCGRATH, *Lecturer in Psychiatry, University of Manchester*

Handbook of Schizophrenia Vol I: The Neurology of Schizophrenia. Edited by HENRY A. NASRALLAH and DANIEL R. WEINBERGER. Amsterdam: Elsevier. 1986. Pp 416. $92.50.

This excellent book is the first of five volumes dealing with various aspects of schizophrenia. In it the authors set out to present the scientific evidence for the neurological basis of the disorder. The first chapters are presented in roughly the same order as that of a neurologist's clinical examination, and cover history, neurological examination, differential diagnosis, laboratory tests, etc. The core of the book reviews the findings from brain imaging techniques (computerised tomography, magnetic resonance imaging, computerised mapping of electrophysiological data, cerebral blood flow studies, and positron emission tomography). There follow several chapters covering theoretical models of schizophrenia based on current neuropathological findings.

The basic premise of the work, and this runs coherently through the various contributions, is that schizophrenia is a manifestation of a brain disease in a final common pathway for many disorders or genetic or acquired origin. This leads to attempts firstly at defining possible sub-types of the syndrome and, secondly, at relating phenomenological findings to disorders of brain structure and function as revealed by neuropathological and *in vivo* brain imaging techniques. It is clear that providing the impetus for much of this research is the advance in computer technology, allowing development of these non-invasive brain imaging techniques. This in turn has renewed interest in, and to some extent allowed reinterpretation of, many phenomenological observations in schizophrenic patients. Hence, such interesting chapters as those by Manschreck on motor abnormalities, Nasrallah on cerebral asymmetries, and Shelton & Weinberger on X-ray computerised tomography are complimentary without being merely reiterative.

Such attempts at relating structure to function can only be preliminary. The authors resist the temptation to construct over-elaborate theoretical models that outstrip their empirical foundation, and overall the presentation is critical, well referenced, and contains clear discussions of the methodological shortcomings of the various techniques mentioned. Perhaps, given the conceptual framework of the book, it would have been more appropriate for the editors to follow Bleuler's example and title it 'The neurology of the schizophrenias'.

STUART MCLAREN, *Clinical Research Fellow, Unit of Human Psychopharmacology, The London Hospital*

Understanding Psychological Testing in Children: A Guide for Health Professionals. By STEWART GABEL, GERALD D. OSTER and STEVEN M. BUTNIK. New York: Plenum Medical. 1986. Pp 184. £26.50.

Many medical people feel that psychological testing is well within their capability, with or without training, and it is unlikely that this book will alter those opinions. It is aimed at primary health care professionals, who are often concerned with behavioural and emotional problems of childhood. In a brief, dry style it describes how psychologists assess children, choose their batteries of tests, and reach their therapeutic conclusions. There is a brief introduction to how tests work, and a spot of insight into concepts and jargon. There is also instruction on how to choose the appropriate private psychologist.

This American book was first published in 1943 and, though doubtless re-edited, has some glaring gaps. For example, the Raven's Progressive Matrices, a test used world-wide, is not even mentioned; nor are Cattell's various personality inventories, although the TAT, the Rorschach, and the much queried Draw-a-Person (which used to be Draw-a-Man) are given great prominence. Descriptions are given of American tests of perception, social/adaptive skills, educational achievement, and neuropsychological procedures, and the text is well supplied with succinct case histories. However, there is no name index.

The approach is not so much as a guide as a euphemistic listing, as all the tests, including intelligence tests such as the Stanford-Binet, are presented without a whiff of criticism. Because of this lack of direction and discussion the book fails both the readers' latent curiosity and the rich variety of valuable test material in use.

JOAN FREEMAN, *Honorary Tutor, Department of Education, University of Manchester*

Recent Advances in Epilepsy – 3. Edited by TIMOTHY A. PEDLEY and BRIAN S. MELDRUM. Edinburgh: Churchill Livingstone. 1986. Pp 315. £38.00.

This is the third volume of a series entitled "Recent advances in epilepsy", edited by two well-known researchers in the field, one more interested in experimental aspects of epilepsy, the other a clinician. To some extent this reflects the mixture of topics covered in the book, although in this volume clinical chapters predominate. Of particular interest for psychiatrists will be the chapter by Wieser on psychomotor seizures of hippocampal-amygdalar origin, in which some of the elegant studies carried out with implanted depth electrodes are summarised, including the clinical correlates of mesio-basal limbic system seizures. The chapter by Quesney on seizures of frontal lobe origin is clinically important in the sense that these seizures are probably commoner than recognised, on account of diagnostic difficulty and confusion with non-epileptic (pseudo) seizures, or because of the psychopathological accompaniments of the condition. Toone provides one of the best reviews of sexual disorders in epilepsy that is to be found, and includes a summary of his own work on recent hormonal investigations in patient populations. Finally, Stores provides an interesting chapter on non-convulsive status epilepticus in children, again a problem which child psychiatrists see from time to time and whose evaluation can be difficult but rewarding.

The other chapters provide a pot-pourri of information with much recent research for those with a wider interest in epilepsy. Neurocytology and chemistry, genetics, ambulatory monitoring, benign focal epilepsies of childhood, and the progressive myoclonic epilepsies are some of chapter topics. It is a pleasure to have Professor Aicardi provide an up-to-date review of the syndrome that is named after him, and the topic of toxicity of anticonvulsant drugs is reviewed by Schmidt, although the neurocognitive and behavioural sequelae of these is covered in another volume. There is an update of ethosuximide and the barbiturates, and reviews of the use of ambulatory EEG monitoring and of the benign focal epilepsies of childhood.

The quality of the production and the editorial control is as high as that of the previous two volumes, and the book can be recommended to psychiatrists who have an interest in epilepsy and who would wish to keep up-to-date with advances in the area. This reviewer would encourage the editors to press on with Volume 4.

MICHAEL R. TRIMBLE, *Consultant Physician in Psychological Medicine and Senior Lecturer in Behavioural Neurology, The National Hospitals for Nervous Diseases, London*

Paediatric Perspectives on Epilepsy. Edited by EUAN ROSS and EDWARD REYNOLDS. Chichester: John Wiley. 1985. Pp 163. £17.50.

1985 was a vintage year for books on childhood epilepsy. *Paediatric Perspectives* summarises a symposium held in 1984, and gathers together an easily digested, indexed overview of (largely British) attitudes to various key issues. The contributions are succinct and go beyond diagnosis and drug treatment to a consideration of the child and his family.

Many children with problematic epilepsy are managed by neurologists, and Ross and Reynolds have edited a book that should appeal not only to their colleagues but also to others in search of a readable update on a small corner of their clinical practice. That the book does not encompass recent advances in imaging, telemetry, or ambulatory monitoring realistically reflects the sad state of funding of the departments from which most of the contributors come. In contrast, *Epileptic Syndromes in Infancy, Childhood and Adolescence* (J. Roger *et al*, 1985, John Libbey Eurotext) deals uniquely with the nosological insights and obfuscations provided by the Marseilles School of Gastaut and its European disciples and contempories. Here the emphasis is on extended EEG recordings, fine-grain analysis of seizure types, and sub-division into new syndromes. This volume records a 1983 conference. It is a larger, better produced and more expensive book.

No symposium ever resulted in a coherent textbook, but there was no need, for 1985 also saw the rebirth of O'Donohoe's masterwork, *The Epilepsies of Childhood* (2nd edition, 1985, Butterworths). If I could only have one of these three books it would be this one. *Paediatric Perspectives* covers less ground but it is quicker to read. Neither book overlaps with the French one.

MICHAEL PRENDERGAST, *Clinical Lecturer in Child and Adolescent Psychiatry, The Bethlem Royal Hospital, Beckenham*

Psychological Therapies for the Elderly. Edited by IAN HANLEY and MARY GILHOOLY. Beckenham: Croom Helm. 1986. Pp 200. £17.95.

This collection of ten papers by different authors has been compiled by the editors to reflect the usefulness of applied psychology in the field of the elderly. It should be of interest to psychologists and others working with the elderly, and to those involved in the training of such workers. The chapters vary greatly in quality and relevance. The first chapter is a useful review of some of the issues involved, but psychiatrists and geriatricians will not be endeared by comments such as, "Psychiatric and geriatric services are often organised without rhyme or reason and remain almost totally lacking in accountability for the quality of service given." The author describes a typical long-stay ward as having 35 people with severe dementia and four nurses. Those of us whose teams, without the help of psychologists, have managed to reduce the numbers of residents on long-stay wards to under 20 may be forgiven for thinking that the author is out of touch. The second chapter, on dynamic psychotherapy with the elderly, provides interesting reading. The relevance of the three case histories described must be judged by the reader; the first is a bereaved man of around 60 who is still at work, the second a lady in her early sixties in a responsible executive position, and the third a truly elderly lady of 84 with a 33-year-old lover.

There follow two excellent papers on the usefulness of reminiscence therapy and reality orientation therapy. The next two papers, on day care and self-help groups, review provision predominantly in America. A helpful paper on the difficult topic of introducing change and improvement in the care provided by institutions is followed by a straightforward and readable account of the development of a video-assisted teaching package. There is then a down-to-earth account by a residential social worker on life in a home for the elderly, run in a dignified and homelike manner. The final chapter, on legal and ethical issues, is useful but fails to deal with such important topics as informed consent from patients with organic brain syndromes, or patients' access to information.

In summary, this is a short and readable book, more than half of which is of interest and value to those working with the mentally ill elderly. It is of doubtful value for money in hardback, but certainly a useful addition to the library shelf.

CAROL TROTTER, *Consultant Psychiatrist for the Elderly, St James's Hospital, Portsmouth*

Practical Psychiatry of Old Age. By JOHN WATTIS and MICHAEL CHURCH. Beckenham: Croom Helm. 1986. Pp 195. £19.95(hb), £8.95 (pb).

The stated aim of the authors, to provide a practical reference book on the psychiatry of old age directed primarily at community health care teams, junior hospital doctors, and nurses, is more than adequately achieved. The combination of a psychiatrist and a clinical psychologist produces a refreshing 'middle of the road' view and the clear literary style, easy-to-understand diagrams and liberal use of case histories results in a volume which should be comprehensible to professional and layman alike.

The book adopts a problem-solving approach rather than one based solely on diagnosis, but still manages to emphasise the importance of thorough physical and psychiatric assessment and the need for accurate diagnosis and classification of mental illness in the elderly in order to produce effective management strategies. The chapter on depression offers sound practical advice, but it was disappointing that the management of the suicidal elderly patient, a common and difficult clinical situation, was not considered in greater detail. Confusion and hypochondriasis, two of the more frequent presenting symptoms in the primary care setting, are analysed in depth with suitable emphasis placed on the importance of complementary medical, psychological, and social approaches.

Two chapters shine out in particular. Firstly, the place of individual and family therapy, often overlooked in more 'organically orientated' textbooks, is discussed in depth, and the importance of practical psychotherapeutic skills in both patient management and relatives' support is underlined. Secondly, neuropsychological assessment in the elderly, so often a minefield of confusion, is presented in a detailed systematic way which must rate as one of the most easily understandable expositions available in the psychogeriatric literature.

This is an excellent book, highly recommended to all practising clinicians and a 'snip' at £8.95.

MIKE NOWERS, *Lecturer in Mental Health, University of Bristol*

Practical Psychotherapy. By MYRON F. WEINER. New York: Brunner/Mazel. 1986. Pp 322. $30.00.

I warmly welcome this admirable guide to pragmatic psychotherapy. Psychoanalytic psychotherapy has long suffered as the poor relation of psychoanalysis, while finding it difficult to establish a credible separate identity. Weiner starts from the premise that there is much to be learnt from behavioural, humanistic, and ego-supportive therapies which can be incorporated into everyday work with patients. He illustrates his approach with numerous clinical examples throughout the text.

There are excellent accounts of the life cycle, factors which induce change in psychotherapy, mechanisms of defence, therapist interventions, levels of therapy, and termination. Those countless practitioners who have found in Irvin Yalem's *Group Psychotherapy* a comprehensive and readable guide which is essentially practical rather than theoretical may feel that this book fills a similar need in the field of individual psychotherapy.

My only reservations concern the extent to which formulations are couched in Freudian drive and structural theory while failing to sufficiently reflect the contribution of object relation theory or self-psychology. It should be remembered that it is written for a North American audience, where these ideas still form the bedrock of psychoanalytic theory.

I would like to see this readable guide made widely available to trainees, who become easily disillusioned with over esoteric texts whose content is apparently divorced from their experience with their patients.

I. F. MACILWAIN, *Consultant Psychiatrist/ Psychotherapist, Netherne Hospital, Surrey*

Psychotherapist's Casebook. Edited by IRWIN L. KUTASH and ALEXANDER WOLF. London: Jossey-Bass. 1986. Pp 552. £49.50.

The aim of this admirable book is to present the reader with brief descriptions of a wide range of psychotherapeutic styles. The range is quite broad, including individual, group, and family therapy and both the traditional, orthodox methods and more innovative and controversial ones. Each of the 30 chapters is written by an acknowledged expert – sometimes by the orginator of the particular approach described, e.g. Carl Rogers wrote the chapter on client-centred therapy and Albert Ellis the one on rational emotive therapy. The title derives from the fact that each contributor has been required to provide some illustrative clinical material. Some have rather overdone this, leaving themselves insufficient space to present an adequate account of the psychodynamic theory on which the therapy is based and to give the reader a reasonable idea of what the therapeutic procedure amounts to.

This is an all-American book, and perhaps reflects the present American scene. I would like to have seen a chapter on the English object-relations school and also on the cognitive therapy of depression. To my mind there was no justification for a chapter on psychopharmacological therapy. The chapters on psychoanalysis and psychoanalytic therapy should have been fused into one. Also, two chapters on the existential approach seems excessive. The humanistic therapies, on the other hand, could have been better represented by including one chapter on bioenergetics and the body-centred therapies and another on the cathartic therapies.

In the main, the book is lucidly written and is a pleasure to read. It is, however, a sad reminder that psychotherapy is still appallingly compartmentalised. Hopefully the day will dawn when all competent therapists will have a knowledge of and expertise in the whole spectrum of psychotherapeutic strategies. Perhaps the publication of books such as this will help to bring that day a little closer.

JOHN BIRTCHNELL, *Scientific Officer and Senior Lecturer, Institute of Psychiatry, London*

Schizophrenia: New Pharmacological and Clinical Developments. Edited by A. A. SCHIFF, SIR MARTIN ROTH and H. L. FREEMAN. London: Royal Society of Medicine. 1985. Pp 121. £12.00.

Although it is perhaps astonishing to find a drug with novel actions on the mind, available for 15 years, whose clinical application is still unclear, it is not surprising that examination of this drug, sulpiride, is but one of the topics in this deceptively slim volume covering several new developments in understanding and treating schizophrenia. The actions of the drug are one of the absorbing themes presented at a symposium.

The scientific articles cover the 'negative' symptoms of schizophrenia, the nature of depression which can occur in schizophrenia, rapid stabilisation of acute schizophrenia with fluphenazine decanoate, and the result of a 14-year follow-up study using this depot preparation. The second session focused on sulpiride, dopamine and tardive dyskinesia, combining chemical and clinical considerations in a stimulating proportion. Taken as a whole, these papers examine well-known modern treatment, the problems remaining in its wake, and changing theoretical considerations, as a background to viewing the future prospect of the available and anticipated members of the substituted benzamide group.

Each paper is summarised and followed by brief discussion. Several psychiatrists present cases, and the whole work is brought together in Sir Martin Roth's succinct concluding overview. The book is well produced, containing much of clinical significance in this chemically and symptomatically enigmatic area.

JOHN POLLITT, *Medical Director, Hayes Grove Priory Hospital, Kent*

Hypnosis and Hypnotherapy. By DAVID T. ROWLEY. Beckenham: Croom Helm. 1986. Pp 192. £19.95 (hb), £8.95 (pb).

There is a mounting deluge of books on hypnosis and its applications. There are those which are based on the scientific foundations of hypnosis and its established areas of application; there are those covering the work and ideas of Milton Erickson and his followers, most of which are of dubious quality; and there are a few contributions by lay practitioners which cannot be taken seriously. David Rowley's book belongs to the first category.

The book is an introductory account of the nature of hypnosis and associated phenomena, its history, theories, measurement, some basic procedures, and therapeutic applications. It is general and comprehensive, and written in a non-technical and readable style. The author does not present any new material or formal theorising of his own, but he includes occasional personal reflections which are interesting, particularly concerning the nature of altered states of consciousness. There is only one chapter on clinical applications and, as the author covers a wide variety of problems, many of the discussions are too brief and superficial. This is a shortcoming of many introductory texts on hypnosis.

The book provides a very useful overview of hypnosis for medical and psychological practitioners, health workers, researchers and students in the biological and social sciences, and the educated layman. It is not a manual, and does not aim to teach the reader hypnotic procedures. Those who are already familiar with hypnosis and its applications may give the book a miss, unless they wish to refresh themselves quickly on such matters as history, theories, and measurements of susceptibility, for which purposes they should find the book admirably suited.

MICHAEL HEAP, *Principal Clinical Psychologist, Middlewood Hospital, Sheffield*

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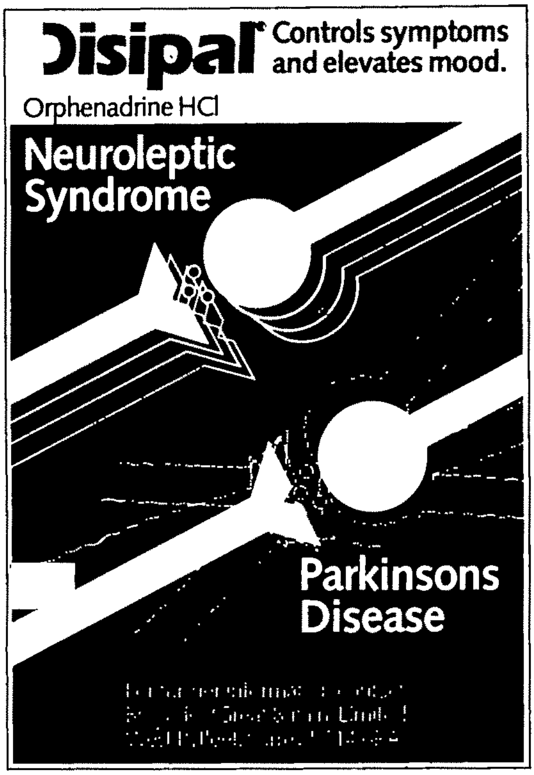

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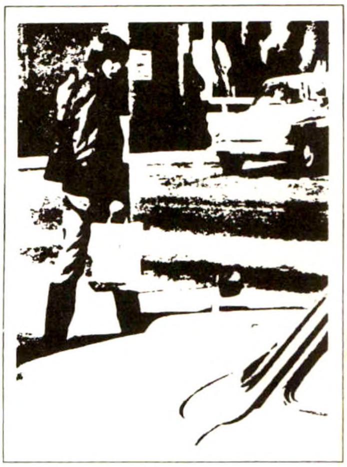

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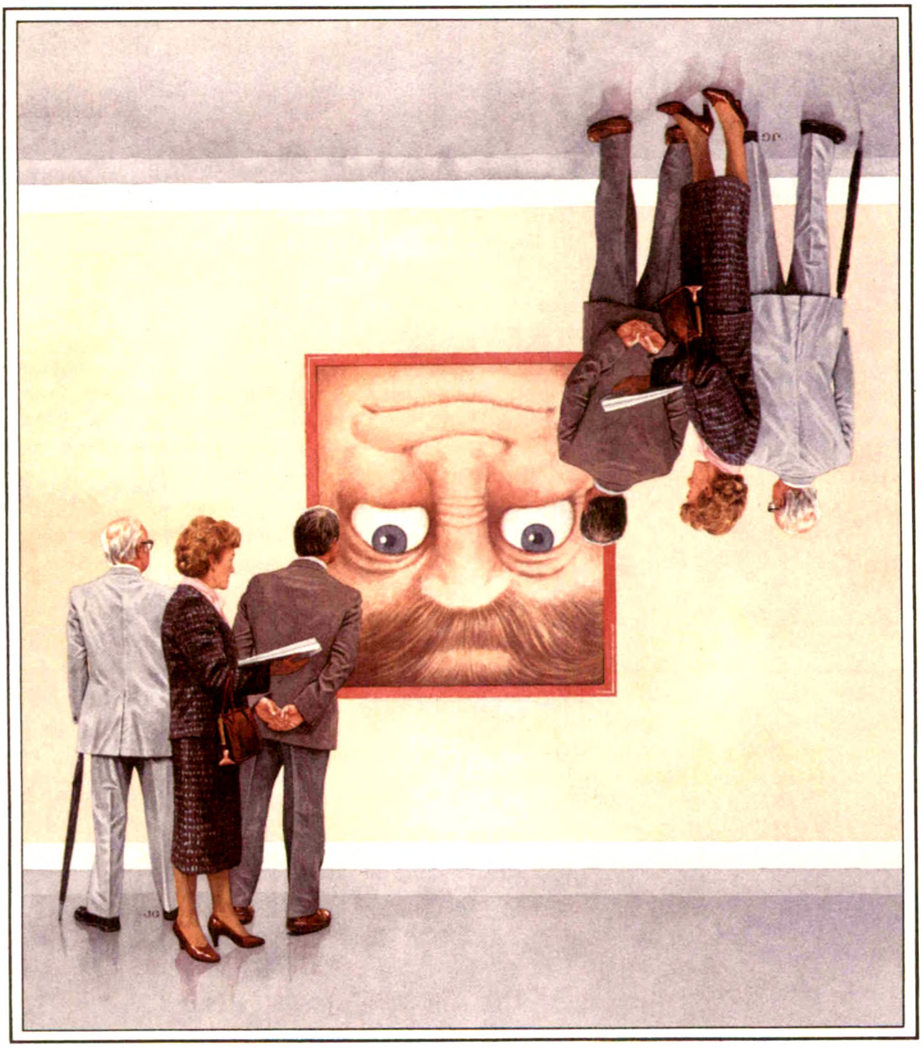

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Lecture

Is There Really A Split In Schizophrenia?

The Genetic Evidence

P. McGUFFIN, A. FARMER and I. I. GOTTESMAN

Although the clinical presentation and course of schizophrenia is highly variable, it is unclear whether this reflects heterogeneity at an aetiological level. The genetic evidence is reviewed concerning 'traditional' clinical subtypes as well as more novel categories derived from multivariate statistical methods and Crow's type I–type II classification. Recent data based on a twin series and re-analysis of older published family material suggest that attempts to divide up schizophrenia have resulted in splits between two or more categories of disorder which occupy different positions on the same continuum of liability. Thus, apparent heterogeneity is more likely to be due to quantitative difference in familial-genetic loading rather than qualitative differences. Similarly, the hypothesis that schizophrenia can be broadly divided into two groups, one genetic and the other non-genetic has little to support it. It seems improbable that any further useful and genetically relevant subdivision of schizophrenia can be effected on purely clinical grounds. Further progress awaits developments in the discovery of endophenotypes and the application of molecular genetic marker strategies.

Current concern with categories such as 'type I' and 'type II' (Crow, 1980), positive-symptom versus negative-symptom (Andreasen, 1985; Pogue-Geile & Harrow, 1985) and 'sporadic' versus familial schizophrenia (Murray *et al*, 1985) represents a new and interesting phase in an old and unresolved debate over the unity and diversity of the disorder (*British Medical Journal*, 1969; Farmer *et al*, 1984). E. Bleuler (1911) introduced the term 'schizophrenia' to cover *a group* of disorders which Kraepelin (1896) had implied could be comfortably subsumed under a single category. Many researchers since, impressed by the clinical variety of patients described as 'schizophrenic', have taken a Bleulerian stance and have aimed to be splitters rather than Kraepelinian lumpers of the syndrome.

Psychiatric geneticists and biological psychiatrists, in particular, have been on the side of the splitters. The case was put authoritatively and succinctly by Eliot Slater in a paper written nearly 40 years ago (Slater, 1947). He wrote: "In view of the fact that schizophrenia is a good deal more common than any single genetically determined disorder is otherwise known to be, heterogeneity is inherently probable".

Our purpose, therefore, in this article is to review both the older and more recent genetic evidence concerning heterogeneity in schizophrenia and to decide whether it supports the widely held point of view that we are dealing with several, perhaps many conditions.

On lumping and splitting

In his encyclopaedic catalogue of mendelising genetic disorders McKusick (1983) favours the view that what is called 'schizophrenia' is a mixture of different entities. However, elsewhere he points out (McKusick, 1969) that the leading principles of genetic nosology include not only the idea of heterogeneity, but also of pleiotropism. Pleiotropism is the phenomenon of a single aetiological factor (for example a single gene) having multiple effects. A nice example with psychiatric relevance is the demonstration by Penrose (1952) of the various (biochemical, morphological and behavioural) phenotypic manifestations of the phenylketonuria gene. However, we can broaden the notion of pleiotropism to include non-genetic medical conditions and here syphilis and systemic lupus erythematosis provide excellent examples of how a specific pathogenic process can have clinical manifestations which are many and various, often leading to diagnostic error and perplexity.

In cases of late syphilis the contrasts in presentation between one patient and another may be quite

---

Based on a lecture given at the Royal College of Psychiatrists Annual Meeting on 9th July 1985

disparate, whereas in psychiatry classification and differentiation of syndromes is based on comparatively subtle differences, and moreover on differences which are liable to change over time. We therefore ignore at our peril McKusick's (1969) warning that when dealing with heterogeneity, purely phenotypic differences may provide "the most treacherous basis for decision". Against this, it must be allowed that progress in medicine has depended to a large extent on teasing apart broad clinical syndromes and on the delineation of specific, narrower disease entities. Little more than a century ago conditions such as 'dropsy' and anaemia were considered adequate diagnostic entities which were only subsequently separated by a combined process of refining clinical descriptions and pursuing all available clues concerning aetiologies and pathogeneses. In schizophrenia the pathogenesis remains obscure but there is compelling evidence of an important genetic contribution to the aetiology (Gottesman & Shields, 1982). Indeed if we are to start by lumping all of the conservatively diagnosed Western European studies of schizophrenia together, genes probably account for about two-thirds of the variation in liability to the disorder (Gottesman & Shields, 1967; McGuffin, 1984; McGue *et al*, 1985). Clinical heterogeneity in schizophrenia undoubtedly exists but it is only of limited interest if this does not reflect *aetiological* heterogeneity. Therefore whether there is a genetic split (or set of splits) in schizophrenia might be viewed as the most important current issue in the nosology of the syndrome.

Clinical and 'traditional' subtypes

Family data

Leonhard (1959) has provided probably the most elaborate and extensive scheme for splitting schizophrenia on the basis of descriptive psychopathology. Other workers, particularly those in the Soviet Union, have devised different schemes largely based on course and outcome (Snezhevnevsky, 1968). However, such complicated approaches have not won widespread acceptance and most twin and family researchers have adopted more time-honoured subtypologies largely based on Kraepelinian-Bleulerian categories. Particular attention has been focused on a paranoid versus hebephrenic (or paranoid versus non-paranoid) classification. The largest single dataset concerned with schizophrenic probands and their families is that of Kallmann (1938). By modern standards there are a number of methodological deficits. These include diagnoses in relatives which were mainly not made blind to proband information, an absence of explicit diagnostic criteria, and a lack of a suitable control group.

Nevertheless, the sample size and the wealth of detail provided encourages our close attention. Kallmann (1938) was himself unimpressed by the degree of familial homotypia in his material, i.e. the tendency for affected pairs of first-degree relatives to belong to the same subtype of schizophrenia. He pointed out that only in about 70% of affected relative pairs was the subtype identical. However, Slater (1947) subsequently retabulated Kallmann's data and performed a χ^2 analysis which showed that the tendency for like to go with like in parent-offspring pairs classified as hebephrenic, catatonic and paranoid, occurred at the 0.01 level of significance. There have been at least eight other studies (reviewed by Kendler & Davis, 1981) which have addressed much the same question. Most have shown a tendency for paranoid versus non-paranoid schizophrenia to 'breed true'. Only three comparatively recent studies (Tsuang *et al*, 1974, 1980; Scharfetter & Nusperli, 1980) carried out diagnosis of relatives blind to the proband subtype. Two of these studies (Tsuang *et al*, 1974; Scharfetter & Nusperli, 1980) found a tendency towards homotypia which in the latter study (1980) occurred at a better than chance level. However, the more recent study of Tsuang *et al* (1980) used narrow operational criteria (Tsuang & Winokur, 1974) to define a paranoid subtype and not only found absence of homotypia but also a tendency at long-term (30-40 years) follow-up for paranoid schizophrenia to progress to a hebephrenic or 'undifferentiated' picture.

A rather different way of examining family data is to concentrate on quantitative rather than qualitative differences between subtypes. Thus if we divide up probands according to subtype but simply examine the frequency of schizophrenia among relatives irrespective of subtype there is a fairly consistent pattern in nearly all studies in that the relatives of patients with paranoid schizophrenia tend to show a lower morbid risk for all types of schizophrenia than do the relatives of patients with non-paranoid or hebephrenic disorders. Kendler & Davies (1981) have suggested that this is sometimes an artefact of the method of age correction. The simplest and commonest method of age correction is the shorter (or abridged) Weinberg method (Slater & Cowie, 1971; McGuffin, 1984a). Here it is assumed that there is a (more or less) well-defined period of risk for acquiring a disorder and by a simple method of weighting of those relatives who have not lived through this period an adjusted denominator is calculated to estimate the life time expectancy (or morbid risk) of disorder. Kendler & Davies argue that if the period of risk is assumed to be the same for all subtypes of disorder then the rates in relatives of older, later onset cases such as paranoid schizophrenics will tend to be underestimated. However, this bias cannot account for all the differences in the literature. For example, in Kallmann's (1938) study the differences in the frequency of schizophrenia in relatives of index cases which 'nuclear' (hebephrenic and catatonic) versus 'peripheral' (paranoid and simple) disorders are apparent even in the non-age corrected data.

Twin studies

The available twin data is mainly in keeping with the family findings. Kringlen (1967) and Fischer (1973) showed that in monozygotic twins, there was virtually complete homotypia for subtype in twin pairs concordant for schizophrenia. However, co-twin assessment was not, however, performed blind to that in the proband. This was rectified in the study of Gottesman (1968) where there was again a marked tendency toward subtype concordance even though diagnosis was here made blindly. One of the drawbacks of these earlier studies, as with the earlier family data is that they were carried out without using explicit operational criteria. We have therefore reexamined the Maudsley twin series originally studied by Gottesman & Shields (1972) and have applied the subtyping criteria of Tsuang & Winokur (1974) to the detailed case abstracts. In our hands these criteria have been found to provide very good inter-rater reliability (Farmer *et al*, 1983; McGuffin *et al*, 1984). The results of the subtyping of the twin material are shown in Table I. In common with previous investigators we find a marked tendency toward subtype concordance. However, this is not complete in the monozygotic (MZ) sample and it is apparent that when the proband has a non-paranoid form of disorder we obtain higher concordance rates for schizophrenia as a whole than when the proband has a paranoid condition. This is therefore in good agreement with the results reported by Gottesman (1968) and by Kringlen (1967) both of whom found a similar pattern and hints that the distinction between paranoid and other forms of schizophrenia is quantitative rather than qualitative.

TABLE I
Probandwise concordance in the Maudsley (1948–1964) Schizophrenic Twin Series

Subtypes of Tsuang & Winokur (1974) Concordance for	*MZ*	*%*	*DZ*	*%*
Schizophrenia, where proband is:				
Paranoid	2/5	40	1/14	7
Non-paranoid (hebephrenic and 'undifferentiated')	13/21	62	3/20	15
Subtype, where both twins are diagnosed schizophrenic	13/15	87	3/3	100

MZ = monozygotic, DZ = dizygotic.

Explanatory models

The transmission of schizophrenia is best considered in terms of liability/threshold models (Falconer 1965; Gottesman & Shields, 1967; Reich *et al*, 1972). Clearly schizophrenia does not show a typical Mendelian pattern of segregation and *discordance* of over 50% in pooled data from recent series of identical twins (Gottesman & Shields 1972) is sufficient evidence that environmental as well as genetic factors are involved. 'Liability' is considered to be a continuously distributed variable in the general population which is determined by both genes and environment (acting in a predominantly additive fashion) such that only those individuals whose liability exceeds a certain threshold value at some point in time, manifest the disorder. Single major locus models, where a single gene is the sole source of familial liability have been shown to be inadequate in explaining the familial aggregation of schizophrenia as a whole (O'Rourke *et al*, 1982). It seems more likely that in schizophrenia we are dealing with many genes at different loci acting in a mainly additive way (Gottesman & Shields, 1967) or possibly a major gene against a polygenic background (McGue *et al*, 1985). Reich and his colleagues (1972, 1979) have extended Falconer's (1965) original single threshold model to include disorders which occur in two or more forms. It is assumed that liability is normally distributed (or could be transformed to normality) and that those individuals who lie beyond a first threshold will show a 'broad', milder form of the disorder while those beyond a second, more extreme, threshold will show a 'narrow', more severe form. Relatives of affected individuals will show a mean liability which is greater than that of the general population and thus more will lie beyond the threshold for being affected. The 'isocorrelational' version of the model (Reich *et al*, 1979) predicts that the mean liability of relatives of 'narrow' cases will be greater than that of 'broad' cases. The model is illustrated in a somewhat simplified form in Fig. 1. Thus in schizophrenia we might consider schizophrenia-as-a-whole as a 'broad' form, with non-paranoid (or hebephrenic or 'nuclear') categories as a narrow form, and paranoid schizophrenia as 'broad-but-not-narrow' category.

TABLE II

A reanalysis of Kallmann's (1938) family study of schizophrenia

			Relatives		
Proband subtype	*Class of relative*	*Total at risk (BZ)*	*% Affected*	*Observed n affected*	**Expected n*
Peripheral (paranoid or simple)	Children	307	9.1	28	32.5
Nuclear (hebephrenic or catatonic)	Children	373	17.7	66	63.9
Peripheral (paranoid or simple)	Siblings	739	6.2	46	39.6
Nuclear (hebephrenic or catatonic)	Siblings	1257	8.2	103	106.1

*Expected *n* affected under two threshold 'isocorrelational' models (Reich *et al*, 1979) where nuclear schizophrenia = 'narrow' from disorder and peripheral schizophrenia = 'broad-but-not-narrow' form assuming population morbid risk of broad form (K_pB) = 1%, $\chi^2 = 0.332$, 1d.f. (NS), parameter estimates: $r_c = 0.524$, $r_s = 0.361$, $K_pN = 0.54\%$.

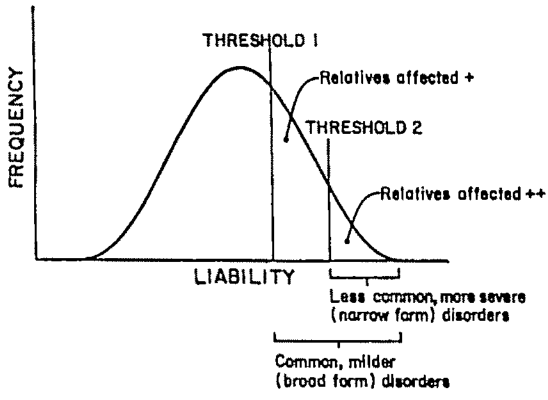
THRESHOLD 1
Relatives affected +
THRESHOLD 2
Relatives affected ++
FREQUENCY
LIABILITY
Less common, more severe (narrow form) disorders
Common, milder (broad form) disorders

FIG. 1 Two-threshold multifactorial model.

As an illustrative example we have attempted to fit a model of this type using Kallmann's (1938) data (Table II). This was carried out using a FORTRAN computer program, TETCOR, where the best fit is found iteratively by minimising a χ^2 function using the MINUIT optimisation routine (CERN, 1974). We have assumed that population morbid risk of the broad form (K_pB) is 1% and we have employed the program to calculate the best fit values of the remaining parameters, comprising the population morbid risk of the narrow form (K_pN) and the correlations in liability. As we can see from Table II, if we allow different correlations in liability for parent–offsprint (r_c) and sibling–sibling (r_s) pairs we obtain an excellent fit (cf. Barn, 1982). We have not attempted a similar model-fitting procedure on the other smaller datasets but as we have already commented the overall pattern in most of these is similar. We therefore consider that most of the family and twin evidence on the transmission of 'traditional' subtypes of schizophrenia is in keeping with the notion of differing thresholds on a single continuum of liability rather than of the existence of qualitatively distinct subentities.

Statistically derived subtypes

As instruments with which to 'carve nature at the joints' multivariate statistical methods have found favour in studies of affective disorder (Kendell, 1975; Paykel, 1981) but have been much less popular in schizophrenia research. Some investigators have attempted to use cluster analytic methods for defining subtypes within the schizophrenia syndrome (e.g. Strauss *et al*, 1973; Bartko *et al*, 1981) but only one group (Farmer *et al*, 1983, 1984) have viewed their results from a specifically genetic perspective. Farmer *et al* (1983) applied two mathematically different methods of cluster analysis to data on chronic schizophrenics. The variables used were from a comprehensive checklist of symptoms derived from commonly used operational diagnostic criteria. Two fairly distinct clusters were derived and there was surprisingly good agreement between the two clustering programmes. Simulation studies indicated that the level of agreement would occur by chance less than once in a hundred. The clusters remained stable when extra cases were added. Furthermore, where index cases and their schizophrenic relatives were assigned to subgroups the level of homotypia was significantly better than chance expectation.

Subsequently the characteristics of the groups were further explored using discriminant analysis (Farmer

TABLE III
Probandwise concordance in the Maudsley (1948–1964) Schizophrenic Twin Series

Subtypes of Farmer et al *(1983, 1984)* *Concordance for*	*MZ*	*%*	*DZ*	*%*
Schizophrenia where proband is:				
P-type	4/12	33	1/17	6
H-type	11/14	79	3/17	18
Subtype, where both twins are diagnosed schizophrenic	11/15	73	4/4	100

MZ = monozygotic; DZ = dizygotic.

et al, 1984). Because of their resemblance to some descriptions of more 'traditional' subtypes the group with onset before 25 years, poor premorbid work adjustment and such clinical features as blunting of affect, thought insertion and third-person auditory hallucinations was named hebephrenic-like or 'H-type', while the group with later onset, poor premorbid *social* adjustment and well organised delusions was named paranoid-like or 'P-type'. Using discriminant function scores the twins from the Gottesman & Shields (1972) study were assigned to H- and P-subtypes and the concordance examined (Table III), a pattern very similar to that for the Tsuang & Winokur (1974) criteria emerged. That is, there is a tendency towards homotypia, which is nevertheless incomplete in genetically identical twins. But when subtype in co-twins is ignored and only concordance rates for schizophrenia as a whole considered according to whether the proband is H- or P-type, and the co-twins of H-type probands have higher rates of schizophrenia than do the co-twins of P-type probands. The findings are therefore suggestive once more of a quantitative rather than a qualitative distinction. Although no formal model fitting has been attempted it might be again postulated that H-type schizophrenia is but another narrow-form disorder with P-type schizophrenia a broad-but-not-narrow form.

Type I, type II and negative symptom schizophrenia

A finding which has achieved a high level of consistency across different centres is of enlarged lateral cerebral ventricles on the computerised tomographic (CT) brain scans of a proportion of schizophrenics. This was first reported by Johnstone and colleagues (1976) and has been replicated in nearly all subsequent studies (Reveley, 1985) (however, the recent criticisms of Smith & Iacono (1986) regarding schizophrenia/control comparisons must be noted). The finding of ventricular enlargement has caused renewed interest in what Johnstone *et al* (1976) have termed the 'dementia of dementia praecox'. Crow (1980) has proposed that there may exist two syndromes, a type I disorder, characterised by positive symptoms and responsiveness to neuroleptics, while a type II syndrome is more closely associated with negative symptoms, poor response to treatment and perhaps with cognitive impairment and morphological changes reflected in CT brain scans. Crow suggested (1980) that although the syndromes overlap to a considerable extent, the type I disorder is likely to be associated with abnormal dopaminergic mechanisms while the type II disorder would have some other (perhaps viral-infective) pathology. We have attempted with author's guidance to cast Crow's (1980) clinical criteria in an operational format to facilitate investigation of genetic aspects of this subtypology and have been able to demonstrate good inter-rater reliability using this approach (Farmer *et al*, 1987).

As with other subtyping systems described above we have reexamined the clinical abstracts for the Gottesman & Shields (1972) schizophrenic twin series and have reclassified affected individuals (Table IV). Pure type II cases are rare in this series but once again we see a tendency towards homotypia which is incomplete. Furthermore, there is some suggestion that the mixed form is a 'more genetic' condition than is type I schizophrenia in that there is a very marked difference between MZ and DZ concordance rates where the proband has a mixed type disorder.

Unfortunately such a small amount of data does not permit any definite conclusions about the Crow subtypology and we are unaware of any other familial

TABLE IV
Probandwise concordance in the Maudsley (1948–1964) Schizophrenic Twin Series

Subtypes of Crow (1980) *Concordance for*	*MZ*	*%*	*DZ*	*%*
Schizophrenia, where proband is:				
type I	8/15	53	4/21	19
Mixed type	7/11	64	0/11	0
Type II	0/0	0	0/2	0
Subtype, where both twins are diagnosed schizophrenic	11/15	73	3/4	75

MZ = monozygotic, DZ = dizygotic.

data which might help clarify the issue. Furthermore, as a result of the CT scan studies of the Northwick Park group, Crow's own views on what forms the most useful clinical basis for a type I–type II separation are evolving and changing (Crow, 1985).

There is, however, some evidence which has a bearing on a related concept, that of a separation of schizophrenia into two groups on the basis of whether negative or positive symptoms predominate. Dworkin & Lenzenweger (1984) have examined the available published extracts from systematic twin studies. Using their own unpublished system, which they find to be reliable in rating the presence of negative symptoms, they were able to divide up probands from monozygotic pairs into 'high negative' and 'low negative' categories. A fairly consistent picture emerged, with all twin studies, except for that of Kringlen (1967) showing higher concordance where the proband exhibited a high rate of negative symptoms. This approach is rather different from and considerably simpler than the Crow subtypology. Nevertheless, there is clearly a resemblance between these results and those of our own efforts to apply the operationalised Crow criteria to the Maudsley schizophrenic twin series.

It is therefore tempting to speculate that in separating off a group of patients with type II mixed, or predominantly negative symptoms, we are simply selecting a more severely affected group which, as with non-paranoid, hebephrenic or 'H-type' cases tend to occupy a more extreme position on a continuum of liability. It might be argued therefore that all of these findings are reminiscent of the earlier work of Gottesman & Shields (1968, 1972) who took a more straightforward approach to the question of severity. The probands from identical twin pairs were divided according to time spent in hospital. Where the proband had spent less than 2 years in hospital there was a concordance of only 27% as against a rate of 77% where a proband had spent more than 2 years in hospital since the onset of his/her illness. Similarly, where the proband had shown an inability to stay out of hospital for at least 6 months since the onset of their disorder, the rate of schizophrenia in the co-twins was 75%.

Non-genetic schizophrenia?

The evidence considered thus far would seem to favour the existence of 'more genetic' and 'less genetic' varieties of schizophrenia lying on the same continuum with cut-off points dependent on chosen clinical criteria. A more radical suggestion has recently been put forward by Murray *et al* (1985), that 'sporadic' or non-genetic forms of schizophrenia may be quite common and that presence or absence of a family history of the disorder provides a useful starting point for such a classification scheme. There are undoubtedly many organic conditions which can present with a schizophrenic like disorder (Davison & Bagley, 1969; Propping, 1983) but well-established cases of 'symptomatic' schizophrenia which, from a genetic viewpoint, might be considered as 'phenocopies' are uncommon in clinical practice. Murray and his colleagues (1985) suggest that sporadic cases of schizophrenia may be commoner than are generally supposed and that the environmental causes most often go undetected. They point out that identical twins discordant for schizophrenia show marked differences in cerebral ventricular size measured on CT brain scans. Although the numbers involved were small Reveley *et al* (1982) found significant tendency for the affected proband to have larger ventricles than the non-schizophrenia co-twin. It was postulated that this might reflect early trauma such as birth injury, and a subsequent study (Reveley *et al*, 1984) found a relationship between enlarged cerebral ventricles, negative-family history of psychiatric illness and positive history of early cerebral insults in schizophrenic twins. However, it should be noted that 'family-history negative' here meant absence of illness in relatives other than the co-twin so that the sample of 'family history negative' probands appears to have included some individuals with affected co-twins. Thus the apparent elegance and simplicity of a family-history positive/family-history negative division may be dangerously deceptive.

There are a number of general objections to a family-history positive/family-history negative split. First, any plausible model of transmission (McGue *et al*, 1985) would predict that those patients without a family history of schizophrenia would comprise a high proportion of genetic cases who are 'chance isolates' (Elandt-Johnson, 1971) as well as those who are true non-genetic cases. However, there would be no straightforward way of differentiating between the two. Second, the chances of obtaining a positive family history of psychiatric disorder are closely related to the quality of information available. Thorough family interview studies inevitably uncover more secondary cases among relatives than do studies based on history alone. Third, the proportion of family-history positive cases must also depend on family size and on the proportion of relatives who have passed through the age of risk. Most authors agree that there is an increased rate of schizophrenia and of 'spectrum' disorders among the relatives of schizophrenia rather than an increase in psychopathology generally (Gottesman & Shields, 1982). Nevertheless, Reveley *et al* (1984) have taken a broad perspective on what constitutes a 'family-history positive' case and include a family history of *any* major psychiatric disorder is about 10% (and recent studies suggest that this is a conservative estimate (Sturt *et al*, 1984; Helzer *et al*, 1985)) then we would obtain a distribution of family-history positive cases by chance alone like that shown in Table V. Obviously this is not entirely realistic since it does not take into account the tendency of most psychiatric disorders to aggregate in families (McGuffin, 1984). However, the table serves to illustrate that family size cannot be ignored as a potential confounding variable in studies of this type.

TABLE V

Proportion of a population expected to be family-history positive for a 10% trait by chance

*Proportion (%)**	*Number of relatives*
10	1
19	2
27	3
34	4
41	5
47	6
52	7

*proportion, p, calculated as $p = 1-(1-\pi)^n$ *where* π = population frequency and n = number of relatives

A more direct criticism of the hypothesis of Murray *et al* (1985) is that we might expect to see a consistent relationship between presence or absence of family history and cerebral ventricular size in other studies of schizophrenics. However, as Table VI shows, there is a disappointing lack of agreement between studies carried out in different centres. Some (e.g. Turner *et al* 1986) support Murray's hypothesis; one (Nasrallah *et al*, 1982) produced findings in the opposite direction of that predicted, while most others actually find no significant differences between the family-history positive and family-history negative groups. This is perhaps not surprising in the light of the recent analysis presented by Eaves *et al* (1986) which demonstrated that the discriminatory power of a familial/non-familial distinction is actually very low.

TABLE VI

CT brain scan studies VBR in family-history positive (FHP) vs family-history negative (FHN) schizophrenics

Authors	*Summary of finding*
Reveley *et al* (1984)	Mean VBR in FHN twins significantly *greater* than in FHP twins
Nasrallah *et al* (1982)	Mean VBR in FHN patients significantly *less* than in FHP patients.
Owen *et al* (1985)	More FHP than FHN patients with VBR greater than control mean.
Farmer *et al* (1985)	No significant difference between FHP and FHN patients.
Williams *et al* (1985)	
Reveley & Chitkara (1985)¹	
Turner *et al* (1986)	Mean VBR in FHN patients significantly *greater* than in FHP patients.

1. One-way anova showed significant differences with VBR in FHN patients >VBR in FHP patients >VBR in controls.

Do earlier twin or family studies provide any clarification? Only a minority of even the most thoroughly investigated schizophrenics have secondary cases in their first- or second-degree relatives and over 60% must be classified as 'family-history negative' (Bleuler, 1978). Given that this is so, it could be asked whether discordance in identical twins might be due to an admixture of genetic and non-genetic cases. The question was first investigated by Luxenburger (1928). He found that discordant pairs had other affected family members as often did concordant pairs, results which were subsequently replicated by other investigators (Kringlen, 1967). Furthermore, a study of the adult offspring of MZ twins discordant for schizophrenia revealed a similar morbid risk for the disorder in both the children of the schizophrenic probands and the children of the non-schizophrenic co-twins (Fischer, 1971). (The latter finding has been confirmed and extended in the offspring of MZ and DZ twins by Gottesman & Bertelsen (in preparation)). Both findings strongly suggest that a genetic/sporadic admixture hypothesis to explain discordance for schizophrenia in identical twins is incorrect but supports the idea that 'sporadic' or non-genetic forms of schizophrenia are uncommon. This being so, the studies of discordant twins should provide an ideal means of pinpointing specific environmental stressors which may precipitate the disorder in the genetically susceptible as well as indicating possible protection factors. Unfortunately, results from older studies are disappointingly inconsistent. Pollin & Stabenau (1968) are often quoted as finding that in discordant MZ pairs the schizophrenic index case was more likely to have had a history of birth trauma and low birth weight and to have had a submissive dependent premorbid personality than his unaffected co-twin. Other studies provide some support regarding premorbid personality factors but despite evidence of the general role of birth trauma (McNeill & Kaij, 1978) there has been no convincing replication of the *specific* birth history findings (Gottesman & Shields, 1982).

Biological and genetic markers

Thus far, we have mainly discussed subcategories based upon clinical signs and symptoms, or what Gottesman & Shields (1972) have called 'exophenotypes'. It might be hoped that the discovery of biological markers or 'endophenotypes' for schizophrenia might take us a step nearer toward primary gene products and facilitate less ambiguous methods of classification (McGuffin, 1984*b*). Attempts to subclassify schizophrenics according to biological criteria have been reviewed by Meltzer (1979) but there is, for the most part, a disappointing inconsistency in results. The biochemical marker which has perhaps excited the most attention and which may be of genetic relevance is platelet monoamine oxidase (MAO). Potkin *et al* (1978) reported that platelet MAO activity was significantly lower in paranoid than in non-paranoid schizophrenic patients. Unfortunately, other groups, (e.g. Meltzer *et al*, 1980) have been unable to replicate this finding and the weight of evidence from other studies (reviewed by Kendler & Davis, 1981) does not favour a clear separation of groups of schizophrenics on the basis of platelet MAO activity. Further doubt concerning the usefulness of MAO as a marker derives from the studies of Rice *et al* (1982, 1984) which suggest that platelet MAO is controlled by a single major locus but that the less common of its two alleles is the one coding for *high* MAO activity and that the vast majority of the population, over 90%, carry at least one 'low MAO' gene. Clearly there is some difficulty in reconciling this finding with suggestions that platelet MAO may be a marker for schizophrenia, a disorder where the lifetime incidence in the population is 1% or less.

Genetic markers are a special category of biological marker. Although still often used in a rather broad sense in the psychiatric literature (e.g. Shuckitt, 1986) the term genetic marker is probably best restricted to cover traits which have a simple mode of inheritance and which are demonstrably polymorphic (i.e. where there are two or more alleles with a gene frequency of at least 1%) (Giblett, 1969). The better known genetic markers include red cell groups such as ABO, rhesus, MN and soon as well as major histocompatibility (HLA) antigens, various blood protein polymorphisms and red cell enzymes. In addition to these 'conventional' markers the advent of 'the new genetics' of recombinant DNA (Emery, 1984) has meant that many more genetic markers are now available and that the day is not far off when the human genome (i.e. the set of 22 pairs of autosomes and two sex chromosomes) will be extensively mapped. So far over 900 genes have been assigned to chromosomal locations (De La Chapelle, 1985), but only about 25% of genes so far mapped are detectably polymorphic (*American Journal of Human Genetics*, 1983) and only a proportion of these have been investigated in relation to schizophrenia (see recent review by McGuffin & Sturt, 1986).

Possible associations discovered by recent population studies involving blood complement factors (Rudduck, 1985) merit further investigation, but most genetic marker findings in schizophrenia to date have been disappointingly inconsistent and convincing replications of positive findings have been rare. A notable exception is the evidence concerning the HLA system and subtypes of schizophrenia.

Associations between certain specificities of the HLA system and a number of common diseases are now well established (Thomson, 1981). Such disorders include juvenile diabetes, multiple sclerosis and ankylosing spondylitis. Most HLA associated diseases are not rare have a tendency to run in families, but show complex patterns of inheritance which are not classically Mendelian. Not surprisingly, therefore, schizophrenia came under scrutiny not long after the first HLA-disease reports (Cazzulo *et al*, 1974; Eberhard *et al*, 1975). However, studies of schizophrenia as a whole provided no clear pattern of association across different centres (McGuffin, 1979). It was only when schizophrenia was broken down into possible subtypes that a more consistent set of results emerged. In particular there was evidence of an association between paranoid schizophrenia and HLA A9, while the hebephrenic subtype showed a less consistent association with HLA A1 (McGuffin *et al*, 1981). There have now been a total of nine published studies which provide data on an A9-paranoid schizophrenia association (reviewed by McGuffin & Sturt, 1986). Seven of these are consistent with the presence of an association and when the data are pooled it seems highly unlikely that the association has arisen by chance ($\chi^2 = 19.378$, calculated by Woolf's (1955) method, P corrected for number of comparisons $= 0.0003$). There are, however, a number of difficulties. First, it is not clear whether the diagnosis of a paranoid subtype is comparable in the different centres. Second, A9 is a composite antigen consisting of two subspecificities, AW23 and AW24 (Festenstein & Demant, 1978), and it is uncertain which of these is more important in the paranoid schizophrenia association. Third, and perhaps most importantly, despite its high statistical significance the strength of the association is low. The estimated relative risk of developing paranoid schizophrenia for an A9 positive individual compared with an A9 negative individual is 1.6. If we assume, as we have done earlier, a liability/threshold model for the transmission of schizophrenia, then using the method given by Edwards (1965) we calculate that the contribution of the HLA locus to variation in liability to paranoid schizophrenia is only about 1.1%. It therefore appears that the HLA studies may indicate a minor susceptibility locus rather than an important major gene effect in paranoid schizophrenia. The results would therefore require only some slight modification of our earlier speculations that the separation between paranoid and other types of schizophrenia is entirely quantitative and based purely on different thresholds on a liability continuum. Instead we would have to allow that there may also be overlapping sets of polygenes each of which modify the clinical picture in subtly different ways.

By contrast with the association studies, linkage studies which seek to detect a marker gene in close proximity to a major disease gene have been predominantly negative with respect to HLA. Turner (1979) reported that there was suggestive, but inconclusive, evidence of linkage between HLA and 'schizotaxia' in 'typical' families multiply affected by schizophrenia but not in 'atypical' families in which multiple family members were affected not just by schizophrenia but by other conditions such as affective disorder. McGuffin *et al* (1983) failed to replicate this finding and subsequent investigations (Chadda *et al*, 1986; Goldin *et al*, 1987; Andrews *et al*, 1987) are also negative.

So far, there are no published results of studies of schizophrenia and the new genetic markers of recombinant DNA technology, so called restriction fragment length polymorphisms (Botstein *et al*, 1980). But there is considerable optimism about the future usefulness of such markers in detecting major gene effects and resolving problems of heterogeneity in schizophrenia and other common diseases (White *et al*, 1985). Unfortunately the statistical analysis of linkage studies involving conditions with complex and non-Mendelian patterns of inheritance, presents formidable problems which will need to be solved before the 'new genetics' can yield tangible benefits for schizophrenia research (Sturt & McGuffin, 1985).

Conclusions

Although Eugen Bleuler is often regarded as the original instigator of the search for separate subforms of schizophrenia, his son Manfred Bleuler (1978) has more recently concluded that there is little compelling evidence of their existence. This accords well with our own assessment that despite considerable efforts to define subtypes and despite the conviction of many research workers that natural lines of cleavage might exist, no convincing split extending back to a genotypic level has yet been demonstrated in schizophrenia. Time honoured clinical separations of schizophrenia into paranoid and hebephrenic or other non-paranoid subtypes produce a tendency toward homotypia in family and twin studies as do some more recently derived statistically based categories (Farmer *et al*, 1984). However, neither approach produces a clear qualitative separation and the available data can best be explained using liability threshold models in which the subtypes of disorders lie on the same continuum. Although theoretically attractive, subtypologies based on the presence or absence of negative symptoms or on a Crow type I–type II pattern of symptoms do not appear to provide separate, genetically distinct subentities. Similarly a separation of schizophrenia into a distinct familial and sporadic categories is difficult to justify on the basis of current evidence. Rather than providing a means of 'carving nature at the joints', the systems of classifying subtypes which are so far available appear to offer different ways of slicing up the salami. Undoubtedly the salami has more genetic content at one end than at the other, but as yet there is no convincing evidence that we are dealing with two or more completely different varieties of sausage.

Nearly all of the debate on the unity or diversity of schizophrenia has so far been based upon recurring clusters of clinical features. We have been dealing with what Gottesman & Shields (1972) have called 'exophenotypes' which are distantly removed from what must be occurring at a genotypic level. Future prospects for a better understanding of subtle morphological or pathophysiological change which may occur in the living brain of the schizophrenic have been greatly enhanced by recent developments in brain imaging (Buchsbaum *et al*, 1982; Herold & Frackowiak, 1986) and refinements of regional blood flow techniques (Weinberger *et al*, 1986). However, for the present perhaps the most consistent pointer toward a qualitative distinction between subtypes of schizophrenia has so far come from genetic marker studies involving the HLA system. The size of the HLA-paranoid schizophrenia association is small and would be quite in keeping with a multifactorial view of the aetiology of the condition. However, this represents a starting point for future genetic marker studies and signals the passage of psychiatric genetics into a new phase in which the molecular biological aspects of mental disorders must inevitably merit more detailed attention.

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References

American Journal Of Human Genetics (1983) The human gene map. *American Journal of Human Genetics*, **35**, 134–156.

Andreasan, N. C. (1985) Positive vs negative schizophrenia: A critical evaluation, *Schizophrenia Bulletin*, **11**, 380–389.

Andrew, B., Watt, D. C., Gillespie, C. & Chapel, H. (1987) A family study of genetic linkage in schizophrenia. *Psychological Medicine*, (in press).

Baron, M. (1982) Genetic models of schizophrenia, *Acta Psychiatrica Scandinavia*, **65**, 263–275.

Bartko, J. J., Carpenter, W. T. & Strauss, J. S. (1981) Statistical basis for explaining schizophrenia. *American Journal of Psychiatry*, **138**, 941–945.

Bleuler, E. (1911) *Dementia praecox or the group of schizophrenias*, translated by J. Zinkin (1950). New York: International Universities Press.

Bleuler, M. (1978) *The Schizophrenic Disorders*, (translated by S. Clemens). New Haven and London: Yale University.

Botstein, D., White, R. L., Skolnick, M. & Davis, R. W. (1980) Construction of a genetic linkage map in men using restriction fragment length polymorphisms. *American Journal of Human Genetics*, **32**, 312–331.

British Medical Journal (1969) Unity and diversity in schizophrenia (Editorial). *British Medical Journal*, **4**, 673–674.

Buchsbaum, M. S., Ingvar, D. H., Kessler, R., Waters, R. N., Cappeletti, J., Van K. Ammen, D. P., King, A. C., Johnson, J. L., Manning, A. G., Plynn, R. W., Mann, L. S., Bunney, W. E. & Sokoloff, L. (1982) Cerebral glucography with position tomography. *Archives of General Psychiatry*, **39**, 251–259.

Cazzullo, C. L., Smeraldi, E. & Pennati, G. (1974) The leukocyte antigen system HLA as a possible genetic marker of schizophrenia. *British Journal of Psychiatry*, **25**, 25–27.

Cern (1974) *Minuit: a package of programmes to minimise a function of a variable*. Geneva: CERN Data Handling Division.

Chadda, R., Kulhara, P., Singh, I. & Sehgal, S. (1986) HLA antigens in schizophrenia: a family study. *British Journal of Psychiatry*, **149**, 612–615.

Crow, T. J. (1980) Molecular Pathology of schizophrenia: more than one disease process. *British Medical Journal*, **280**, 66–68.

Crow, T. J. (1985) The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin*, **11**, 471–486.

Davison, K. & Bagley, C. R. (1969) Schizophrenic-like psychoses associated with organic disorders of the central nervous system: a review of the literature. In: *Current Problems in Neuropsychiatry: Schizophrenia, Epilepsy and the Temporal Lobe*, (ed. R. N. Herrington). Ashford, Kent: Headley Bros.

De la Chapelle, A. (1985) Mapping hereditary disorders. *Nature*, **317**, 472–473.

Dworkin, R. H. & Lenzenweger, M. F. (1984) Symptoms and the genetics of schizophrenia: implications for diagnosis. *American Journal of Psychiatry*, **141**, 1541–1546.

Eaves, L. J., Kendler, K. S. & Schulz, S. C. (1986) The familial sporadic classification: its power for the resolution of genetic and environmental etiologic factors. *Journal of Psychiatric Research*, **20**, 115–130.

Eberhard, G. & Franzen, G., Low, B. (1975) Schizophrenia Susceptibility and HLA Antigens. *Neuropsychobiology*, **1**, 211–217.

Edwards, J. H. (1965) Associations between blood groups and disease. *Annals of Human Genetics*, **29**, 77–83.

Elandt-Johnson, R. C. (1971) *Probability Models and Statistical Methods in Genetics*. New York: John Wiley & Sons.

Emery, A. E. H. (1984) *An Introduction to Recombined DNA*. Chichester: Wiley.

Falconer, D. S. (1965) The inheritance of liability to certain diseases, estimated from the incidence among relatives. *Annals of Human Genetics*, **29**, 51–76.

Farmer A. E., McGuffin, P. & Spitznagel, E. (1983) Heterogeneity in schizophrenia: a cluster analytic approach. *Psychiatry Research*, **8**, 1–12.

——, —— & Gottesman, I. I. (1984) Searching for the split in schizophrenia: a twin study perspective. *Psychiatry Research*, **13**, 109–118.

——, Jackson, R. & Storey, P. (1985) Classifying schizophrenia (letter). *Lancet*, **i**, 33.

——, Jackson, R., McGuffin, P. & Storey, P. (1987) Cerebral ventricular enlargement in schizophrenia consistencies and contradictions. *British Journal of Psychiatry*, **150**, 324–330.

Festenstein, H. & Demant, P. (1978) *HLA and H2. Current Topics in Immunology*, Vol. 9. London: Edward Arnold.

Fischer, M. (1971) Psychoses in the offspring of schizophrenia monozygotic twins and their normal co-twins. *British Journal of Psychiatry*, **115**, 981–990.

—— (1973) *Genetic and environmental factors in schizophrenia. Acta Psychiatric Scnadinavia* (Suppl) 238.

Giblett, E. R. (1969) *Genetic Markers in Human Blood*. Oxford: Blackwell.

Goldin, L. R., De Lisli, L. E. & Gershon, E. S. (1987) The relationship of HLA to schizophrenia in 10 nuclear families. *Psychiatry Research*, (in press).

Gottesman, I. I. (1968) Severity/concordance and diagnostic refinement in the Maudsley-Bethlem schizophrenia twin study. *The Transmission of Schizophrenia*, (eds D. Rosenthal & S. S. Kety). Oxford: Pergamon Press.

—— & Shields, J. (1967) A polygenic theory of schizophrenia. *Proceedings of the National Academy of Sciences*, **58**, 199–205.

—— & —— (1972) *Schizophrenia. A Twin Study Vantage Point*. Academic Press, London.

—— & —— (1982) *Schizophrenia, The Epigenetic Puzzle*. Cambridge: Cambridge University Press.

Gottesman, I. L. & Bertelsen, A. (in prep.) The offspring of twins concordant and discordant for schizophrenia.

Helzer, J. E., Stolzman, R. K., Farmer, A. E., Brockington, I., Plesons, D., Singerman, B. & Works, J. (1985) Comparing the DIS with a DIS/DSM III-based physician re-evaluation. In *Epidemiologic Field Methods in Psychiatry*, (ed. W. W. Eaton & L. G. Kessler). Orlando: Academic Press.

Herold, S. & Frackowiak, J. (1986) New methods in brain imaging (editorial). *Psychological medicine*, **16**, 241–246.

Johnstone, E. C., Crow, T. J., Frith, C. D. & Kreel, L. (1976) Cerebral ventricular size and cognitive impairment in chronic schizophrenia. *Lancet*, **ii**, 924–926.

——, ——, ——, Stevens, M., Kreez, L. & Husband, J. (1978) The dementia of dementia praecox. *Acta Psychiatric Scandinavia*, **57**, 305–324.

Kallmann, F. J. (1938) *The Genetics of Schizophrenia*. New York: J. J. Augusta.

Kendell, R. E. (1975) *The Role of Diagnosis in Psychiatry*. Oxford: Blackwell Scientific Publications.

Kendler, K. S. & Davis, K. L. (1981) The genetics and biochemistry of paranoid schizophrenia and other paranoid psychoses. *Schizophrenia Bulletin*, **7**, 689–709.

Kraepelin, E. (1896) *Psychiatrie, ein Lehrbuch fur studierende und Arzte*, 5th ed. Leipzig: Barch.

Kringlen, E. (1967) *Heredity and Environment in the Functional Psychoses*. Heinman: London.

Leonhard, K. (1959) *Autfelung der Endogen Psychosen*. Berlin: Akademic Verlag.

Luxenburger, H. (1928) Vorlaufiger Bericht uber psychiatrische Serien untersuchurgen an 2 willilngen. *Zeitschrift fur gesamte Neurologie und Psychiatrie*, 116, 297–326.

McGue, M., Gottesman, I. I. & Rao, D. C. (1985) Resolving genetic models for the transmission of schizophrenia. *Genetic Epidemiology*, 2, 99–110.

McGuffin, P. (1979) Is schizophrenia an HLA associated disease? *Psychological Medicine*, 9, 721–728.

—— (1984) Genetic influences on personality, neurosis and psychosis. In *Scientific Principles of Psychopathology*, (ed. P. McGuffin, M. F. Shanks & R. Hodgson). London: Grune & Stratton.

—— (1984*a*) Principles and method in psychiatric genetics. In *Scientific Principles of Psychopathology*, (ed. P. McGuffin, R. Hodgson & R. Shanks). London: Grune & Stratton.

—— (1984*b*) Biological markers and psychosis. *Psychological Medicine*, 14, 255–259.

—— & Sturt, E. (1986) Genetic Markers in Schizophrenia. *Human Heredity*, 36, 65–68.

——, Farmer, A. E. & Yonace, A. (1981) HLA antigens and subtypes of schizophrenia. *Psychiatry Research*, 5, 115–122.

——, Festenstein, W. & Murray, R. M. (1983) A Family study of HLA antigens and other genetic markers in schizophrenia. *Psychological Medicine*, 13, 31–43.

——, Farmer, A. E., Gottesman, I. I., Murray, R. M. & Reveley, A. M. (1984) Twin concordance for Operationally Defined Schizophrenia: Confirmation of Familiarity and Heretability. *Archives of General Psychiatry*, 41, 541–545.

McKusick, V. A. (1969) On lumpers and splitters, or the nosology of genetic disease. *Perspectives on Biology and Medicine*, 12, 298–301.

—— (1983) *Mendelian Inheritance in Man*, Baltimore and London: John Hopkins University Press.

McNeill, R. F. & Kaij, L. (1978) Obstetric factors in the development of schizophrenia: complications in the birth of pre-schizophrenics and in reproduction of schizophrenic parents. In *The Nature of Schizophrenia*, (eds L. C. Wynne, R. L. Cromwell & S. Matthyse). Chichester: Wiley.

Meltzer, H. Y. (1979) Biology of schizophrenic subtypes: a review and proposal for method of study. *Schizophrenia Bulletin*, 5, 460.

——, Arora, R. C., Jackman, H., Pscheidt, G. & Smith, M. D. (1980) Platelet monoamine oxidase and plasma amino oxidase in psychiatric patients. *Schizophrenia Bulletin*, 6, 213–219.

Murray, R. M., Lewis, S. & Reveley, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, i, 1023–1026.

Nasrallah, H. A., Charles, G. T., McCalley-Whitters, M. & Kuperman, S. (1982) Cerebral ventricular enlargement in subtypes of chronic schizophrenia. *Archives of General Psychiatry*, 39, 774–777.

O'Rourke, D. H., Gottesman, I. I., Suarez, B. K., Rice, J. & Reich, T. (1982) Refuation of the general single locus model in the aetiology of schizophrenia. *American Journal of Human Genetics*, 34, 630–649.

Owen, D. G. C., Johnstone, E. C., Crow, T. J., Frith, C. D., Jaoue, J. R. & Kreel, L. (1985) Lateral ventricular size in schizophrenia: relationship to the disease process and its clinical manifestations. *Psychological Medicine*, 15, 27–41.

Paykel, E. S. (1981) Have multivariate statistics contributed to classification? *British Journal of Psychiatry*, 139, 357–362.

Penrose, L. S. (1952) Measurement of pleiotropic effects in phenyl ketonuria. *Annals of Eugenics*, 18, 120–124.

Pogue-Geile, M. F. & Harrow, M. (1985) Negative symptoms in schizophrenia: their longitudinal course and prognostic importance. *Schizophrenia Bulletin*, 11, 427–439.

Pollin, W. & Stabenau, J. R. (1968) Biological psychological and historical differences in a series of monozygotic twins discordant for schizophrenia. *The Transmission of Schizophrenia*, (eds D. Rosenthal & S. S. Kety). Oxford: Pergamon.

Potkin, S. G., Cannon, H. E., Murray, D. L. & Wyatt, R. J. (1978) Are paranoid schizophrenics biologically different from other schizophrenics? *New England Journal of Medicine*, 298, 61–66.

Propping, P. (1983) Genetic disorders presenting as 'schizophrenia'. Karl Bonhoeffer's early view of the psychoses in the light of medical genetics. *Human Genetics*, 65, 1–19.

Reich, T., James, J. W. & Morris, C. A. (1972) The use of multiple thresholds in determining the mode of transmission of semi-continuous traits. *Annals of Human Genetics*, 36, 163–184.

——, Cloniger, C. R., Wette, R. & James, J. (1979) The use of multiple thresholds and segregation analysis in analysing the phenotypic heterogeneity of multifactorial traits. *Annals of Human Genetics*, 42, 371.

Reveley, A. M., Reveley, M. A., Clifford, C. A. & Murray, R. M. (1982) Cerebral ventricular size in twins discordant for schizophrenia. *Lancet*, i, 540–541.

——, —— & Murray, R. M. (1984) Cerebral ventricular enlargement in nongenetic schizophrenia: A controlled twin study. *British Journal of Psychiatry*, 144, 89–93.

—— (1985) CT scans in schizophrenia. *British Journal of Psychiatry*, 146, 367–371.

Reveley, M. A., Chitkara, B. (1985) Subtypes in schizophrenia. (Letter) *Lancet*, i, 1503.

Rice, J. P., McGuffin, P. & Shaskan, E. G. (1982) A commingling analysis of platelet monoamine oxidase activity. *Psychiatry Research*, 7, 325–335.

Rice, J., McGuffin, P., Goldin, L. R., Shaskan, E. G. & Gershon, E. S. (1984) Platelet monoamine oxidase activity: Evidence for a single major locus. *American Journal of Human Genetics*, 36, 36–43.

Rudduck, C. (1985) *Genetic markers and schizophrenia*. PhD Thesis, University of Lund.

Scharfetter, C. & Nusperli, M. (1980) The group of schizophrenias, schizoaffective psychoses, and affective disorders. *Schizophrenia Bulletin*, 6, 586–591.

Shuckitt, M. (1986) Trait (and state) markers of predisposition to psychopathology. In *Psychiatry*, Vol. 3, 53, 1–19, (ed. R. Michels, J. O. Cavenar, H. R. H. Brodie, Arnold M. Cooper, S. B. Guze, L. L. Judd, G. L. Klerman & A. J. Solnir). Philadelphia: Lippincott.

Slater, E. (1947) Genetical causes of schizophrenic symptoms. *Monatsschrift fur Psychiatrie und Neurologie*, 113, 50–58. (Reprinted in *Man, Mind and Heredity*, (ed. J. Shields & I. I. Gottesman).

Slater, E. & Cowie, V. (1971) *The Genetics of Mental Disorders*. Oxford: Oxford University Press.

Smith, G. W. & Iacono, W. G. (1986) Lateral ventricular size in schizophrenia and the choice of a control group (letter), *Lancet*, i, 1450.

Snezhevnevsky, A. V. (1968) The symptomatology, clinical forms and nosology of schizophrenia. In *Modern Perspectives in World Psychiatry*, (ed. J. G. Howells). Edinburgh: Oliver and Boyd.

Strauss, J. S., Bartko, J. J. & Carpenter, W. T. (1973) The use of clustering techniques for the classification of psychiatric patients. *British Journal of Psychiatry*, 122, 531–538.

Sturt, E. & McGuffin, P. (1985) Can linkage and marker association resolve the genetic aetiology of psychiatric disorders? Review and argument. *Psychological Medicine*, 15, 455–462.

——, Kamakura, N. & Der, G. (1984) How depressing life is: Lifelong morbidity risk for depressive disorder in the general population. *Journal of Affective Disorders*, 7, 109–122.

THOMSON, G. (1981) A review of the theoretical aspects of HLA and disease association. *Theoretical Population Biology*, 20, 168–208.

TSUANG, M. T. & WINOKUR, G. (1974) Criteria for sub-typing schizophrenia. *Archives of General Psychiatry*, 31, 43–47.

——, —— & CROWE, R. (1980) Morbidity risks of schizophrenia and affective disorders among first degree relatives of patients with schizophrenia, mania, depression and surgical conditions. *British Journal of Psychiatry*, 137, 497–504.

——, FOWLER, R. L., CADORET, R. J. & MONNELLY, E. (1974) Schizophrenia among first degree relatives of paranoid and nonparanoid schizophrenics. *Comprehensive Psychiatry*, 15, 295–302.

TURNER, S., TOONE, B. & BRETT-JONES, J. (1986) Computerised tomography and changes in early schizophrenia—preliminary findings. *Psychological Medicine*, 16, 219–225.

TURNER, W. J. (1979) Genetic markers for schizotaxia. *Biological Psychiatry*, 14, 177–205.

WEINBERGER, D. R., BERMAN, K. F. & ZEC, R. F. (1986) Physiologic dysfunction of dorsolateral prefrontal cortex in schizophrenia I. Regional cerebral blood flow evidence. *Archives of General Psychiatry*, 43, 114–125.

WHITE, R., LEPPERT, M., BISHOP, T., BARKER, D., BERKOWITZ, J., BROWN, C., CALLAHAN, P., HOLM, T. & JEROMINSKI, L. (1985) Construction of linkage maps with DNA markers for human chromosomes. *Nature*, 313, 101–105.

WILLIAMS, A. O., REVELEY, M. A., KOLAKOWSKA, T., ARDEN, M. & MANDELBROTE, B. M. (1985) Schizophrenia with good and poor outcome: II Cerebral ventricular size and its clinical significance. *British Journal of Psychiatry*, 146, 239–246.

WOOLF, B. (1955) On estimating the relation between blood groups and disease. *Annals of Human Genetics*, 19, 251–253.

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Lecture

Behavioural Psychotherapy in General Psychiatry
Helping Patients to Help Themselves*

I. MARKS

It is widely believed by psychiatrists that patients who require behavioural psychotherapy need to be referred for such treatment to a specialist in the subject. Two assumptions underlie this idea. One is that behavioural treatment requires a detailed knowledge of learning theory which is possessed by few psychiatrists. The other assumption is that behavioural methods are too time-consuming for a busy psychiatrist in a routine outpatient or primary care clinic.

Neither of these assumptions is borne out by the evidence. Hundreds of general psychiatrists and nurses in the UK have in the last few years learned to give behavioural psychotherapy successfully to a wide variety of problems without formal training in learning theory and after very brief instruction in the treatment methods themselves. Moreover, these effective behavioural applications often required the clinician to devote no more time to the patient than is customary with pharmacological and other forms of psychiatric treatment in routine use. The evidence for these statements comes from extensive experience of tutors running courses in behavioural psychotherapy for senior psychiatrists, and from the teachers of nurses. Within days of instruction many clinicians become able to find ways of helping patients in routine psychiatric and general practice clinics by behavioural means.

This encouraging development arises partly from the recent move away from the idea of behavioural treatment as something done to patients towards a realisation that behavioural methods form a system of self-help that the patient learns from a clinician who largely acts as coach and monitor. In most cases there is no need to practise behavioural manoeuvres with the patient in the natural situation, only to guide them on what to do there.

The more usual pattern of a behavioural session in the clinic takes a different form, the practice part of the treatment now being mainly carried out by the patient without a clinician being present (steps 3 & 4 in Table I). Once the detailed nature of the patient's problem has been carefully unravelled in the course of an interview, the therapist explains to the patient (and a relative too if one is available as a co-therapist) how he can overcome the problem; he asks him to use a suitable manual, if one is available, to assist him in the self-help programme and to record every day in a structured but simple diary the tasks carried out and their outcome. The patient then leaves the clinic to practise the suggested procedures as homework between sessions while keeping a daily record of this homework and its results. He brings this homework diary back to the therapist in the next session for discussion and suggestions how to proceed further. Sometimes it is also helpful for patients to phone the therapist once or twice between sessions to make an interim report on progress and setbacks and to get ideas on how to deal with these. The patient is taught to anticipate setbacks and how to manage them.

In fact this model has been the norm in treating sexual problems for many years. The clinician never stands in the patient's bedroom telling him how to improve his lovemaking. Rather, the therapist, after taking a careful history from the patient and partner, works out suitable exercises for the couple to follow in the bedroom; these bedroom exercises constitute homework sessions outside the clinic. We can see this in an actual case that improved after a total of only 3½ hours of nurse-therapist time with the couple, including initial assessment.

A man of 36 complained of premature ejaculation for 3 years. Coitus took place once a week, with ejaculation within 45 seconds of penetration, though ejaculation could be delayed up to 3 minutes if his wife was in the female superior position. He refused to let her touch his genitals lest that precipitate ejaculation. The marriage was otherwise satisfying.

TABLE I
Process of behavioural treatment

1. Assess in clinic
2. Suggest behavioural methods to produce improvement
3. Practise these methods in the natural situation[1]
4. Record outcome in daily diary[1]
5. Monitor results
6. Repeat points 3–5 until adequate improvement obtained
7. Follow-up

[1] Used to be carried out with a clinician; now usually done by patient alone

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*Maudsley Bequest Lecture given at the Winter Quarterly Meeting of the Royal College of Psychiatrists, 22 January 1986

TABLE II

Diary of self-exposure homework: example from an agoraphobic. From Marks (1978)

	Session						
Day	Date	Began	Ended	The exposure task I performed was:	(0 = complete calm, 8 = absolute panic) My anxiety during the task was:	Comments, including coping tactics I used:	Co-therapist, if any: *J. Smith* (Co-therapist's signature that task was completed)
Wednesday		2:30 p.m.	4:30 p.m.	Walked to local supermarket and surrounding shops, bought food and presents for family, had coffee in restaurant	7	Felt worse when shops were crowded, practised deep-breathing exercises	J. Smith (husband)
Thursday		10 a.m.	11:30 a.m.	Walked to local park, sat there for ½ hour till I felt better, then caught a bus to town and back home	6	Felt giddy and faint, practised imagining myself dropping dead	J. Smith
Friday		2 p.m.	4 p.m.	Rode a bus to town and back 3 times till I felt better about it	5	Worse when bus was crowded – did deep-breathing exercises	J. Smith

Plan for next week: Repeat exposure exercises in bus, park and shops every day until my anxiety is no higher than 3. Thereafter start visits to my hairdresser, and short surface train journeys.

The couple was instructed in mutual caressing that gradually included the genitals for lengthening periods, and how to use the squeeze technique to delay ejaculation. Coitus was first banned and then gradually reintroduced as the couple became skilled with the squeeze method. Within weeks, coital time was prolonged to 30 minutes in varying positions of intercourse, and when last seen at 1 month follow-up, progress was maintained.

Even severe agoraphobia can respond with very little therapist investment, merely appropriate advice.

A woman of 40 was enduringly helped by only 1½ hours of my time. She had been almost housebound for 5 years, from classic agoraphobia. First, with her and her husband we mapped her *avoidance profile* – which phobic situations she needed to enter in her everyday life yet avoided regularly because they evoked panic, so leading to handicap. This discussion yielded a profile of the public places she avoided, such as walking outside her home and going alone on a bus or train.

From the avoidance profile we devised a systematic *exposure-homework* programme that she could embark on. Starting with those avoided situations which she especially wanted to be able to re-enter in everyday life (e.g. walking outside her home alone, going alone on a bus), she was asked to daily go into at least one such situation for an hour or more until the ensuing panic had subsided somewhat. This exposure had to be carried out with*out* taking sedative drugs or alcohol. Every day she had to repeat the self-exposure exercise until she felt comfortable in the formerly panic-evoking situation. As continuing exposure produced habituation to particular places, she was to slowly extend self-exposure to further avoided places. I explained how she should keep a daily diary of her exposure-homework exercises, and asked her to post them to me (Table II). This she did, weekly at first and then at lengthening intervals. Her diligent self-exposure programme led her within weeks to become mobile for the first time in years. She kept up her progress without seeing me again but relapsed 4 years later, feeling low from some family problems. Seeing her once more for another hour prompted her to revive her original exposure homework programme. On doing this she recovered her gains. At 9 years follow-up she had residual fears but remained much improved and mobile.

That is a gratifying result for 2½ hours of time from a clinician. Even less therapist time – merely 1 hour – was needed to reduce a variety of troublesome compulsive rituals in an 11-year old girl (Zikis, 1983).

Her rituals had been troublesome for 6 years. The therapist began the exposure and response prevention with Jane and her mother together, after which mother continued this programme for 2 hours daily with Jane for a week, by when the rituals disappeared, then for an hour daily for 3 weeks, after which Jane did the homework on her own. Tics that had been associated with the rituals also disappeared, and Jane remained well to 1-year follow-up.

The single 45-minute session with the therapist had been carried out in the mother's presence. Jane was instructed to throw her glove down on the floor and to pick it up with her hand once only, and was told she was not allowed to throw it down again and pick it up with the other hand in the usual manner of one of her rituals. She was also asked to touch the handle of a door with her hand once only without repetition. These two tasks initially evoked anxiety and tics which gradually faded away as Jane performed the tasks several times without the ritual. Homework prescriptions for Jane to carry out included: (a) while pouring milk into her tea, to deliberately spill some drops of milk on the floor or table once only, (b) after telephoning from home, to put down the receiver once only, (c) on going to bed to close her eyes and avoid opening them again while saying 'I am not going cross-eyed – I do not see double'.

The point of these vignettes is not to show what wonderful things clinicians can do. Rather, they illustrate what might be achieved by means of minimal interventions of the right kind for the right types of problem. We are not talking about a general placebo effect in which saying almost anything soothing might help. Advice to relax, for example, will not improve phobics or obsessive-compulsives; nor will anti-exposure instructions. When such patients were given advice to take a holiday from anxiety and systematically avoid whatever was upsetting them they did not improve, though these same patients did improve on being asked to embark on an exposure programme (Greist *et al*, 1980; Marks *et al*, 1987). Nor is persuasion of a premature ejaculator to talk about childhood problems likely to prolong his ejaculatory time, whereas giving him some basic sexual skills training has a fair chance of doing so. Nor can a child with enuresis or encopresis be expected to become drier and cleaner if parents systematically reward him for being wet and dirty rather than for being dry and clean; the shaping has to be appropriate.

Though we are talking of a touch of the rudder, the touch must be in the right direction. Even simple procedures have to be executed properly; psychological interventions can be as specific in their effects as are drugs.

Cases responding to brief behavioural guidance of the above kind are not rare. General psychiatrists who are alert to the possibilities of behavioural treatment might find patients every week who could benefit from suitable detailed advice which is tailored to the patient's particular problem.

Some people can in fact treat themselves without ever even needing advice from a clinician, devising and completing a methodical self-help programme without instruction, once they know what principle to apply systematically. Take for example an autobiographical note in 1770, from Wolfgang von Goethe, who as a 20-year-old law student at Strasbourg University decided to apply the exposure principle to remedy his phobias of heights, diseased bodies, loud noises, and dark, lonely places.

> I ascended quite alone the highest pinnacle of the cathedral spire, and sat in the so-called neck under the knob or crown, as it is called, for a quarter of an hour, before I ventured to step out again into the open air, where, on standing on a platform scarcely an ell square, without anything particular to hold on to, one sees before one the boundless land, while the nearest objects and ornaments conceal the church and everything on which and above which one stands. It is exactly as if one saw oneself carried up into the air in a balloon. Such anxiety and pain I repeated so often until the impression became quite different to me, and I have therefore derived great advantage from these practices in mountain travels and on geological studies, and on great buildings, where I have vied with the carpenters in running over the freelying beams and the cornices of the building, and even in Rome, where one must run similar risks in order to obtain a nearer view of important works of art.

Goethe went on to treat his other fears similarly.

The amount of behavioural help that sufferers need from a clinician varies enormously. Many sufferers merely lack the knowledge of what principle to apply, and on learning what to do from the clinician or on reading a suitable self-help manual, can then go off and apply it on their own without further help. Detailed manuals and diaries to record homework help sufferers to execute their programme properly. In planning a self-exposure programme phobics and compulsive ritualisers have to remember that long exposure periods reduce fear more than do shorter ones. Some cases need prompting to get started and can then take off on their own; others require more help from the clinician in monitoring their progress at intervals. Clinicians need to teach patients to expect setbacks and to rehearse dealing with them alone, but be available for occasional booster sessions that some people require. All these cases can be treated fairly easily by a general psychiatrist.

When patients need additional help (e.g. certain phobics and obsessive-compulsives require some therapist-aided exposure), then at that point the average general psychiatrist might wish to call in specialist help. A few such cases want therapists to continue helping them indefinitely with exposure, despite having repeatedly improved during sessions; the long-term outcome in such cases is dubious, as the therapist must eventually fade out of the scene.

The best controlled evidence for the value of self-treatment in the behavioural area is in phobics and obsessive-compulsives. At least 20 controlled studies have found self-exposure to be helpful for such problems, most of these studies being in agora-, social, and specific phobics, while only one controlled trial found no benefit from self-exposure (reviewed by Marks, 1987). In the largest controlled study (Ghosh *et al*, 1987; Ghosh & Marks, 1986) marked gains from self-exposure occurred despite there being no interaction with a clinician beyond initial screening. Over 10 weeks 71 phobic patients were randomised to get self-exposure instructions from a self-help book (Marks, 1978), a psychiatrist, or a computer. Apart from a 1½-hour initial assessment, mean clinician time per patient in the three groups was respectively only 0, 1.6, and 1.5 hours. All three groups improved markedly and equally up to 6 months follow-up, in both the agoraphobic ($n = 40$, Ghosh & Marks, 1986) and social/specific phobic subgroups. Neither the clinician nor the computer conferred any advantage over the manual.

An obvious question is whether all three treatment conditions in the above study were doing anything more than placebo. In other controlled studies patients who expected to improve with another procedure – anti-exposure – failed to get better (Greist *et al*, 1980) and while anti-exposure almost abolished gains from anti-depressants – imipramine in the case of agoraphobics (Telch *et al*, 1985) and clomipramine in the case of ritualisers (Marks *et al*, 1987). Improvement from self-exposure thus has little to do with the mere expectation of improvement, but a great deal to do with the component of exposure that causes anxiety to subside.

The study of Marks *et al* (1987) also found that adding therapist-aided exposure after 8 weeks of a self-exposure programme conferred surprisingly few advantages in ritualisers, the long-term gains in both groups being similar. On the face of it, compulsive ritualisers seem to need to do more complicated things by way of treatment than, say, agoraphobics. However, our results are in line with Dutch findings that self-exposure helped obsessive-compulsives (Boersma *et al*, 1976 and Emmelkamp & Kraanen in controlled studies, and Hoogduin, 1985 in uncontrolled work).

Self-exposure seems to have better results when patients use an appropriate manual and diary, recording homework tasks for regular review. Still to be tested is the value of reading a manual without any prompting from a clinician. Two patients in the book-instructed condition of Ghosh *et al* (1987) had in fact previously bought the book on their own initiative but failed to follow its directions until they were asked to do so by the psychiatrist in the trial; initial contact with a clinician is thus an important motivator for some people. This raises critical theoretical issues. Why should certain people who know exactly what to do need to have a guru before they can do it? Some can learn a language, tennis or carpentry by self-practice with the aid of books and tapes without any coaching, while others, even if highly intelligent, find such tasks a losing battle despite intensive skilled teaching. Many ill-understood variables affect this process.

The need for therapist-aided exposure for phobics and obsessive-compulsives has diminished as self-exposure manuals have been refined. This is welcome as therapist-aided exposure is time-consuming and thus expensive. Patients on the author's waiting list are asked to first try treating themselves, using *Living With Fear* as a manual, and not infrequently improve enough therewith to obviate the need for a therapist. For most of the remaining cases the clinician's role is to assess the problem and to teach people to help themselves better.

Clinicians on the lookout for behavioural applications might find quite straightforward ways of helping certain problems.

In shy students, simple practice at dating helped them become more sociable (Arkowitz *et al*, 1975). They had a programme of 6-weekly practice-dates, each with a different matched opposite-sex partner who had similar anxiety. Subjects were told that the aim was not to find an ideal partner but to increase dating comfort and frequency. Each week the student was sent the name and phone number of the partner to be dated that week. Practice-dating increased the frequency of dates with partners outside the study until 3 months follow-up, and this continued in the 60% who could be followed up for 15 months; practice-dating also reduced social avoidance and distress and enhanced social skill. Improvement was greater after practice-dating than after a waiting list control (Christensen *et al*, 1975; Kramer, 1975) or reading and discussing a manual (Royce, 1975).

Of course great caution is needed before extrapolating from such students to cases with severe social deficits, but the principle of self-treatment is worth trying to adapt for them. In patients with social skills deficits, Stravynski (personal communication) found that to the end of treatment a similar social-homework approach helped as much as did social skills training; follow-up is now in progress.

General psychiatrists can give effective behavioural guidance not only to many phobics and obsessive-compulsives but also to some patients with sexual problems, e.g. premature ejaculation. Simple advice has even helped paedophiliacs with erectile dysfunction.

A clergyman came to me because he had not consummated his marriage of 3 years' duration: he had regularly masturbated little boys and lacked interest in women. It transpired that he had been impotent twice on his honeymoon and that he and his wife had not tried to have intercourse in the 3 years thereafter. I gave him the advice given by John Hunter to an impotent man two centuries ago, i.e. to lie naked with his wife and engage in mutual caressing but on no account to have intercourse with her. Soon he was achieving erections during caressing and gradually became able to sustain them. At that point he was asked to start partial and later complete coitus. This was successful, and his paedophiliac urges disappeared.

Obviously this is hardly a cure for all paedophiliacs; the point is that behavioural measures that take little therapist time can be of great help, provided the clinician is aware of such possibilities. Most behavioural psychotherapy can probably be given by non-specialists, with specialists only being needed for complex cases whose management might be especially difficult and time-consuming.

With little expenditure of time, general psychiatrists could help many patients by giving appropriate brief behavioural guidance and intermittent monitoring of the patients' homework.

References

Arkowitz, H., Christensen, A. & Royce, S. (1975) Treatment of social inhibition by real-life practice. Paper to AABT, 1975.

Boersma, K., Den Hengst, S., Dekker, J., Emmelkamp, P. M. G. (1976) Exposure v response response prevention in obsessive-compulsive patients. *Behaviour Research & Therapy*, **14**, 19–24.

Christensen, A., Arkowitz, H. & Anderson, J. (1975) Practice dating as treatment of college dating inhibitions. *Behaviour Research & Therapy*, **13**, 321.

Emmelkamp P. M. G. & Kraanen, J. (1978) Therapist-controlled v self controlled exposure in vivo in obsessive-compulsive patients. *Behaviour Research & Therapy*, **15**, 491–495.

Ghosh, A. & Marks, I. M. (1986) Self-directed exposure for agoraphobia: a controlled trial. *Behavior Therapy*, (in press).

——, —— & Carr, A. C. (1987) Self exposure for phobias. *British Journal of Psychiatry*, (in press).

Greist, J., Marks, I. M., Berlin, F. & Noshirvani, H. (1980) Avoidance versus confrontation of fear. *Behavior Therapy*, **11**, 1–14.

Hoogduin, C. A. L. (1985) Mislukking en success bij de ambulante behandeling van dwangneurose. PhD Dissertation, University of Leiden.

Kramer S. R. (1975) Behavior rehearsal and practice dating for heterosexual social inhibition. PhD dissertation, University of Texas at Austin.

Marks, I. M. (1978) *Living with Fear*. New York: McGraw-Hill.

—— (1987) *Fears, Phobias and Rituals*. New York: Oxford University Press.

——, Basoglu, M., Lelliott, M. *et al*, (1987) Clomipramine and exposure in OCD. A replication (in press).

Royce, W. S. (1975) Practice and feedback as treatment for social isolation. PhD dissertation, University of Oregon.

Telch, M., Agras, W. S., Taylor, C. B. *et al*, (1985) Imipramine and behavioural treatment for agoraphobia. *Behaviour Research & Therapy*, **23**, 325–335.

Zikis, P. (1983) *In vivo* exposure and response prevention for rituals and tics in an 11-year old girl. *Behavioural Psychotherapy*, **11**, 75–81.

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Review Article

Prevalence Studies in Schizophrenia

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Over 70 prevalence studies of schizophrenia published since 1948 are reviewed. Despite problems of method, the studies suggest that there may be significant differences in prevalence among different populations and that prevalence may change over time. Further research is needed on how much differences in diagnosis or the course of schizophrenia contribute to the variable prevalence rates. The paper suggests the following as areas for additional enquiry: a possible north-south gradient, special populations (e.g. American Hutterites, northern Swedes, western Irish, castes in India) and ongoing case registers in areas of suspected high and low prevalence.

> Warm climates are not those which produce the greatest amount of insanity, but rather temperate climates, subject to great atmospheric vicissitudes, and especially those whose temperature is alternately cold and humid, damp and warm.
> Jean E. Esquirol (1845)

Geographical comparisons of the prevalence of schizophrenia have been of interest to mental health specialists for well over a century. When Emil Kraepelin voyaged to Singapore and Java in 1904 he specifically looked for cases of dementia praecox, and concluded that "in all civilized nations" the disease "comes under observation in approximately the same forms and everywhere accounts for the greatest number of the permanent inhabitants of institutions" (Kraepelin, 1919).

In recent years there has been a resurgence of interest in prevalence as epidemiological studies have multiplied. No longer can the field be characterised, as it was in 1963, as composed of "a small amount of inadequate research combined with a large amount of adequate criticism" (Mishler & Scotch, 1963). Since 1948 over 70 prevalence studies of schizophrenia have been published in which many used sophisticated techniques equivalent to epidemiological studies in other areas of medicine.

The interpretation of these studies, however, has varied widely. Jablensky & Sartorius (1975) reviewed 23 prevalence studies with an almost ten-fold difference from the lowest to the highest rate and concluded that "with few exceptions . . . the data on the prevalence of schizophrenia appear to be very similar in different cultures." More recently, in summarising the World Health Organisation (WHO) study of the incidence of schizophrenia in which a two-fold difference was found using a narrow definition and an almost three-fold difference using a broad definition, Sartorius *et al* (1986) concluded that "the results provide strong support to the notion that schizophrenic illness occurs with comparable frequency in different populations." Similarly Leff (1981) reviewed 11 prevalence studies of schizophrenia with an almost five-fold difference and concluded that "we can state on the basis of the studies reviewed that there is no evidence suggesting that schizophrenia is much more frequent in one part of the world than in another." On the other hand, Torrey (1980) reviewed a large number of studies and concluded that "the difference is greater than ten-fold and cannot be ignored," while Eaton (1985) also noted "roughly a ten-fold variation in the rates" and asserted that the differences were highly significant.

One reason for the differing interpretations of the data is problems of method inherent in prevalence studies of schizophrenia. Differences in diagnostic standards have varied widely around the world and certainly account for some of the different rates. Differing life expectancies will also affect rates, as will the age distribution of the population (e.g. large numbers of young children will lower the rate). For this reason some researchers have calculated the schizophrenia prevalence rate for a given age cohort (e.g. 18–65) or applied an age correction factor (e.g. the Weinberg abridged method). Another factor to be considered is the course of the illness; there is evidence from several studies that schizophrenia has a more benign course in developing countries which would produce lower prevalence rates.

Incidence studies of schizophrenia have the advantage of counting cases at their inception so the rates are not as affected by course and outcome; their big disadvantage, however, is that a significant subgroup of persons develop schizophrenia insidiously over months or years and it is virtually impossible to date the onset. Some studies of incidence have defined it as when the person was first referred for psychiatric care but this measure is affected by cultural factors and the availability of care. For these reasons incidence studies have been less used than prevalence studies and will not be included in this review. As Cooper (1978) summarised: "Reported incidence rates for schizophrenia tend to vary more widely than reported prevalence rates, and on the whole are probably less reliable."

It should be noted that all major diseases in which both genetic and non-genetic factors are thought to be important have significant prevalence differences. Heart disease varies approximately six-fold (Thom *et al*, 1985), rheumatoid arthritis ten-fold (Utsinger *et al*, 1985), type I diabetes 30-fold (Marble *et al*, 1975) and multiple sclerosis at least 50-fold (Kurtzke, 1975). In all these diseases there have been problems of method of narrow versus broad diagnostic definitions and differing mortality rates, similar to problems faced with schizophrenia. In addition, all current aetiological hypotheses of schizophrenia (genetic, diathesis-stress, biochemical, nutritional, infectious, psychosocial) would logically predict significant differences in the prevalence of the disease. If schizophrenia has no such differences it would be a unique disease and that fact alone would be one of its most significant aspects. In looking at the prevalence studies of schizophrenia it would be surprising if differences were not found.

Range of prevalence

Table I summarises schizophrenia prevalence studies with approximately 1000 or more subjects which have been published (or summarised) in English since 1948. According to Dr Assen Jablensky of WHO several additional studies have been published in the Russian literature (pers. comm.) The studies are arranged from the lowest prevalence to the highest. Prevalence rates are given per total population but the rates are not necessarily comparable because of the problems with method previously noted. In some studies prevalence rates were calculated only for age-corrected cohorts but, since different cohorts were used in different studies, these rates are also not comparable. Countries with large numbers of young children have a greater difference between the prevalence rate per total population and age-corrected rate.

The prevalence period used in the studies varies from cases symptomatic at the time of the survey (point prevalence) to cases which have occurred at any time during the person's life (lifetime prevalence). Cases of schizophrenia in which the person has recovered will be excluded from the shorter prevalence periods, while the longer periods must rely on recall of past clinical states. Case finding is done by using key informants to identify suspected cases who are then interviewed, doing house-to-house surveys (census method), or utilising ongoing case registers. For schizophrenia there is evidence that using key informants in rural areas produces results very similar to those obtained by house-to-house surveys (Nielsen & Nielsen, 1977).

The range of prevalence from the lowest reported rate (0.3 per 1000 among the ultraconservative agrarian Amish sect in the USA) to the highest reported rate (17.0 per 1000 in a restudy of an isolated population in northern Sweden) is over 50-fold. The Amish findings must be qualified, however; only four cases of schizophrenia were diagnosed among 12 500 Amish but 22 cases diagnosed with bipolar disorder had initially been called schizophrenia on hospital records. One can argue, therefore, that some of the bipolar patients would have been diagnosed with schizphrenia by other investigators and the schizophrenia rate is artificially low.

The three studies with the next lowest prevalence rates – in Ghana, among Taiwanese Aborigines, and among the Hutterites in the USA – cannot be as easily dismissed. Rin & Lin's (1962) study of the Aborigines, for example, was cited by Mishler & Scotch (1963) as having the best methods for case finding of any study done to that time. Leff (1981) criticised the study because cases of 'malarial psychosis' were not counted as schizophrenia, but in fact acute psychosis secondary to malaria is relatively common in tropical countries and should not be confused with schizophrenia. Among the 10 cases of schizophrenia diagnosed by Rin & Lin two had lasted less than 3 months and another four had recovered in less than 2 years. Despite a life expectancy of only 40–45 years among this population (which would reduce the number of older schizophrenics), the disease appears to have been genuinely uncommon in this group at the time of the study.

High prevalence was reported in Book's two studies of northern Sweden done 25 years apart (Book, 1953; Book *et al*, 1978), Vaisanen's study of communities in both northern and southern Finland (1975), and the study by Torrey *et al* (1984) of an area in western Ireland. It should be noted that both Book and Torrey *et al* selected areas for study which were reputed to have a high prevalence rate for psychosis, but this was not true for the Finnish study. The comparatively high schizophrenia prevalence rates found by Vaisanen *et al* (1975) were confirmed by a restudy of the same communities 5 years later in

TABLE I
Prevalence studies of schizophrenia

						Schizophrenia Prevalence/1000	
Country	*First author*	*Year published*	*Type of study*[1]	*Total population*	*Prevalence period*	*Total population*	*Age corrected*[2]
USA: Amish	Egeland	1983	R, K	12 500	5 years	0.3	0.5
Ghana	Sikanarty	1984	U, K	45 195	point	0.6	1.1 (15+)
Italy	Zimmerman-Tansella	1985	U, CR	74 852	1 year	?	1.3 (14+)
Taiwan: Aborigines	Rin	1962	R, C	11 442	lifetime	0.9	1.5 (15+)
USA: Hutterites	Eaton	1955	R, K	8 542	lifetime	1.1	2.1 (15+)
Argentina	Bonhour (in Sauna)	1966 1969	U, R, C	4 442	?	1.1	?
Scotland	Wing	1967	U, CR	185 000	1 year	?	2.5 (15+)
Scotland	Robertson (in Eaton)	1981 1985	U, CR	477 700	point	1.4	?
Iran	Bash	1974	U, C	928	point	?	2.2 (7+)
Canada	Roy	1970	R, K	28 096	lifetime	1.6	2.4 (15+)
Canada	Murphy	1964	R, K	18 470	point	?	2.4 (15+)
Ireland (Dublin)	Walsh (pers. comm.)	1986	U, CR	234 300	point	1.7	2.5 (15+)
India	Thacore	1975	U, C	2 696	1 year	1.9	?
Yugoslavia	Kulcar	1971	R, C	36 326	3 months	?	2.9 (20–64)
Botswana	Ben-Tovim	1986	R, K	2 526	1 year	2.0	4.4 (15+)
Italy	DeSalvia (pers. comm.)	1986	U, CR	92 104	1 year	2.0	2.6 (15+)
England (London)	Wing	1976	U, CR	154 900	point	2.0	?
Taiwan	Lin	1969	U, R, C	29 184	lifetime	2.1	?
Japan	Kato	1969	U, C	5 207	point	2.1	5.0 (W[2])
England	Gibbons (in Eaton)	1980 1985	?	?	1 year	2.1	?
Taiwan	Lin	1953	U, R, C	19 931	lifetime	2.2	5.9 (W)
Japan	Kato	1969	U, C	2 712	point	2.2	4.9 (W)
India	Nandi	1980	R, C	4 053	point	2.2	?
India	Dube	1970	R, C	28 468	lifetime	2.3	?
Japan	Kato (1954 Study)	1969	U, R, C	23 993	1 year	2.3	?
Japan	Kato (1963 Study)	1969	U, R, C	44 092	1 year	2.3	?
India	Sethi	1974	U, C	4 481	point	2.5	3.4
Germany	Hafner	1980	U, CR	308 975	point	2.5	?
Germany	Dilling	1984	R, C	1 536	point	?	3.9 (16+)
USSR	Krasik (in Cooper)	1965 1978	R, ?	?	?	2.6	?
Denmark	Nielson	1976	R, C	6 013	point	2.7	3.5 (15+)
Bulgaria	Temkov (in Jablensky)	1975 1986	U, CR	140 758	point	2.8	?
England (Salford)	Wing	1976	U, CR	130 900	point	2.8	?
England (Salford)	Freeman	1986	U, CR	91 700 (15+)	1 year	?	6.8 (15+)
Yugoslavia	Crocetti	1971	U, C	4 282	3 months	?	4.2 (20–64)
India	Nandi	1975	R, C	1 060	point	2.8	5.0
USSR	Krasik (in Cooper)	1965 1978	U, ?	?	?	3.1	?
Sweden	Stromgren	1948	R, K	45 930	lifetime	3.3	4.7 (W)
England	Wing	1967	U, CR	175 304	1 year	3.4	4.4 (15+)
USA: New Haven	Hollingshead	1958	U, K	238 831	6 months	3.6	?

TABLE I *(continued)*

Country	*First author*	*Year published*	*Type of study*[1]	*Total population*	*Prevalence period*	*Schizophrenia Prevalence/1000*	
						Total population	*Age corrected*[2]
USSR	Zharikov	1968	U, CR	400 000	point	3.6	5.1 (16+)
Sri Lanka	Wijesinghe	1978	U, C	7 653	6 months	3.7	5.5 (15+)
Sri Lanka	Jayasundera	1969	R, C	4 718	?	3.8[3]	?
Korea	Yoo	1961	R, C	?	lifetime	3.8	9.6 (W)
USSR	Rotstein (in Jablensky)	1977 1986	U, R C	?	?	3.8	?
Sweden: Stockholm	Halldin	1984	U, C	2 009	1 year	?	6.0 (18–65)
USA: St Louis	Myers	1984	U, C	3 004	6 months	?	6.0 (18+)
USA: rural	Blazer	1985	R, C	1 936	6 months	?	6.0 (18+)
Japan: Hachijo Island	Kato	1969 (1940 Survey)	K	8 318	point	3.9	9.1 (W)
USA: Rochester	Babigian	1985 (1970 Survey)	U, CR	712 000	1 year	4.1	?
Scotland	Mayer-Gross	1948	R, K	56 231	lifetime	4.2	?
India	Elnagar	1971	R, C	1 363	lifetime	4.3	?
Australia: Aborigines	Jones	1973	R, K	1 800	lifetime	4.4	?
Australia: Aborigines	Eastwell	1975	R, K	3 400	lifetime	4.4	8.7 (17+)
Sweden	Hagnell	1966	R, C	3 310	lifetime	4.5	5.1 (11+)
Sweden	Sjogren	1948	K, R	8 736	45 years	4.6	8.3 (W)
USA: Baltimore	Wing	1967	U, CR	950 000	1 year	?	7.0
Japan: Hachijo Island	Kato	1969 (1961 Survey)	K	12 027	point	4.7	?
India	Carstairs	1976	R, C	1 233	lifetime	?	7.2 (15+)
Ireland: 3 counties 1982 data	Walsh (pers. comm.)	1986	R, CR	165 223	point	4.9	7.1 (15+)
Yugoslavia	Crocetti	1971	U, C	4 282	3 months	?	7.3 (20–64)
Yugoslavia	Kulcar	1971	R, C	15 756	3 months	?	7.4 (20–64)
USA: Rochester	Babigian	1985 (1975 Survey)	U, CR	702 000	1 year	5.1	?
Norway	Bremer	1951	R, C	1 325	lifetime	5.3	6.5 (11+)
India	Nandi	1979	R, C	3 718	point	5.6	?
Ireland: 3 counties 1973 data	Walsh	1980	R, CR	149 422	point	5.6	8.1 (15+)
Canada: Indians	Roy	1970	R, K	4 723	lifetime	5.7	11.0 (15+)
Norway	Fugelli	1975	R, C	1 726	2 years	5.8	6.9 (10+)
Ireland: Co. Roscommon 1982 data	Walsh (pers. comm.)	1986	R, CR	45 324	point	5.9	8.1 (15+)
Sweden	Larsson	1954	R, K	13 085	45 years	6.3	?
Sweden	Essen-Moller	1956	R, C	2 550	lifetime	6.7	8.1 (15–45)
USA: Baltimore	Myers	1984	U, C	3 481	6 months	?	10.0 (18+)
USA: New Haven	Myers	1984	U, C	3 058	6 months	?	11.0 (18+)
USA: Durham	Blazer	1985	U, C	1 862	6 months	?	11.0 (18+)
Ireland: Co. Roscommon 1973 data	Walsh	1980	R, CR	45 426	point	7.1	9.8 (15+)
Finland	Vaisanen	1975	U, R, C	991	point	?	15.1 (15–64)
Sweden (suspected high prevalence area)	Book	1953	R, K	8 981	point	9.4	23.9 (15–45)

continued

TABLE I *(continued)*

Country	*First author*	*Year published*	*Type of study*[1]	*Total population*	*Prevalence period*	*Total population*	*Schizophrenia Prevalence/1000 Age corrected*[2]
Ireland (suspected high prevalence area)	Torrey	1984	R, K	2 848	6 months	12.6	17.4 (15+)
Sweden (restudy of high prevalence area)	Book	1978	R, K	5 748	point	17.0	?

R: Rural; U: Urban; K: Key; CR: Case register; C: Census

1. Type of census is characterised as either the key informant type (key) in which official and hospital records are examined, key persons are asked to name all mentally ill persons, and all suspected cases are then interviewed; or the census type (census) in which a house-to-house survey is conducted in addition to the above. For schizophrenia there is evidence that the key method over a long prevalence period identifies all cases of schizophrenia in the community and thus is equal to rates obtained by the census method (Nielsen & Nielsen, 1977). However, the key method for a point prevalence would presumably not detect all cases extant and thus would not be directly comparable to rates obtained by the census method.
2. Age-corrected schizophrenia rates are calculated by number of cases per population in the susceptible age group. Since these differ the rates in this column are not strictly comparable one to another. Many of them are calculated using the Weinberg abridged method (indicated by W) which is a measure of the expectancy of developing schizophrenia if a person lives through the 16–40 age-susceptible group. It is computed by the number of schizophrenic patients divided by (a) the total number of people who have reached age 40 plus (b) one-half the population in the 16–40 group; this product is multiplied by 100. W figures are comparable with one another in the chart (see Lin, 1953).
3. The Sri Lanka rate of 3.8 is based on the findings of the first two studies which the researchers claimed were considerably more reliable than the last two studies. If all four studies are used the rate becomes 3.2 per 1000.

which the number of persons found with psychoses was 50% higher than the original study (Lehtinen & Vaisanen, 1981); unfortunately the authors do not provide a diagnostic breakdown of the psychoses so the restudy is not included in Table I.

There are other suggestions that schizophrenia prevalence rates in northern Scandinavia are high. Bremer (1951) reported a rate of 5.3 per 1000 for a northern Norwegian village; the village was restudied 30 years later and the number of persons with 'functional psychosis' (no diagnostic breakdown given) had increased (Bjarnar *et al*, 1975). Fugelli (1975) surveyed two northern Norwegian islands and found 10 cases of schizophrenia among 1726 residents (5.8 per 1000); in addition he diagnosed another 15 cases of 'organic and senile psychosis' and 17 cases of 'other psychosis' yielding a prevalence of all psychoses of 24.3 per 1000. Anderson (1975) studied another area of northern Norway and found a prevalence rate for all psychoses (no diagnostic breakdown given) of 24 per 1000 for the Norwegian inhabitants and 54 per 1000 for the Lapps.

The area in western Ireland in which Torrey *et al* (1984) found a schizophrenia prevalence rate of 12.6 per 1000 (included 21 with DSM-III schizophrenia, 11 with schizoaffective disorder, and four with atypical psychosis) has not yet been restudied. The authors reported widespread belief among mental health professionals in the western counties that some areas have much more schizophrenia than others. The prevalence rate of the area studied in County Roscommon is more than twice the 5.9 per 1000 rate found by case register for the county as a whole and more than seven times the rate found for the St Loman's case register in Dublin for 1982 (Walsh, pers. comm., 1986). Emigration from western Ireland has been high for over a century but the authors claim this would account for only a small portion of the high prevalence observed. Schizophrenia has also been noted to be common among Irish immigrants to the USA so the high Irish rate cannot be explained simply by saying that the healthier individuals left and the pre-schizophrenic individuals remained at home.

High and low prevalence pockets

A further perusal of schizophrenia prevalence studies, worldwide, suggests an uneven distribution between some population groups which are geographically contiguous. The Amish and Hutterite prevalence rates in the USA, for example, are less than 25% of the rates reported for rural (North Carolina) or urban (New Haven, Rochester, Baltimore, Durham, St Louis) areas in recent studies. Differences in the prevalence of schizophrenia in the USA are also suggested by studies of hospitalisation rates although it must be recognised that these are determined by availability of beds and other factors in addition to true prevalence. A 1903 study of hospital admission rates for schizophrenia in the USA noted that "the greatest proportion of insanity is in the northeast . . . If from this center of greatest prevalence of insanity we draw a line in any direction — west, south, or southwest — we see that no matter which way we go we find a steady decrease until we strike the Pacific slope" (White, 1903). First admission figures for schizophrenia in various states for subsequent years have confirmed White's conclusion (Torrey, 1980).

As previously noted the Taiwanese Aborigines have a schizophrenia rate less than half that of the Taiwanese Chinese, the northern Swedes studied by Book have a rate twice that of southern Swedes, and the western Irish studied by Torrey *et al* have a rate twice that of their neighbours a few kilometres away. The most intensive study of intrapopulation differences, however, was the research done in Yugoslavia by Kulcar *et al* (1971). It had long been rumoured that the Istrian peninsula had an unusually large number of people with psychosis, and this was subjected to sophisticated study over 10 years. It was found that both schizophrenia and bipolar disorder were higher in the study areas compared to other parts of Croatia; the schizophrenia prevalence rate (for people ages 20–64) was 7.4 per 1000 in villages and 7.3 per 1000 in an urban area on the Istrian peninsula compared with 2.9 per 1000 (villages) and 4.2 per 1000 (urban) elsewhere in Croatia.

The existence of high and low prevalence pockets has been observed elsewhere. Researchers in Japan did two studies of Hachijo Island and reported the prevalence of schizophrenia to be 3.9 and 4.7 per 1000; surveys done elsewhere in Japan at the time consistently found a prevalence rate of 2.1–2.3 per 1000 (Kato, 1969). Murphy & Vega (1982) found that "first admissions for schizophrenia in Northern Ireland are significantly higher for the Roman Catholics than for the rest of the population." Murphy (1968) also reported that Catholics in Canada had significantly higher hospitalisation rates for schizophrenia compared with non-Catholics. In a study in Singapore, Murphy (1968) reported that Indian (mostly Tamil-speaking) students at the University of Singapore had twice as high a schizophrenic prevalence rate as the Malay students; Chinese students had an intermediate rate. Differential prevalence rates for schizophrenia have also been observed in the Soviet Union with its many ethnic groups; it was recently reported, for example, that schizophrenia is significantly less common among Uzbeks in the Samarkand region compared with Russians in the Moscow region (Yursinova, 1982).

India is another country which has provided intriguing findings on the prevalence of schizophrenia. In a series of studies in villages in West Bengal, Nandi and his colleagues (1975, 1979, 1980) found the prevalence rate for schizophrenia to vary from 2.2 to 5.6 per 1000. The highest schizophrenia rates were found among Indians of the highest castes. This is the ninth group of researchers over the past 50 years to find that schizophrenia occurs more commonly in India in individuals of higher castes (Dhunjibhoy, 1930; Shaw, 1930; Rao, 1966; Dube, 1970; Elnager *et al*, 1971; Dube & Kumar, 1972; Saxena *et al*, 1972; Kapur, 1975; Carstairs & Kapur, 1976; Varma *et al*, 1980). This observation contrasts with claims in the USA and Western Europe that schizophrenia occurs more commonly in lower socioeconomic groups. At least a partial explanation for the latter is 'downward drift' of individuals prone to schizophrenia; it is difficult to imagine how 'upward drift' could account for the Indian findings however.

Reports that there are large areas of the world in which schizophrenia is uncommon must be viewed with caution since many such reports are based on impressions only. Where schizophrenia is in excess it can be counted, but where there is a deficit it can always be said that the cases were missed or died off. In sub-Saharan African countries there have been such reports over many years. The surveys by Giel & Van Luijk (1969*a*,*b*) in Ethiopia and Ahmed's work in the Sudan (1979) (0.4 per 1000) are two more recent examples of such impressions. The best study methodologically from Africa was that of Ben-Tovim & Cushine (1986) of rural Botswana; omitting the patient with an onset at age 60 they reported a prevalence rate of 2.0 (total population) or 4.4 (15+) per 1000 which are higher rates than previous African studies.

The South Pacific is the other area of the world from which there have been reports of schizophrenia being uncommon. Torrey *et al* (1984) studied hospitalisation records for psychiatric disorders in Papua New Guinea and concluded that there was a 20-fold difference between some highland districts (very low prevalence) and some coastal districts (comparatively higher prevalence). Zigas & Rodrigue (Dohan *et al*, 1983) also had the impression that schizophrenia was rare in some highland areas. Murphy (1984), however, stated that "I myself could find only a two- to three-fold difference when I conducted a key-informant village survey in various highland and coastal districts.'

Elsewhere in Melanasia, in the Solomon Islands Clark (Dohan *et al*, 1983) found no schizophrenia among 3000 Kwaio adults living in remote villages but a prevalence rate of 2.2 per 1000 among 32 000 adults in the coastal region. In Polynesia, Murphy &

Taumoepeau (1980) reported a low prevalence of schizophrenia (and also organic and affective psychosis) on Tonga. Dale (1981) did an extensive survey of Micronesian islands and reported a point prevalence for schizophrenia of 1.5 per 1000 (age 15 and over). Among the various islands, however, there was an east-to-west gradient with rates of 0.8 per 1000 in the Marshall Islands to 9.7 per 1000 on Yap. Three decades earlier a survey had found no individuals with schizophrenia on Yap (Dohan *et al*, 1983).

Given the lack of methodological rigour in most of these surveys it is not possible to draw any firm conclusions. The impression remains, however, that there are some areas in the world where schizophrenia is uncommon. These areas are tropical, and when compared with the higher schizophrenia prevalence rates reported for western Ireland and northern Scandinavia there is the suggestion of a possible north–south gradient in the disease's distribution similar to the gradient reported for heart disease (Thom *et al*, 1985) and multiple sclerosis (Kurtzke, 1975).

Changes in prevalence over time

Research on prevalence rates of schizophrenia is complicated by the fact that the rates may change over time. Suggestions that the prevalence of schizophrenia may have increased sharply in the nineteenth century have elicited much controversy (Torrey, 1980; Hare, 1983). In Books studies of northern Sweden the schizophrenia prevalence rate increased from 9.4 to 17.0 per 1000 over 25 years, while in Finland the prevalence of schizophrenia apparently increased less dramatically over 5 years (Lehtinen & Vaisanen, 1981). In the USA there are also suggestions that the schizophrenia rate may have increased in recent years; 1984 studies in Baltimore and New Haven using a narrow definition of schizophrenia report schizophrenia prevalence rates (Myers *et al*, 1984) which appears to be twice as high as earlier studies which used a broader definition in Baltimore (Lemkau *et al*, 1942, 1943) and New Haven (Hollingshead & Redlich, 1958).

Schizophrenia prevalence rates may also decrease. The rural areas in southern Sweden studied by Essen-Moller (1956) was restudied 10 years later by Hagnell (1966) who found a substantial decrease in the number of individuals with schizophrenia. Shepherd (1957) claimed that England's Buckinghamshire County experienced a major decrease in first admissions for schizophrenia between 1931–1933 and 1945–1947. In Scotland, Eagles & Whalley (1985) claimed that schizophrenia had decreased in that country between 1969 and 1978. It is also interesting to speculate whether the difference in Scotland between Mayer-Gross' 1948 study (4.2 per 1000) and the 1981 Aberdeen case register (1.4 per 1000) is a result of a decrease in schizophrenia or because the two studies covered different geographical regions (southern and northeastern regions respectively). In Ireland, data from the three county case register for 1973 (Walsh *et al*, 1980) compared with 1982 (Walsh, pers. comm., 1986) suggests that a decrease in schizophrenia prevalence may be taking place in that country as well. Finally, a recent New Zealand study suggests that schizophrenia may also be decreasing there (Joyce, 1987). Some of these apparent changes may of course also be due to changing diagnostic practices as in recent decreases in first admissions for schizophrenia in Denmark (Munk-Jorgensen, 1986; Munk-Jorgensen & Jorgensen, 1986).

There are also examples of schizophrenia prevalence rates remaining relatively constant over time. Odegard (1971) reported that in Norway between 1926 and 1965 the number of individuals in hospital with 'functional psychoses' increased very little, while case registers in Denmark and Iceland also showed no substantial change (Hafner & Heiden, 1986). Dilling & Weyerer (1984) studied villages in Southern Germany and found an age-corected schizophrenia prevalence rate of 3.9 per 1000. Brugger had carried out two research studies in the same region 40 years earlier and found virtually identical age-corrected rates (3.8 and 3.9 per 1000) (Torrey, 1980). In both sets of studies the institutionalised schizophrenics were not included.

Perhaps the most intriguing report of a changing schizophrenia prevalence rate came from studies in Northern Ghana. From 1934 to 1937 Fortes, trained in both psychology and anthropology, studied Tallensi villages there; among approximately 5000 people he found only one who was psychotic, a 22-year-old man who had become ill in adolescence. In 1963 Fortes returned to the village accompanied by his wife, Dr Mayer, a psychiatrist. In the same population where 27 years earlier there had been a single psychotic individual there were now 17; of these, 13 were diagnosed with schizophrenia and one each with manic-depressive psychosis, acute reactive psychosis, postpartum psychosis, and involuntional depression. Fortes & Mayer (1969) noted that Tallensi elders were themselves alarmed over the rise in psychosis; organic factors (such as trypanosomiasis) or immigration were rejected as being causal factors. Fortes was also certain that he had not missed the cases in his earlier study: "What was quite startling . . . is that several of these cases occurred in families which were specially well known to me in 1934–1937 . . . These were the families of my best friends and informants, some of whom are still living. Had such cases occurred among them in 1934–1937 I could not have missed them'' (Fortes & Mayer, 1969).

Prognosis and seasonality

As mentioned previously, the course and prognosis of schizophrenia will affect prevalence rates if they differ among different groups. If, for example, a much higher percentage of patients fully recover in country A versus country B, then the prevalence rate in the country A will be lower even if the incidence rates in the two countries are equal.

There is evidence that prognosis and outcome do vary considerably, with schizophrenic patients in developing countries having a higher percentage of very good outcomes. Murphy & Raman (1971) brought attention to this fact in their study of Mauritius in which, 12 years after hospital admission, 59% of individuals with schizophrenia were completely recovered; a comparison with British figures showed that, 5 years after admission, only 34% of individuals with schizophrenia had completely recovered. Waxler (1979) did a similar 5-year follow-up in Sri Lanka and found that 40% of the patients had recovered completely. The most definitive study of this problem to date was the multi-nation WHO study of schizophrenia. At 2-year follow-up in this study the percentage of patients with a 'very favourable' outcome ranged from 57% (Nigeria) and 49% (India) to 24% (England), 23% (USA), 21% (Columbia), 14% (Czechoslovakia), 9% (USSR) and 6% (Denmark). In all of these studies it is important to note that the percentage of schizophrenic patients having a 'very unfavourable' outcome is relatively comparable; the difference is that more patients in developing countries recover completely while more patients in developed countries have intermediate outcomes.

Seasonality of schizophrenic births is another interesting aspect of the disease which may have a bearing on prevalence differences. It has now been shown in every northern hemisphere country which has been studied with an adequate number of subjects that a disproportionate number of individuals, who later in life develop schizophrenia, were born in the late winter and early spring months. The studies to date include England and Wales, Ireland, Denmark, Norway, Sweden, the USA, Japan, and the Phillipines and are extensively reviewed in several recent publications (Bradbury & Miller, 1985; Torrey & Kaufmann, 1986; Boyd *et al*, 1986). The excess schizophrenic births vary between 7 and 20% in these studies and the phenomenon is not necessarily constant within a single country (e.g. northern Sweden shows more seasonality than southern Sweden, the northern states in the USA more than the southern states). Nor is the seasonality fixed over time with evidence from Japan, England and Wales, and the USA suggesting shifts. Although the number of countries studied is still too few to make firm generalisations, there does not appear to be a relationship between the percentage of excess schizophrenic births and the countries with higher or lower prevalence rates for schizophrenia. In view of the firmly established seasonality of schizophrenic births, however, it would be surprising to find that the prevalence of schizophrenia did not vary around the world since seasons do vary from region to region.

Discussion

Despite methodological limitations, prevalence studies of schizophrenia published since 1948 suggest that there may be significant differences among population groups. Although the magnitude of these differences is debatable, I believe that a ten-fold range of prevalence for schizophrenia is supported by the data; this would be approximately comparable to the prevalence range reported for rheumatoid arthritis.

Since all hypotheses regarding the aetiology of schizophrenia would logically predict prevalence differences, it is not surprising that advocates of these hypotheses have utilised prevalence data to support their positions. Book (1953, 1978) explained his findings in northern Sweden by genetic mechanisms of inbreeding among the isolated population. Dohan *et al* (1983) claimed that the distribution of schizophrenia best fit nutritional theories of neuroactive peptides from grain glutens. Torrey (1980) put forth neuroviruses as the most logical explanation for the prevalence differences, and Murphy (1975) invoked psychosocial mechanisms such as the mother–son relationship in Ireland and the stress of acculturation in Ghana (Murphy, 1972) to explain the findings in those countries. The fact is that the studies on the geographical distribution of schizophrenia done to date do not point in any single aetiological direction.

The prevalence studies of schizophrenia do suggest important questions which need to be pursued. What portion of the lower rates reported from developing countries can be accounted for by the more benign course of schizophrenia in those countries? Is there a north–south gradient as has been shown for heart disease and multiple sclerosis? How much of the prevalence differences or changes over time are due to diagnostic differences? Groups such as the American Hutterites, northern Swedes, western Irish, and castes in India should be studied in detail with the collection of genetic, biochemical, nutritional, virological, immunological, and psychosocial data. Migrants from suspected high prevalence or low prevalence areas need to be followed to see whether their prevalence rate changes in their new home, similar to the work done in multiple sclerosis. Data on the seasonality of schizophrenic births should be collected in many more countries to ascertain its possible relationship to the prevalence of the disease. Ongoing case registers should be set up in areas thought to have a very high or very low prevalence.

There is one important advantage in studying the prevalence of schizophrenia—it does not require sophisticated and expensive laboratory equipment. We have grown accustomed in the waning years of the twentieth century to equating research with labora-tories. This is unfortunate, for some of the potentially most promising research on schizophrenia can be pursued with a diagnostic manual, pocket computer, and clinical acumen. There are methodological problems to be certain, but they are not any more formidable than those in laboratory research. As tragic a disease as schizophrenia is, it is also one of the greatest intellectual challenges of contemporary medicine.

References

AHMED, A. H. (1979) Consanguinity and schizophrenia in Sudan. *British Journal of Psychiatry*, **134**, 635-636.

ANDERSON, T. (1975) Physical and mental illness in a Lapp and Norwegian population. *Acta Psychiatrica Scandinavica* (suppl.) **263**, 47-56.

BABIGIAN, H. M. (1985) Schizophrenia epidemiology. In *Comprehensive Textbook of Psychiatry* IV (eds H. I. Kaplan & B. J. Saddock). Baltimore: Williams & Wilkins.

BASH, K. W. & BASH-LIECHTI, J. (1974) Studies on the epidemiology of neuropsychiatric disorder among the population of the city of Shiraz, Iran. *Social Psychiatry*, **9**, 163-171.

BEN-TOVIM, D. I. & CUSHNIE, J. M. (1986) The prevalence of schizophrenia in a remote area of Botswana. *British Journal of Psychiatry*, **148**, 576-580.

BJARNAR, E., REPPESGAARD, H., ASTRUP, C. (1975) Psychiatric morbidity in Berlevag. *Acta Psychiatrica Scandinavica* (suppl.) **262**, 60-67.

BLAZER, D., GEORGE, L. K., LANDERMAN, R., PENNYBACKER, M., MELVILLE, M. L., WOODBURY, M., MANTON, K. G., JORDAN, K., LOCKE, B. (1985) Psychiatric disorders: A rural /urban comparison. *Archives of General Psychiatry*, **42**, 651-656.

BOOK, J. A. (1953) A genetic and neuropsychiatric investigation of a North-Swedish population. *Acta Genetica et Statistica Medica*, **4**, 1-100.

——, WETTERBERG, L. & MODRZEWSKA, K. (1978) Schizophrenia in a North Swedish geographical isolate 1900-1977: epidemiology, genetics and bio-chemistry. *Clinical Genetics*, **14**, 373-394.

BOYD, J. H., PULVER, A. E. & STEWART, W. (1986) Season of birth: schizophrenia and bipolar disorder. *Schizophrenia Bulletin*, **12**, 173-186.

BRADBURY, J. N. & MILLER, G. A. (1985) Season of birth in schizophrenia: A review of evidence, methodology, and aetiology. *Psychological Bulletin*, **98**, 569-594.

BREMER, J. (1951) A social psychiatric investigation of a small community in northern Norway. *Acta Psychiatrica et Neurologica Scandinavica* (suppl.) **62**, 1-166.

CARSTAIRS, G. M. & KAPUR, R. L. (1976) *The Great Universe of Kota: A Study of Stress, Change, and Mental Disorder in an Indian Village*. Berkeley: University of California Press.

COOPER, B. (1978) Epidemiology. In *Schizophrenia: Towards a New Synthesis* (ed. J. K. Wing). London: Academic Press.

CROCETTI, G. M., LEMKAU, P. V., KULCAR, Z. & KESIC, B. (1971) Selected aspects of the epidemiology of psychoses in Croatia, Yugoslavia: The cluster sample and the results of the pilot survey. *American Journal of Epidemiology*, **94**, 126-134.

DALE, P. W. (1981) Prevalence of schizophrenia in the Pacific Island populations of Micronesia. *Journal of Psychiatric Research*, **16**, 103-111.

DHUNJIBHOY, J. E. (1930) A brief resume of the types of insanity commonly met with in India with a full description of 'Indian Hemp Insanity' peculiar to the country. *Journal of Mental Science*, **76**, 254-264.

DILLING, H. & WEYERER, S. (1984) Prevalence of mental disorders in the small-town, rural region of Traunstein (Upper Bavaria). *Acta Psychiatrica Scandinavica*, **69**, 60-79.

DOHAN, F. C., HARPER, E. H., CLARK, M. H., RODRIGUE, R. & ZIGAS, V. (1983) Where is schizophrenia rare? *Lancet*, **ii**, 101.

DUBE, K. C. (1970) A study of prevalence and biosocial variables in mental illness in a rural and an urban community in Uttar Pradish, India. *Acta Psychiatrica Scandinavica*, **46**, 327-359.

—— & KUMAR, N. (1972) An epidemiological study of schizophrenia. *Journal of Biosocial Science*, **4**, 187-195.

EAGLES, J. M. & WHALLEY, L. J. (1985) Decline in the diagnosis of schizophrenia among first admissions to Scottish mental hospitals from 1969-1978. *British Journal of Psychiatry*, **40**, 151-154.

EASTWELL, H. (1975) Schizophrenic typology in Aboriginal Australia. Mimeo.

EATON, J. W. & WEIL, R. J. (1955) *Culture and Mental Disorders: A Comparative Study of the Hutterites and Other Populations*. Glencoe, Illinois: Free Press.

EATON, W. W. (1985) Epidemiology of schizophrenia. *Epidemiologic Reviews*, **7**, 105-126.

EGELAND, J. A. & HOSTETTER, A. M. (1983) Amish study, I: Affective disorders among the Amish, 1976-1980. *American Journal of Psychiatry*, **140**, 56-61.

ELNAGAR, M. N., MAITRA, P. & RAO, M. N. (1971) Mental health in an Indian rural community. *British Journal of Psychiatry*, **118**, 499-503.

ESQUIROL, J. E. (1845) *Mental maladies* quoted in *A Documentary History of Psychiatry* by C. E. Goshen (1967). New York: Philosophical Library.

ESSEN-MOLLER, E. (1956) Individual traits and morbidity in a Swedish rural population. *Acta Psychiatrica et Neurologica Scandinavica* (suppl.) **100**, 1-160.

FORTES, M. & MAYER, D. Y. (1969) Psychosis and social change among the Tallensi of Northern Ghana. In *Psychiatry in a Changing Society* (eds S. H. Foulkes & G. S. Prince). London: Tavistock.

FREEMAN, H. & ALPERT, M. (1986) Prevalence of schizophrenia in an urban population. *British Journal of Psychiatry*, **149**, 603-611.

FUGELLI, P. (1975) Mental health and living conditions in a fishing community in northern Norway. *Acta Psychiatrica Scandinavica* (suppl.) **263**, 39-42.

GIEL, R. & VAN LUIJK, J. N. (1969*a*) Psychiatric morbidity in a small Ethiopian town. *British Journal of Psychiatry*, **115**, 149-162.

—— & —— (1969*b*) Psychiatric morbidity in a rural village in South-Western Ethiopia. *International Journal of Social Psychiatry*, **16**, 63-71.

Hafner, H. & Heiden, W. van der (1986) The contribution of European case registers to research on schizophrenia. *Schizophrenia Bulletin*, **12**, 26–51.

—— & Klug, J. (1980) First evaluation of the Mannheim community mental health service. *Acta Psychiatrica Scandinavica*, **62** (suppl. 285), 68–77.

Hagnell, O. (1966) *A Prospective Study of the Incidence of Mental Disorder*. Lund: Norstedts-Bonniers.

Halldin, J. (1984) Prevalence of mental disorder in an urban population in central Sweden. *Acta Psychiatrica Scandinavica*, **69**, 503–518.

Hare, E. (1983) Was insanity on the increase? *British Journal of Psychiatry*, **142**, 439–455.

Hollingshead, A. B. & Redlich, F. C. (1958) *Social Class and Mental Illness*. New York: Wiley.

Jablensky, A. (1986) Epidemiology of schizophrenia: A European perspective. *Schizophrenia Bulletin*, **12**, 52–73.

—— & Sartorius, N. (1975) Culture and schizophrenia. *Psychological Medicine*, **5**, 113–124.

Jayasundera, M. G. (1969) Mental health surveys in Ceylon. In *Mental Health Research in Asia and the Pacific* (eds W. Caudill & T.-Y. Lin). Honolulu: East–West Center Press.

Jones, I. H. & Horne, D. J. (1973) Psychiatric disorders among Aborigines of the Western Desert. *Social Science and Medicine*, **7**, 219–228.

Joyce, P. R. (1987) Changing trends in first admissions and readmissions for mania and schizophrenia in New Zealand, 1974 to 1984. *Australia and New Zealand Journal of Psychiatry* (in press).

Kapur, R. L. (1975) An illustrative presentation of a population survey of mental illness in South India. In *Mental Health Services in Developing Countries* (eds T. A. Baasher *et al*). Geneva: World Health Organization.

Kato, M. (1969) Psychiatric epidemiological surveys in Japan. In *Mental Health Research in Asia and the Pacific* (eds W. Caudill & T.-Y. Lin). Honolulu: East–West Center Press.

Kraepelin, E. (1919) *Dementia Praecox and Paraphrenia*. Edinburgh: Livingstone.

Kulcar, Z., Crocetti, G. M., Lemkou, P. V. & Kesic, B. (1971) Selected aspects of the epidemiology of psychoses in Croatia, Yugoslavia: pilot study of communities. *American Journal of Epidemiology*, **94**, 118–125.

Kurtzke, J. F. (1975) A reassessment of the distribution of multiple sclerosis. *Acta Neurological Scandinavica*, **51**, 110–136.

Larrsson, T. & Sjogren, T. (1954) A methodological, psychiatric and statistical study of a large Swedish rural population. *Acta Psychiatrica et Neurologica Scandinavica* (suppl.) **89**, 1–250.

Leff, J. (1981) *Psychiatry Around the Globe: A Transcultural View*. New York: Marcel Dekker.

Lehtinen, V. & Vaisanen, E. (1981) Epidemiology of psychiatric disorders in Finland: A five-year follow-up. *Social Psychiatry*, **16**, 171–180.

Lemkau, P. V., Tietze, C. & Cooper, M. (1942) Mental hygiene problems in an urban district. *Mental Hygiene*, **25**, 624–646.

——, —— & —— (1943) Mental hygiene problems in an urban district: second paper. *Mental Hygiene*, **26**, 100–119.

Lin, T.-Y. (1953) A study of the incidence of mental disorder in Chinese and other cultures. *Psychiatry*, **16**, 313–336.

——, Rin, H., Yeh, E. K., Hsu, C. C. & Chu, H. M. (1969) Mental disorders in Taiwan fifteen years later. In *Mental Health Research in Asia and the Pacific* (eds W. Vaudill & T.-Y. Lin). Honolulu: East–West Center Press.

Marble, A., Krall, L. P., Bradley, R. F., Christlieb, A. R. & Soeldner, J. S. (eds) (1975) *Joslin's Diabetes Mellitus*. Philadelphia: Lea and Febiger.

Mayer-Gross, W. (1948) Mental health survey in a rural area. *Eugenics Review*, **40**, 140–148.

Mishler, E. G. & Scotch, N. A. (1963) Sociocultural factors in the epidemiology of schizophrenia: A review. *Psychiatry*, **26**, 315–351.

Munk-Jorgensen, P. (1986) Decreasing first-admission rates of schizophrenia among males in Denmark from 1970 to 1984. *Acta Psychiatrica Scandinavica*, **73**, 645–650.

—— & Jorgensen, P. C. (1986) Decreasing rates of first admission diagnoses of schizophrenia among females in Denmark 1970–1984. *Acta Psychiatrica Scandinavica*, **74**, 379–383.

Murphy, H. B. M. (1964) Community management of rural mental patients. Final report of USPHS Mental Health Grant. Mimeo.

—— (1968) Cultural factors in the genesis of schizophrenia. In *The Transmission of Schizophrenia* (eds D. Rosenthal & S. S. Kety). New York: Pergamon.

—— (1972) The schizophrenia-evoking rule of complex social demands. In *Genetic Factors in Schizophrenia* (ed. A. R. Kaplan). Springfield, Illinois: C. C. Thomas.

—— (1975) Alcoholism and schizophrenia in the Irish: A review. *Transcultural Psychiatric Research Review*, **12**, 116–139.

—— (1984) Diseases of civilization? *Psychological Medicine*, **14**, 487–490.

—— & Raman, A. C. (1971) The chronicity of schizophrenia in indigenous tropical peoples. *British Journal of Psychiatry*, **118**, 489–497.

—— & Taumoepeau, B. M. (1980) Traditionalism and mental health in the South Pacific: a re-examination of an old hypothesis. *Psychological Medicine*, **10**, 1–12.

—— & Vega, G. (1982) Schizophrenia and religious affiliation in Northern Ireland. *Psychological Medicine*, **12**, 595–605.

Myers, J. K., Weissman, M. M., Tischler, G. L., Holzer, C. E., Leaf, P. J., Orvaschel, H., Anthony, J. C., Boyd, J. H., Burke, J. D., Kramer, M. & Stoltzman, R. (1984) Six-month prevalence of psychiatric disorders in three communities. *Archives of General Psychiatry*, **41**, 959–967.

Nandi, D. N., Ajmany, S., Ganguli, H., Banerjee, G., Boral, G. C., Ghosh, A. & Sarkar, S. (1975) Psychiatric disorders in a rural community in West Bengal: An epidemiological study. *Indian Journal of Psychiatry*, **17**, 87–99.

——, Banerjee, G., Boral, G. C., Ganguli, H., Ajmany, S., Ghosh, A. & Sarkar, S. (1979) Socio-economic status and prevalence of mental disorders in certain rural communities in India. *Acta Psychiatrica Scandinavica*, **59**, 276–293.

——, Mukherjee, S. P., Boral, G. C., Banerjee, G., Ghosh, A., Sarkar, S. & Ajmany, S. (1980) Socio-economic status and mental morbidity in certain tribes and castes in India: A cross-cultural study. *British Journal of Psychiatry*, **136**, 73–85.

Nielsen, J. (1976) The Samso project from 1967 to 1974. *Acta Psychiatrica Scandinavica*, **54**, 198–222.

—— & Nielsen, J. A. (1977) A census study of mental illness in Samso. *Psychological Medicine*, **7**, 491–503.

Odegard, O. (1971) Hospitalized psychosis in Norway: time trends 1926–1965. *Social Psychiatry*, **6**, 54–58.

Rao, S. (1966) Caste and mental disorders in Bihar. *American Journal of Psychiatry*, **122**, 1045–1055.

Rin, H. & Lin, T.-Y. (1962) Mental illness among Formosan Aborigines as compared with the Chinese in Taiwan. *Journal of Mental Science*, **108**, 134–146.

Roy, C., Choudhuri, A. & Irvine, D. (1970) The prevalence of mental disorders among Saskatchewan Indians. *Journal of Cross-Cultural Psychology*, **1**, 383–392.

Sartorius, N., Jablensky, A. & Shapiro, R. (1978) Cross-cultural differences in the short-term prognosis of schizophrenic psychoses. *Schizophrenia Bulletin*, **4**, 102–113.

——, ——, Korten, A., Ernberg, G., Anker, M., Cooper, J. E. & Day, R. (1986) Early manifestations and first-contact incidence of schizophrenia in different cultures. *Psychological Medicine*, **16**, 909–928.

SAUNA, V. D. (1969) Socio-cultural aspects. In *The Schizophrenic Syndrome* (eds L. Bellak & L. Loeb). New York: Grune and Stratton.

SAXENA, B. M., BHASKARAN, K. & ANATH, J. V. (1972) Social class and schizophrenia: A study based on the caste system in India. *Transcultural Psychiatric Research Review*, **9**, 130–133.

SETHI, B. B., GUPTA, S. C., MAHENDRU, R. K. & KUMARI, P. (1974) Mental health and urban life: A study of 850 families. *British Journal of Psychiatry*, **124**, 243–247.

SHAW, W. S. (1930) Some observations on the etiology of dementia praecox. *Journal of Mental Science*, **76**, 505–511.

SHEPHERD, M. (1957) *A Study of the Major Psychoses in an English County*. Maudsley Monographs, No. 3. London: Chapman & Hall.

SIKANERTEY, T. & EATON, W. W. (1984) Prevalence of schizophrenia in the Labadi District of Ghana. *British Journal of Psychiatry*, **69**, 156–161.

SJOGREN, T. (1948) Genetic-statistical and psychiatric investigations of a West Swedish population. *Acta Psychiatrica et Neurologica* (suppl.) 52.

STROMGREN, E. (1948) Social surveys. *Journal of Mental Science*, **94**, 266–276.

THACORE, V. R., GUPTA, S. C. & SURAIYA, M. (1975) Psychiatric morbidity in a North Indian community. *British Journal of Psychiatry*, **126**, 364–369.

THOM, T. J., EPSTEIN, F. H., FELDMAN, J. J. & LEAVERTON, P. E. (1985) Trends in total mortality and mortality from heart disease in 26 countries from 1950 to 1978. *International Journal of Epidemiology*, **14**, 510–520.

TORREY, E. F. (1980) *Schizophrenia and Civilization*. New York: Jason Aronson.

—— & KAUFMANN, C. A. (1986) Schizophrenia and neuroviruses. In *The Neurology of Schizophrenia* (eds H. A. Nasrallah & D. R. Weinberger). Amsterdam: Elsevier.

——, MCGUIRE, M., O'HARE, A., WALSH, D. & SPELLMAN, M. P. (1984) Endemic psychosis in Western Ireland. *American Journal of Psychiatry*, **141**, 966–969.

UTSINGER, P. D., ZVAIFLER, N. J. & EHRLICH, G. E. (eds) (1985) *Rheumatoid Arthritis*. Philadelphia: J. B. Lippincott.

VAISANEN, E. (1975) Psychiatric disorders in Finland. *Acta Psychiatrica Scandinavica* (suppl.) **263**, 22–33.

VARMA, V. K., WIG, N. N., SRINIVSA MURTHY, R. & MISRA, A. K. (1980) Socio-demographic correlates of schizophrenia, affective psychoses and neuroses in a clinic in India. *Acta Psychiatrica Scandinavica*, **61**, 404–412.

WALSH, D., O'HARE, A., BLACK, B., HALPENNY, J. V. & O'BRIEN, P. F. (1980) The treated prevalence of mental illness in the Republic of Ireland: The three county case register study. *Psychological Medicine*, **10**, 465–470.

WAXLER, N. E. (1979) Is outcome for schizophrenia better in nonindustrial societies? The case of Sri Lanka. *Journal of Nervous and Mental Disease*, **167**, 144–158.

WHITE, W. H. (1903) The geographical distribution of insanity in the United States. *Journal of Nervous and Mental Diseases*, **30**, 257–279.

WIJESINGHE, C. P., DISSANAYAKE, S. A. W. & DASSANAYAKE, P. V. L. N. (1978) Survey of psychiatric morbidity in a semi-urban population in Sri Lanka. *Acta Psychiatrica Scandinavica*, **58**, 413–441.

WING, J. K. & FRYERS, T. (1976) *Statistics from the Camberwell and Salford Psychiatric Registers, 1964–1974*. London: Institute of Psychiatry.

WING, L., WING, J. K., HAILEY, A., BAHN, A. K. & SMITH, H. E. (1967) The use of psychiatric services in three urban areas: An international case register study. *Social Psychiatry*, **2**, 158–167.

YOO, P. S. (1961) Mental disorders in Korean rural communities. *Proceedings of the Third World Congress of Psychiatry*. Montreal: McGill University Press.

YURSINOVA, M. S. (1982) Some results of epidemiological research on schizophrenia in Samarkand. *Zhurnal Nevropathologii I Psikhiatrii Imeni S.S. Korsalova*, **82**, 93–98.

ZHARIKOV, N. M. (1968) Epidemiological study of mental illness in the U.S.S.R. *Social Psychiatry*, **3**, 135–138.

ZIMMERMAN-TANSELLA, C., BURTI, L., FACCINCANI, C., GARZOTTO, N., SICILIANI, O. and TANSELLA, M. (1985) Bringing into action the psychiatric reform in South-Verona: A five year experience. *Acta Psychiatrica Scandinavica* (suppl.) **316**, 71–86.

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Review Article

Hostility and Deliberate Self-Poisoning: The Role of Depression

R. FARMER

The evidence for an association between hostility and deliberate self-poisoning is reviewed. Although the concept of hostility is poorly defined and different measuring instruments may not measure the same entity, self-poisoning subjects as a group appear to have very high levels of hostility. However, depression is also associated with hostility and when studies have taken into account the psychiatric diagnosis of overdose patients it is outwardly-directed hostility which distinguishes these individuals from other psychiatric patients or normals. A model is proposed to show the inter-relationships of hostility, depression and self-poisoning.

Following an epidemic rise in incidence in the 1960s and early 1970s deliberate self-poisoning became the most common reason for acute admission of women to hospital, and second only to ischaemic heart disease as the most common reason for men (Farmer, 1986). Its aetiology remains unclear. The motivation to take an overdose is often complex, with frequently a simultaneous interplay of different psychological motives in any individual.

Kreitman (1978) has reviewed some of the possible themes: (1) seeking an interruption from unendurable tension (Shneidman, 1964); (2) attempting to secure the attention of others as in the 'cry for help' (Stengel & Cook, 1958); (3) self-poisoning as a means of inducing guilt in others; (4) testing the benevolence of fate; and (5) true suicidal intention, the intention of actually ending one's life. Notable in two of these, (2) and (3), is the implication that negative, hostile feelings towards other persons may play a major role in self-poisoning. If so, this would have important consequences for the management of these patients, and for the prevention of further attempts and completed suicide. Also, the presence of true suicidal intent, motivation (5), is consistent with the psychodynamic view that hostility may also be directed inwards on the self.

But, what is the empirical evidence for a link between hostility and self-poisoning? When the literature is reviewed it becomes clear that the concept of hostility has several different meanings (Gottschalk *et al*, 1963) and the different instruments which have been developed to measure it do not necessarily measure the same thing (Edmunds & Kendrick, 1980). Hostility directed outwards includes at least four inter-related factors, any one of which may be erroneously equated by an investigator with the global construct of hostility. The factors are (1) an act, either physical or verbal, sometimes called 'aggression' which is interpreted as having a destructive function by an outside observer; (2) a self-reported attitude of dislike, resentment, suspicion towards other persons or objects sometimes called 'hostility'; (3) a subjective experience of an affect called 'anger'; (4) a predisposition of character to become hostile or aggressive. Any particular measuring instrument may focus on one or more aspects of this aggression-hostility-anger complex without indicating explicitly the meaning intended. Likewise the concept of hostility directed towards the self is frequently ill-defined and sometimes it is not differentiated from outwardly directed hostility.

The various kinds of measuring instrument have different theoretical bases, aims and characteristics (Edmunds & Kendrick, 1980). Projective tests aim to evaluate deeper, even unconscious hostility traits, but have been unwieldy to manipulate, difficult to quantify and unreliable. Questionnaires, scales and inventories, while relatively straightforward to administer and score, may suffer from response bias and ambiguity of meaning of specific items. Two scales which are commonly used and whose items were selected as being logically related to particular psychological theories are the Hostility and Direction of Hostility Questionnaire, HDHQ (Caine *et al*, 1967) and the Buss–Durkee Inventory (Buss & Durkee, 1957). These theoretically developed scales, like intuitively derived instruments, depend for their usefulness on the subject's self-knowledge and truthfulness and the perspicuity of the test items. Because of their theoretical orientation, however, their content validity is more clearly defined and it is easier to determine what the devices are supposed to measure. Studies using this kind of measure have been considered particularly important in this review.

The way the HDHQ was designed is congruent with a psychodynamic approach to hostility as Foulds (1965) assumed that hostility was a unitary entity which could, however, be directed inwards on the self or outward against others. Validation studies have offered support for this notion. Foulds *et al* (1960) found that while the correlations between the subscales were positive, suggesting a factor of general hostility, the patterns of correlations indicated that the extrapunitive subscales were measuring something different from the intropunitive ones. Hope (1963) and Philip (1968) tested the assumptions regarding the unitary nature of hostility and its direction inward or outward by principal component analysis of scores on normal and neurotic subjects. A similar component structure was found for both kinds of subject, the first component being unipolar with all five subscales represented, while the second contrasted the intropunitive and extrapunitive measures. The Buss-Durkee Inventory aims to measure aggressiveness and hostility as separate entities. Attempts to assess the empirical validity of the instrument have generally yielded negative results but a number of studies indicate that the subscales possess construct validity (Edmunds & Kendrick, 1980).

Given the conceptual and semantic problems which make comparison between studies using different measures difficult, the evidence for a link between hostility and self-poisoning is reviewed below. Also examined is the evidence for an association between depression and hostility. As many individuals who take an overdose have an identifiable depressive syndrome, if depression is independently related to hostility this would have to be considered in any close examination of hostility in self-poisoning. Those studies which have taken psychiatric diagnosis into account would then be of particular importance. Firstly however, how hostile are overdose subjects generally?

Hostility in self-poisoning subjects considered collectively

In a study by Lester (1967) the resentment and irritability subscales of the Buss-Durkee Inventory differentiated psychology undergraduates who had attempted or threatened suicide ('suicidal') from those who had at the most only considered suicide ('non-suicidal'), scores being higher in the former group. In this study the validity of data on past 'suicidal' behaviour and thoughts depended totally on subjects' recall and self-disclosure, and the theoretical grounds for including subjects who only threatened suicide alongside suicide attempters are uncertain. In a subsequent study (Lester, 1968), when neuroticism was controlled for, the apparent association between resentment and attempted or threatened suicide disappeared. Conte & Plutchik (1974) found that parasuicide patients scored significantly higher than 'non-suicidal' psychiatric patients on various measures of hostility, but the diagnostic profiles of the two groups were quite different and the status of the 'suicide attempters' is ambiguous as several subjects made what are described only as 'gestures'.

Several studies have used the Hostility and Direction of Hostility Questionnaire, HDHQ. Vinoda (1966) compared female parasuicides admitted to hospital, matched psychiatric controls and normal subjects. The parasuicide patients showed a similar pattern of diagnoses to the psychiatric patients who had no record of attempted or threatened suicide. There were significant differences in general hostility among the three groups with highest levels in the parasuicides and lowest in the normal controls. Similar results were obtained by Philip (1970) who assessed 100 admitted parasuicide subjects and drew comparisons with previous data concerning normal persons and neurotic patients (Philip, 1968). Again in a study by Eastwood *et al* (1972) 92 subjects admitted to hospital after parasuicide were found to be relatively hostile with a mean general hostility score almost two standard deviations above that of Philip's normals (1968).

These studies suggest therefore that self-poisoning subjects have relatively high levels of hostility, certainly that they are more generally hostile than normal or neurotically depressed patients. In terms of inwardly-directed hostility, the HDHQ studies suggest that overdose patients may be intermediate between normal and neurotic depressives who tend to have the highest levels.

These studies have looked at overdose subjects as a group and have not considered what effect the presence or absence of psychiatric illness, particularly depression, might have on hostility scores. There is good agreement that depression is the most commonly identified disorder in self-poisoning subjects and personality disorder the second (Vinoda, 1966; Morgan *et al*, 1975; Newson-Smith & Hirsch, 1979; Urwin & Gibbons, 1979), although estimates of the prevalence of depression have varied between 35 and 79% (Weissman, 1974).

In looking at the role of hostility in self-poisoning more closely, if an independent association were to exist between hostility and depression this would have to be taken into account. So what is the evidence for this?

Hostility and depression

Abraham (1911) was one of the first to draw attention to the relationship between depressive illness and hostility. The classical psychoanalytical view (Freud 1917; Abraham, 1924) is that hostility held against a loved 'object' becomes turned against the self when that object is lost, by a process of introjection.

Empirical studies have since confirmed a relationship between hostility and depression but there has been disagreement over the nature of this association.

Certain studies using the HDHQ have suggested that hostility is directed *inwards* predominantly in depression thereby lending support to psychoanalytical theory (Mayo, 1967; Philip 1971; Blackburn, 1974; Blackburn *et al*, 1979). On the other hand, consistent with Rado's observation (1928) of the depressive as 'irritable and brittle, easily taking offence' other studies have emphasised the *extrapunitiveness* of depressed subjects (Wessman *et al*, 1960. Weissman *et al*, 1971). Others again have suggested that hostility may be directed both *inward and outward* in depression (Gershon *et al*, 1968; Klerman & Gershon, 1970; Friedman, 1970).

Multivariate analysis studies of depressed persons represent a different way of looking at hostility and depression. In a study by Schless *et al* (1974) four questionnaires including the Buss–Durkee Inventory were administered to 37 depressed patients and four patterns of hostility expression emerged from factor analysis of the data. These were (1) anxious, guilt-ridden, inward turning of hostility with resentment; (2) verbal hostility with negativatism; (3) anxious, suspicious, resentful, control of hostile feelings; (4) assaultive, verbally abusive, outward expression of hostility. The overall group was dichotomous with regard to the direction of hostility expression, equal numbers showing a predominance of hostility inward or outward. However, sample size was small and it is difficult to exclude both type 1 and type 2 errors, particularly in the absence of any estimates of error.

Paykel (1971) applied a cluster analysis technique to data on 165 depressed patients in a variety of treatment settings although a specific measure of hostility was not used. At the first level of analysis two groups were obtained: one older and more severely ill, the other younger and more mildly ill. Further division produced four groups: (1) psychotic; (2) anxious; (3) hostile; and (4) young depressives with personality disorder. The group 3 subjects had particularly high hostility and self-pity scores. The 'psychotic' group had similar scores on these variables but in the 'hostile' depressives the hostility and self-pity were striking in comparison with the otherwise mild symptomatology. A factor analysis study by Overall *et al* (1966) on only in-patients delineated three groups corresponding to Paykel's first three categories.

From the above it can be seen there is a lack of consensus regarding the predominant direction of hostility in depression. Possible reasons for this include:

Methodological shortcomings

The conclusions that can be drawn from some studies are limited by (1) small sample size (e.g. Gershon *et al*, 1968; Schless *et al*, 1974; Blackburn *et al*, 1979); (2) a failure to use standardised diagnostic criteria for depression (Wessman *et al*, 1960; Friedman, 1970); (3) failure to check inter-rater reliability (e.g. Mayo, 1967; Weissman *et al*, 1971); (4) failure to use validated reliable measures of hostility (Weissman *et al*, 1971; Paykel, 1971). Projective tests have been used in certain studies (e.g. Wessman *et al*, 1960; Gershon *et al*, 1968; Klerman & Gershon, 1970) and may lack the reliability of more structured instruments.

The heterogeneity of the depressed subjects studied

The studies using multivariate analysis suggest that the expression of hostility may differ among different subtypes of patient.

The complex nature of hostility

It is likely that different instruments measure different aspects of hostility. Inwardly- and outwardly-directed hostility may be different phenomena and in certain studies the direction of hostility has not been clearly specified.

It is, however, possible to draw some conclusions. It appears inwardly-directed hostility rises sharply with severe depression. However, certain individuals may be predominantly extrapunitive when depressed. Philip (1969) has suggested that extrapunitiveness as measured by the HDHQ is a personality trait. This view is supported by its relative stability over time (Mayo, 1967; Philip, 1971; Blackburn *et al*, 1979) and also by the high levels of extrapunitiveness in overdose 'repeaters' and self-poisoning subjects who have received treatment for alcohol or drug misuse (Farmer, 1985; Farmer & Creed, 1986). If this is true, depression may act as a releaser for the expression of this personality characteristic. On the other hand, of course, personality difficulties may lead to adverse situations such as relationship or work problems, or alcohol and other drug misuse with consequent emotional distress and even an identifiable depressive syndrome in some cases.

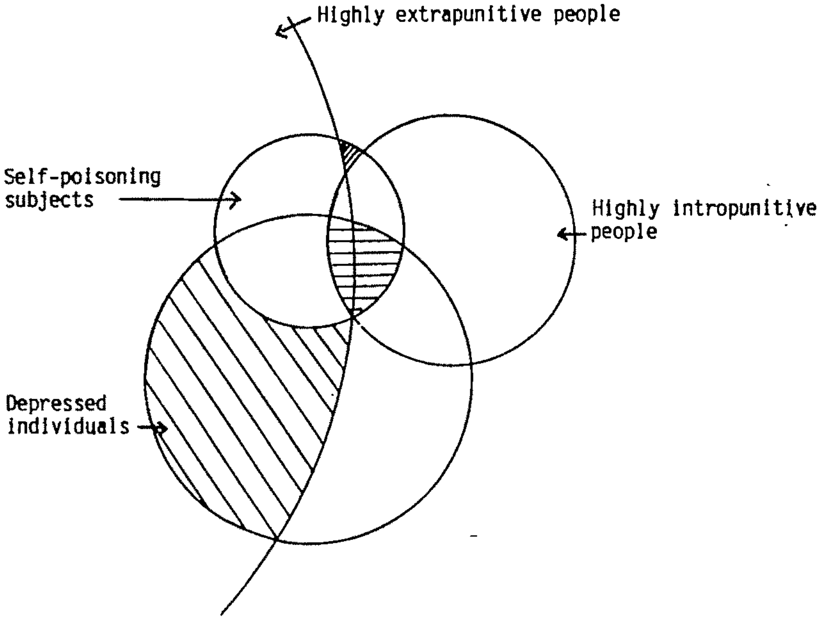
Given the association between hostility and depression, those studies which have taken account of the presence of depression in self-poisoning subjects become particularly important.

Hostility and self-poisoning, the role of depression considered

Certain studies have contributed to an understanding of the relative aetiological roles of hostility and depression in self-poisoning. In a study by Paykel & Dienelt (1971), 189 depressed patients were followed up 10 months after the initial contact and those subjects who had made 'suicide attempts' during that time were compared retrospectively with those who had not. Hostility was assessed from two self-report scales administered at initial interview and by a psychiatrist at initial and follow-up interviews. A distinguishing characteristic of the suicide attempters was their outwardly directed hostility. Guilt which would reflect inwardly directed hostility was not scored higher by the parasuicide group.

Crook *et al* (1975) divided 308 depressed in-patients into 'suicide attempters' and 'non-attempters', although 'attempts' included merely threats of self-harm. The attempters displayed more hostility including irritability towards others on a variety of measures including the Buss–Durkee Inventory, but their younger age compared to the non-attempters is a possible confounding factor.

While these two studies did not ensure that levels of depression were similar in the parasuicide and non-parasuicide groups this was attempted by Weissman *et al* (1973) and Yesavage (1983). In the first study, although an instrument designed specifically to measure hostility was not used, the parasuicides were judged to be more hostile and demanding during interview with evidence of more hostile interpersonal relations outside the clinical setting. Yesavage (1983) found that 'indirect hostility' and irritability as measured by the Buss–Durkee Inventory correlated



The circles represent four overlapping populations: self-poisoning subjects, depressed individuals, people with high levels of intropunitive hostility and people who are highly extrapunitive. The shaded areas represent three subgroups to illustrate use of the model.

▤ 1. Highly intropunitive and depressed self-poisoning subjects.

▨ 2. Self-poisoning subjects with low hostility levels.

▧ 3. Highly extrapunitive and depressed people who do not deliberately poison themselves.

FIG. 1 Hostility and depression in deliberate self-poisoning.

positively with self-destructive acts in depressed in-patients. 'Indirect hostility' referred to damage to property, gossip or practical jokes. Because levels of depression were not associated with self-harm it was suggested this kind of hostility might be a better predictor of parasuicide than the degree of depression.

A different approach involves dividing self-poisoning subjects into depressed and non-depressed. Farmer & Creed (1986) compared HDHQ scores in patients divided according to Present State Examination ratings. While the depressed subjects were more intropunitive, comparison with previous data on psychiatric patients and normals suggested that extrapunitive hostility might characterise self-poisoning subjects as a group.

Therefore, these studies suggest that among depressed patients those who have attempted to harm themselves, usually by taking an overdose, are more outwardly hostile than those who have not. When non-depressed self-poisoning subjects have been compared to normals their high extrapunitive scores have again been a distinguishing factor.

Summary

Self-poisoning patients appear to have very high levels of hostility, both inwardly- and outwardly-directed.

Studies using the HDHQ have found overdose patients to be more generally hostile than normal individuals or psychiatric patients and probably intermediate in intropunitiveness between these two groups.

Not all studies of hostility and self-poisoning have taken account of psychiatric diagnosis. This is important because depression, the most frequently occurring psychiatric disorder in overdose subjects, is independently associated with hostility. Inwardly-directed hostility seems to be related to severe depression but extrapunitive hostility, thought to be a personality trait, may also be associated with depressive symptoms.

Levels of extrapunitive hostility discriminate between non-depressed self-poisoning subjects and normals, and also between those depressed subjects who have deliberately taken an overdose and those who have not. Thus extrapunitiveness appears to characterise self-poisoning subjects.

The figure shows a tentative model of hostility as measured by the HDHQ and depression in self-poisoning in the form of a Venn diagram. Four overlapping populations are represented: self-poisoning subjects, individuals with a depressive syndrome, persons with high levels of inwardly directed hostility, and those who are highly extrapunitive. Any such model must be a oversimplification and the relative sizes of the groups are not claimed to be accurate; they would depend anyway on the thresholds used for depression and high or low hostility scores. Also it is important to distinguish between 'trait' and 'state' measures (Fava, 1986). While extrapunitiveness appears to be a 'trait' (a long-standing, enduring disposition), intropunitiveness seems to be mainly a 'state' which is temporary and changeable. As shown most self-poisoning subjects have high extrapunitive scores. A minority of depressives (mostly severely depressed) and also a minority of overdose patients have high levels of inwardly-directed hostility. Three groups are shown to illustrate the use of this model: (1) those highly intropunitive and depressed subjects (mostly severely depressed) who deliberately take an overdose; (2) the small number of overdose subjects who have low hostility levels including perhaps some of those with altruistic motives (Durkeim, 1951); (3) those highly extrapunitive people (many with personality difficulties) who develop an identifiable depressive syndrome without, however, taking an overdose. Personality-related problems may then be expressed in other ways such as an unstable work record or substance abuse.

The association between extrapunitive hostility, which seems related to personality, and self-poisoning has been stressed and may partly explain why some individuals deliberately take overdoses at times of stress while others do not. A prospective study of depressed subjects, with and without a history of self-poisoning would not only clarify the status of extrapunitiveness as a personality trait but also whether it has any predictive value for self-poisoning. Intropunitive hostility has been found to be associated not only with depression but also with high suicidal intent in self-poisoning subjects (Farmer & Creed, 1986). Again a longitudinal study would determine whether its measurement would assist in the assessment of patients who have taken overdoses.

References

ABRAHAM, K. (1911) Notes on the psychoanalytical investigation and treatment of manic-depressive insanity and allied conditions. In *Selected Papers on Psychoanalysis*. London: Hogarth Press (1927).

—— (1924) A short study of the development of the libido. In *Selected Papers on Psychoanalysis*. London: Hogarth Press (1927).

BLACKBURN, I. M. (1974) The pattern of hostility in affective illness. *British Journal of Psychiatry*, **125**, 141–145.

——, LYKETSOS, G. & TSCANTIS, J. (1979) The temporal relationship between hostility and depressed mood. *British Journal of Psychiatry*, **18**, 227–235.

BUSS, A. G. & DURKEE, A. (1957) An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, **21**, 343–349.

CAINE, T. M., FOULDS, G. A. & HOPE, K. (1967) *Manual of the Hostility and Direction of Hostility Questionnaire (HDHQ)*. London: University of London Press.

CONTE, H. R. & PLUTCHIK, R. (1974) Personality and background characteristics of suicidal mental patients. *Journal of Psychiatric Research*, **10**, 181–188.

CROOK, T., RASKIN, A. & DAVIS, D. (1975) Factors associated with attempted suicide among hospitalised depressed patients. *Psychological Medicine*, **5**, 381–388.

DURKHEIM, E. (1951) *Suicide*. Glencoe, Illinois: Free Press.

EASTWOOD, M. R., HENDERSON, A. S. & MONTGOMERY, I. M. (1972) Personality and parasuicide methodological problems. *Medical Journal of Australia*, **1**, 170–175.

EDMUNDS, G. & KENDRICK, D. C. (1980) *The Measurement of Human Aggressiveness*. Chichester: Ellis Horwood.

FARMER, R. (1985) *Hostility and Deliberate Self-Poisoning*, MSc thesis, University of Manchester.

—— (1986) Deliberate self-poisoning. *British Journal of Hospital Medicine*, **36**, 437–442.

—— & CREED, F. (1986) Hostility and deliberate self-poisoning. *British Journal of Medical Psychology*, **59**, 311–316.

FAVA, G. A. (1986) Irritability. (letter) *British Journal of Psychiatry*, **148**, 338–339.

FOULDS, G. A. (1965) *Personality and Personal Illness*. London: Tavistock Publications.

——, CAINE, I. M. & CREASY, M. A. (1960) Aspects of extra- and intro-punitive expression in mental illness. *Journal of Mental Science*, **106**, 599–610.

FRIEDMAN, A. S. (1970) Hostility factors and clinical improvement in depressed patients. *Archives of General Psychiatry*, **23**, 524–537.

FREUD, S. (1917) Mourning and melancholia. In *Collected Papers* Vol IV. New York: Basic Books.

Gottschalk, L. A., Gleser, C. G. & Springer, K. J. (1963) Three hostility scales applicable to verbal samples. *Archives of General Psychiatry*, **9**, 254–279.

Gershon, E. S., Gromer, M. & Klerman, G. L. (1968) Hostility and depression. *Psychiatry*, **3**, 224–235.

Hope, K. (1963) *The Structure of Hostility among Normal and Neurotic Persons*. Unpublished Ph.D. thesis. University of London.

Klerman, G. L. & Gershon, E. S. (1970) Imipramine effects upon hostility in depression. *Journal of Nervous and Mental Disease*, **150**, 127–132.

Kreitman, N. (1978) Social and clinical aspects of suicide and parasuicide (attempted suicide): a contribution from psychiatric epidemiology. In *Companion to Psychiatric Studies* (ed. A. Forrest, J. Affeck & A. Zealley). Edinburgh: Churchill Livingstone.

Lester, D. (1967) Suicide as an aggressive act. *Journal of Psychology*, **66**, 47–50.

—— (1968) Suicide as an aggressive act: a replication with a control for neuroticism. *Journal of General Psychology*, **79**, 83–86.

Mayo, P. R. (1967) Some psychological changes associated with improvement in depression. *British Journal of Social and Clinical Psychology*, **6**, 63–68.

Morgan, H. G., Burns-Cox, C. J., Pocock, H. & Pottle, S. (1975) Deliberate self-harm: clinical and socio-economic characteristics of 368 patients. *British Journal of Psychiatry*, **127**, 564–574.

Newson-Smith, J. G. B. & Hirsch, S. R. (1979) Psychiatric symptoms in self-poisoning patients. *Psychological Medicine*, **9**, 501–507.

Overall, J. E., Hollister, C. E., Johnson, M. & Pennington, J. (1966) Nosology of depression and differential response to drugs. *Journal of the American Medical Association*, **195**, 946–948.

Paykel, E. S. (1971) Classification of depressed patients: a cluster analysis derived grouping. *British Journal of Psychiatry*, **118**, 275–288.

—— & Dienelt, M. N. (1971) Suicide attempts following acute depression. *Journal of Nervous and Mental Disease*, **153**, 234–243.

Philip, A. E. (1968) The constancy of structure of a hostility questionnaire. *British Journal of Social and Clinical Psychology*, **7**, 16–18.

—— (1969) The development and use of the Hostility and Direction of Hostility Questionnaire. *Journal of Psychosomatic Research*, **13**, 283–287.

—— (1970) Traits, attitudes and symptoms in a group of attempted suicides. *British Journal of Psychiatry*, **116**, 475–482.

—— (1971) Psychometric changes associated with response to drug treatment. *British Journal of Social and Clinical Psychology*, **10**, 138–143.

Rado, S. (1928) The problem of melancholia. *International Journal of Psychoanalysis*, **9**, 420–438.

Schless, A. P., Mendela, J., Kipperman, A. & Cochrane, C. (1974) Depression and hostility. *Journal of Nervous and Mental Disease*, **159**, 91–100.

Shneidman, E. S. (1964) Suicide, sleep and death. *Journal of Consulting Psychology*, **28**, 95–106.

Stengel, E. & Cook, N. (1958) *Attempted Suicide*. London: Oxford University Press.

Urwin, P. & Gibbons, J. G. (1979) Psychiatric diagnosis in self-poisoning patients. *Psychological Medicine*, **9**, 501–507.

Vinoda, K. S. (1966) Personality characteristics of attempted suicides. *British Journal of Psychiatry*, **112**, 1143–1150.

Weissman, M. M. (1974) The epidemiology of suicide attempts. *Archives of General Psychiatry*, **30**, 737–746.

——, Klerman, G. L. & Paykel, E. (1971) Clinical evaluation of hostility in depression. *American Journal of Psychiatry*, **128**, 261–266.

——, Fox, K. & Klerman, G. L. (1973) Hostility and depression associated with suicide attempts. *American Journal of Psychiatry*, **130**, 450–455.

Wessman, A. E., Ricks, D. F. & Tyl, M. M. (1960) Characteristics and concomitants of mood fluctuation in college women. *Journal of Abnormal and Social Psychology*, **60**, 117–126.

Yesavage, J. A. (1983) Direct and indirect hostility and self-destructive behaviour by hospitalised depressives. *Acta Psychiatrica Scandinavia*, **68**, 345–350.

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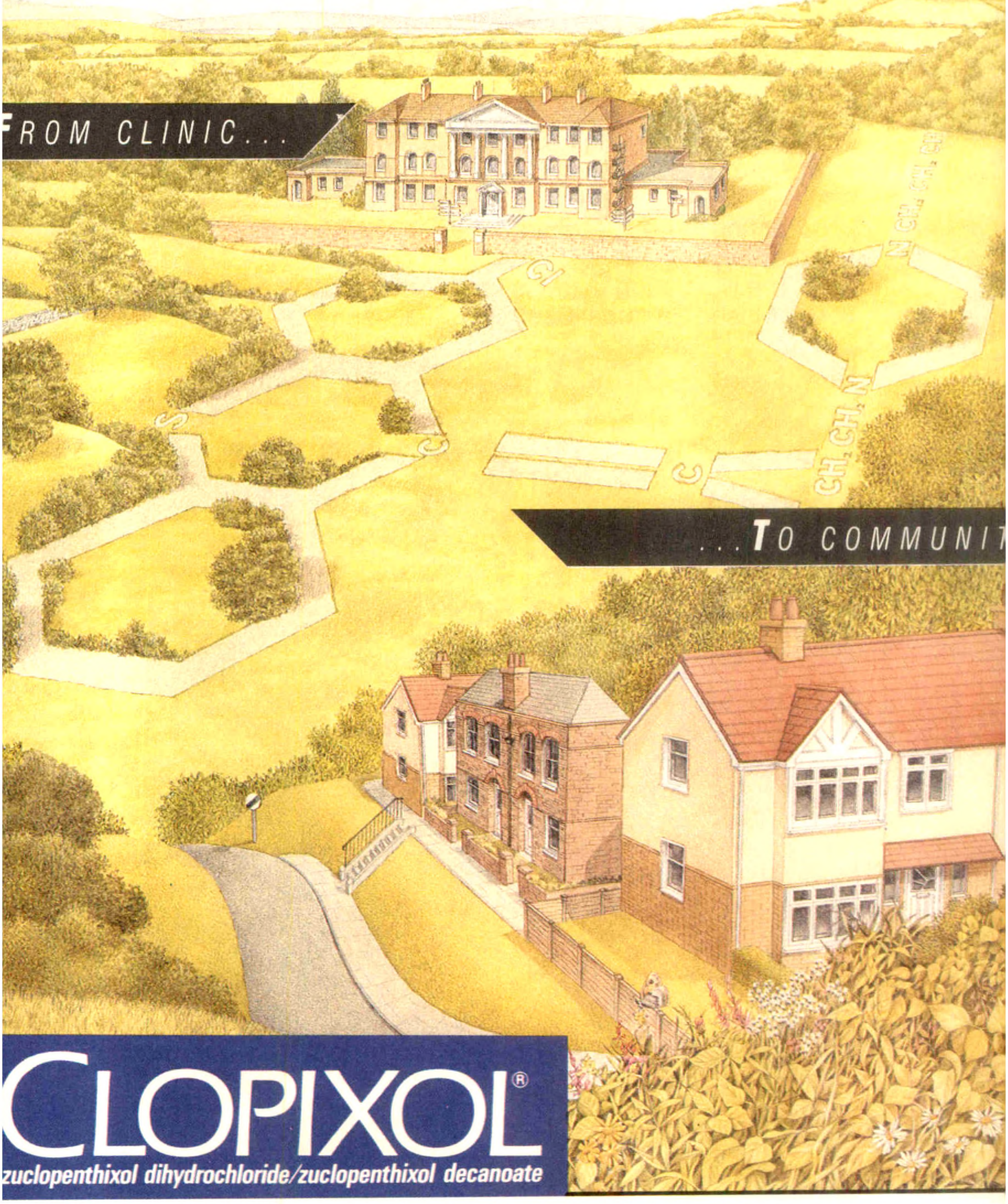

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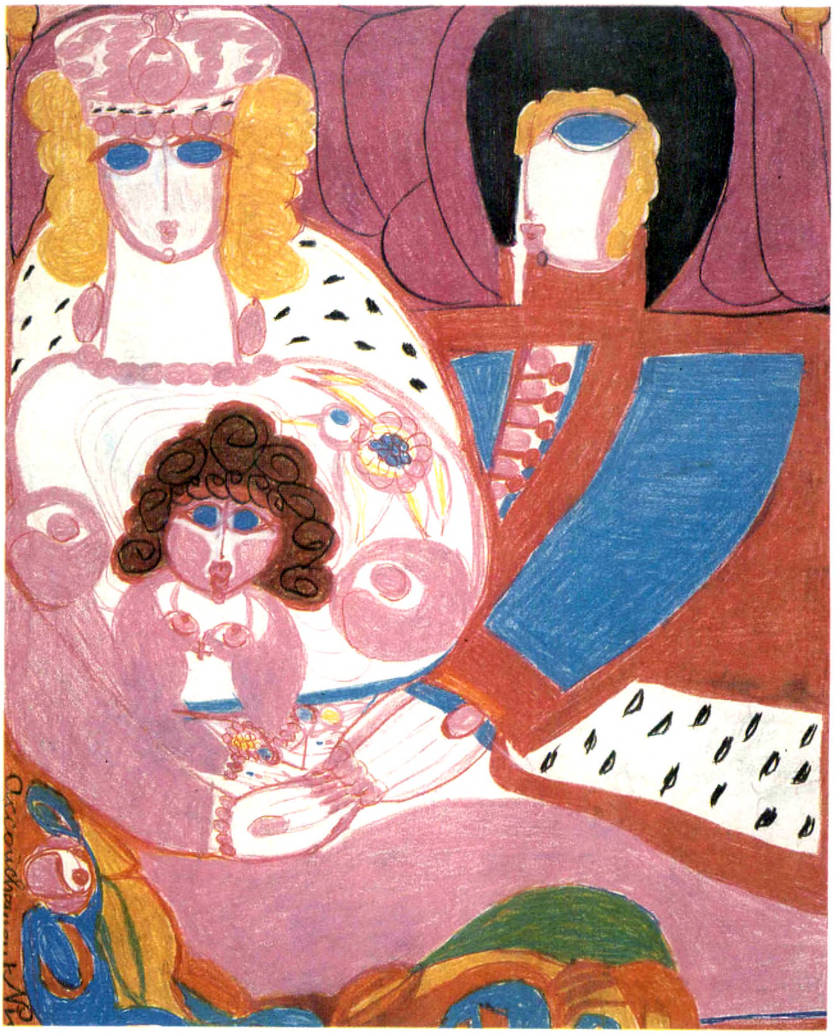

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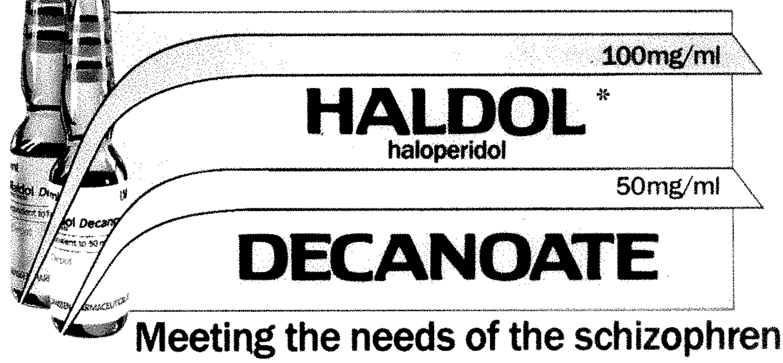
References: 1. Pakes G. E. In 'Therapeutics Today' Ed D. A. W. Johnson. ADIS Press. (1982). 2.
2. Youssef H. A. Acta Therapeutica. (1983) **9**, 215-225



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Depixol Injection and Depixol-Conc. Injection *contain cis(Z)-flupenthixol decanoate, 20mg per ml and 100mg per ml respectively, in thin vegetable oil.*

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Side Effects *Early (frequent) and late (rare) dyskinesias. Extrapyramidal effects sometimes require dose reduction or anti-Parkinson drug treatment.*

Contra-indications *Not recommended for overactive patients, or for those with Parkinsonism, severe arteriosclerosis, senile confusional, or advanced renal, hepatic, or cardiovascular disease, intolerance to other neuroleptics, pregnancy.*

Further Information *Overdosage should be treated, (a) by anticholinergic anti-Parkinson drugs if extrapyramidal symptoms occur, (b) by sedation (with benzodiazepines) in the unlikely event of agitation or excitement, (c) by noradrenaline in saline intravenous drip if the patient is shocked.*

Package Quantities & Basic NHS Prices *Boxes of 10 x 20mg (1ml) £15.53, 10 x 40mg (2ml) £25.99, 10 x 100mg (1ml) ampoules £59.53, 5 x 20mg and 5 x 40mg syringes £8.51 and £13.74, 1 x 200mg (10ml) and 1 x 500mg (5ml) vials £14.74 and £28.27.*

PL Nos 0458/007/0015 PA Nos 115/1/1,2,3,5,6,7,8.

REFERENCES

1. Johnson D A W and Malik N A. Acta Psychiat Scand 1975; 51: 257-267
2. Pinto R, Bannerjee A and Ghosh N. Acta Psychiat Scand 1979; 60: 313-322
3. Hamilton M, Card I R, Wallis C G and Mahmood M R, Psychopharmacol 1979; 64: 225-229
4. Chowdhury M E H and Chacon C. Comp Psychiat 1980; 21: 135-139
5. Haslam M T, Bromham B M and Schiff A A. Acta Psychiat Scand 1975; 51: 92-100
6. Knights A et al. Brit J Psychiat 1979; 135: 515-523
7. Kelly H B, Freeman H L, Banning B and Schiff A A. Int Pharmacopsychiat 1977; 12: 54-64
8. Based on Basic NHS prices at September 1986.

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Female Sex Offenders

ART A. O'CONNOR

Information on 19 women convicted of indecency and 62 women convicted of other sex offences was examined. Those convicted of indecency offences often had poor social skills and had a high incidence of mental illness, mental handicap and alcoholism. Two were convicted of indecent exposure, a rare offence in women. In 39 (63%) of the sex offences with individual victims, the victims were children and in 9 cases the offender was the mother or step-mother. In 25 cases the women were convicted of aiding and abetting a male offender. Of those convicted of indecent assault on persons under 16 and of gross indecency with children, 48% had a previous history of psychiatric disorder.

Sex offences are generally thought of as being committed by men against women or children. However, although rare, sex offences can also be committed by women. According to Home Office Criminal Statistics (Home Office, 1975-1984) 0.95% of all sex offences were committed by women. Table I shows a comparison of male and female sex offences over the 10-year period.

Literature on this topic is very sparse. There are a few case reports of female exhibitionists (Zavitzianos, 1971; Hollender *et al*, 1977; Grob, 1985) but there are no previous systematic studies of female sex offenders. Sarrel & Masters (1982) in their paper concerning 11 male *victims* of female sexual molestation suggest a classification of assaults into: (1) forced assault; (2) baby-sitter abuse; (3) incestuous abuse; and (4) dominant woman abuse. Their paper was mainly concerned with the victims and their subsequent problems.

TABLE I

Convictions for sex offences (excluding bigamy, abduction and prostitution) for the years 1975-1984 in England and Wales. (Source: Home Office Criminal Statistics 1975 to 1984).

	Males	*Females*	*Females as percentage of totals*
Buggery	2067	4	0.19
Attempted buggery[1] and indecent assault on males	6740	112	1.6
Indecent assault on females	23,640	113	0.48
Rape	3462	9	0.26
USI on girl under 13 years	978	6	0.61
USI on girl under 16 years	5223	13	0.25
Incest	1286	40	3.0
Procuration	2217	124	5.6
Indecency with children	2621	41	1.5
Totals	48,234	462	0.95

USI = unlawful sexual intercourse.

1. Offences in this section were classified as 'attempted buggery etc.' until 1977 and then as 'indecent assault on a male'.

Sexual offences against children are causing increasing concern to many agencies. Up to 80% of the victims are female and the commonest offence is unlawful sexual intercourse (Davidson, 1983). The victims are most commonly in the 11-15 year age group (Mzarek *et al*, 1981).

The offenders are often from within the family: 34% are parents or surrogate parents, 2% being mothers (Mzarek *et al*, 1981). The mother may be involved by giving active or passive permission either because it relieves her of sexual activity or because of fear of her husband (Davidson, 1983). The literature on incest is well reviewed by Batten (1983) and aspects of management are reviewed by various authors (Ciba Foundation, 1984).

This paper describes aspects of a group of imprisoned women convicted of sex offences.

Legal background

Crimes of indecency are offences such as indecent behaviour, outraging public decency and indecent exposure. They are dealt with at common law. The act in general has to be lewd, grossly scandalous, outraging public decency or offensive and disgusting. The punishment is a fine or imprisonment or both. There is no limit to the sentence but it must not be inordinate.

Indecent assault is dealt with under the Sexual Offences Act 1956 (Section 14 and 15). An example could be kissing a girl without her consent and suggesting that sexual intercourse take place. Consent to the act is no excuse if the victim, boy or girl, is under the age of 16. The maximum penalty on indictment for indecent assault on a man is 10 years imprisonment. The maximum penalty, if the victim is female and under 13 years of age, is 5 years imprisonment and if she is over 13 years of age the maximum penalty is 2 years imprisonment.

The Indecency with Children Act 1960 is concerned with acts of gross indecency with or towards children under 14 years of age. An example of gross indecency with a child is when a man allows a child to hold his penis and masturbate him. An example of gross indecency towards a child is when a person masturbates in front of a child. The maximum sentence on indictment is 2 years imprisonment.

Rape (Section 1 of the Sexual Offences Act 1956) can only be committed by a man but a woman can be found guilty of aiding and abetting rape or any other sexual offence. By the Accessories and Abettors Act 1861 a person who aids, abets, counsels or procures for the commission of an offence is liable to be tried and punished as the principal offender. The maximum sentence for rape is life imprisonment. Unlawful sexual intercourse with a girl under 13 years and incest with a girl under 13 years also have a maximum sentence of life imprisonment. Unlawful sexual intercourse with a girl under 16 years (S6), sexual intercourse with a defective (S7) and procurement of a defective (S9) (male as principal) all have a maximum sentence of 2 years (Cross & Jones, 1980; The Howard League for Penal Reform, 1985).

Method

Holloway Prison is the main female remand prison for London and South East England. Information was collected on all women committed to Holloway Prison from January 1974 to October 1985, who were convicted of sex offences. They numbered 81 in all. Prison records were examined in all cases and prison medical records in 64 cases. In 17 cases the medical records were missing. Eleven cases were examined personally by the author.

Information was obtained about basic socio-demographic items and also details of the offences and outcome in court. Where possible, information concerning past psychiatric and criminal history was obtained. Psychiatric reports, hospital and probation reports were all consulted where available.

One of the problems of a retrospective study such as this is the incompleteness of case notes. However, it would seem reasonable to document the information available as there is no substantial descriptive study of female sex offenders in the psychiatric literature.

Results

The offenders were divided into three groups according to the legal definition of the offence:

(1) Indecency (19 cases)
(2) Indecent Assault and Indecency with Children (39 cases) and
(3) Unlawful Sexual Intercourse (23 cases).

There were no individual victims in the indecency group but there were in the other two groups.

Indecency

There were 19 cases of indecency: indecent behaviour (14 cases), outraging public decency (three cases) and indecent exposure (two cases). Their average age was 35.9 years. Thirteen were single and six were divorced or separated; nine were of no fixed abode. Five had concurrent convictions for prostitution and four for being drunk and disorderly.

Eight had previous committals to Holloway Prison for similar minor offences such as being drunk and disorderly. Psychiatric diagnoses were made in eight of the group of 19. One had chronic schizophrenia, one had recurrent hypomania, two were mentally handicapped, two were alcoholic and two were dependent on drugs. The remaining 11 had no formal psychiatric diagnosis recorded.

As would be expected, the sentences were very light. Five were sentenced to imprisonment for periods ranging from 5 to 21 days. Five received 1-year conditional discharges, one received a Probation Order, seven were fined sums under £15.00 and one was put on a Hospital Order.

Case 1

M. was a 46-year-old single woman. She was mentally handicapped with an IQ in the range of 46–52. She was an illegitimate child and her mother was a long-term patient in a psychiatric hospital suffering from schizophrenia. M. spent periods in hospitals for the mentally handicapped and also sleeping rough. She had previous convictions for being drunk and disorderly and one conviction for actual bodily harm. The index offence was of outraging public decency by urinating in public. She had taken some alcohol and urinated in the street. She was given a conditional discharge for 1 year. At a later date she was charged with assaulting a policewoman and urinating in public. She was dealt with under Section 37 of the Mental Health Act 1983, and admitted to a hospital for the mentally handicapped.

Case 2

F. was a 25-year-old single chambermaid. She had a history of recurrent hypomanic episodes. She had several hospital admissions for this but also several criminal convictions for offences such as theft (4) and breach of the peace (1). The index offence (outraging public decency) involved exposing her genitals in public and inviting passers by to commit acts of indecency with her. All the offences occurred during hypomanic episodes. These rapidly resolved with treatment while on remand and a Hospital Order was never required. She was fined £5.00 for the index offence.

Case 3

H. was a 21-year-old single woman. She was second in a family of five. All her family were hard working and achieving people and there was no family history of psychiatric illness or criminal behaviour. She was borderline mentally handicapped but was able to read and write adequately and take care of herself. She had few friends but when aged 18 she got to know a group of similar unemployed people. Partly to impress them and gain their admiration she started episodes of stripping in public places. This behaviour continued until it became a frequent occurrence. It was often accompanied by masturbating in public, and occurred particularly when she was under stress. Self-exposure and masturbation often occurred when she had taken some alcohol because, while she enjoyed the activity, she found it embarrassing to start. She had several admissions to a psychiatric hospital for overdoses and short-lived depressions. She served four short prison sentences for theft and threatening behaviour. Although she was brought before the courts on many occasions for her indecent exposure she was never imprisoned for these.

Indecent assault and indecency with children

There were 39 cases in this group and details of the offences are given in Table II. Their average age was 28 years. Eleven of the 39 were married at the time of the offence, nine separated or divorced, two widowed, 15 were single and the marital status of two was unknown. There were only two people of no fixed abode.

There was little information available about the past histories of the 14 in the indecent assault group. However, in the next two categories 48% of the women had a psychiatric diagnosis and a history of previous treatment. Nineteen cases involved indecent assault on persons under 16. Eight of these had previous psychiatric treatment, five for depression, one for schizophrenia and one for personality disorder. The remaining one had been taking flupenthixol for 3 years but the diagnosis is unclear. Of the six in the indecency-with-children group, four had a history of inpatient psychiatric treatment: one for schizophrenia, one for schizo-affective psychosis and two for personality disorder.

The outcome of the court cases is shown in Table III. The more severe sentences were given for offences involving victims under 16 years. Hospital Orders were used in three of the six cases of indecency with children (two schizophrenia, one subnormality) and in only one case of indecent assault on a person under 16 (schizophrenia and subnormality).

TABLE II
Offences of indecent assault and indecency with children

Indecent assault	
indecent assault	9
aiding and abetting indecent assault	1
indecent assault and actual bodily harm	4
	14
Indecent assault on a person under 16 years	
indecent assault on a person under 16 years	12
indecent assault on a person under 16 years and indecency with a child	6
encouraging indecent assault on a person under 16	1
	19
Indecency with children	
indecency with a child	5
attempted gross indecency with a child	1
	6

Information about victims in the 14 indecent assault cases was sparse. One was a 17-year-old girl and the defendant aided and abetted her boyfriend in the assault. One was a 16-year-old girl and the 15-year-old co-defendant claimed it was a joke. Three of the offenders were jointly charged with two boys of offences against a 16-year-old girl (Cases 8, 9 and 10).

Of the 19 cases involving indecent assault on persons under 16 years of age information about the victims was obtained for 14 cases. In all there were eight female and eight male victims. (In one case there were two female victims and in another a male and a female victim.) Some of the motives given for the behaviour were: boredom after a broken marriage complicated by sedative and alcohol abuse (two cases); sex with a 13-year-old boy as revenge against her husband for being unfaithful; punishment of a 13-year-old boy for breaking into her house by taking his trousers off and interfering with him, (aided and abetted by her husband); fear of being beaten by her boyfriend if she did not aid and abet him in indecently assaulting a 9-year-old boy. In one case the mother of the victim was involved, and in one, the step-mother.

In the six cases involving indecency with children two involved boy victims and four involved girls. In two cases the perpetrator was the mother of the children.

TABLE III
Outcome of offences of indecent assault and indecency with children

	Imprisonment (years)				*Suspended Sentence, Conditional Discharge, or fine*	*Probation Order Sup. Order*	*Hospital order*	*Totals*
	≤1	≤2	≤3	≤4				
Indecent assault	6	1			5	2		14
Indecent assault on person under 16	8	2	1	1	4	2	1	19
Indecency with children	1	1				1	3	6
								39

Case 4

L. was a 36-year-old woman of West Indian origin. She was separated from her husband and her four children. The index offence was of attempted gross indecency with a child under 14 years of age. She made improper suggestions to a four-year-old boy within earshot of his parents. She had a past history of schizophrenia for which she had several admissions to hospital. At the time of the index offence she had several bizarre delusions and one of them was that the child was her own. She had a previous charge of child stealing and at the time of that offence she also thought the child was her own. She was dealt with under Section 37 of the Mental Health Act.

Case 5

J. was a 40-year-old separated woman of West Indian origin. On three consecutive days she was arrested for outraging public decency. She called at peoples' houses and asked to use the telephone. When admitted to the house she took all her clothes off and asked to have sex with the man of the house. On the fourth day she exposed her breasts in public and held them close to a young boy who was passing with his mother. She was charged with outraging public decency and indecency to a child. She had a long history of recurrent hypomanic episodes and seemed obviously mentally ill to all concerned. However, she was committed to Crown Court for trial but was regarded as unfit to plead. The charges were withdrawn because the court was reassured that she would get psychiatric treatment under Section 3 of the Mental Health Act 1983. This was achieved by the co-operation of the Crown Court and the area psychiatrist.

Case 6

C. was a 23-year-old woman separated from her husband. Her marriage had been a very unhappy one because of her husband's alcoholism and his violence, including sexual violence, towards her. There was one child, a 3-year-old girl, from the marriage who lived with C. and her common-law husband. This relationship was a loving and caring one and they intended getting married. They had sex on several occasions while the 3-year-old was present and once the man allowed the child to hold his penis. He was convicted of gross indecency towards the child and she of aiding and abetting him. She was sentenced to 2 years imprisonment and he to 3 years imprisonment. She naively felt that she was educating the child by exposing her to loving sex rather than the violent variety she had experienced before. No psychiatric abnormality was found in this woman by the doctors who saw her initially nor by the author who interviewed her while she was serving her sentence.

Case 7

M. was a 23-year-old divorced woman. Her marriage had been very unhappy because of her husband's drinking, beatings and sexual violence towards her. She drank heavily and abused sedatives towards the end of her marriage. They had three children and they lived with her ex-husband. She never knew her real father and there was a history of sexual abuse by her step-father over several years in her early teens. She had several previous convictions for theft. For approximately 1 year before the index offence she had been living with a man and he was planning to end the relationship. He had an 8-year-old daughter who lived with them. M. did housework for a 60-year-old man whose wife was an invalid. She had an affair with him and says that he threatened to tell her common-law husband about it if she did not agree to the sexual assaults on the 8-year-old step-daughter. The offence involved M., the 60-year-old man and the child engaging in various sexual acts and taking photographs. They were both convicted of gross indecency with the child. He was sentenced to 6 months imprisonment and she to 2 years 6 months as it was thought she was the instigator.

Cases 8, 9 and 10

In this case there were three female and two male co-defendants. The first two girls were aged 16 and 17 years and had a history of being in care and previous minor convictions. Both had a disrupted background with paternal alcoholism. The third girl was aged 17 and had no previous criminal background. The boys were aged 14 and 15 years and had known the girls for several months. The first two girls had been in a hostel but moved into a flat with the third girl some months previously. The victim was 16 and was known to the three girls as she stayed in the hostel. She absconded from the hostel several days prior to the offence and the girls were hiding her in their flat. On the afternoon in question the three girls, the victim and the two boys were in the flat. An argument broke out and the victim started teasing the boys, daring them to have sex with her. Everyone became angry towards the victim and took her clothes off. She was held down by the girls while the boys repeatedly rammed a candlestick up her vagina. She subsequently needed treatment for vaginal injuries. The five were convicted of causing actual bodily harm and indecent assault. They were each sentenced to four months Youth Custody. No psychiatric diagnosis was made on any of these three young women by the doctors who saw them initially nor by the author who interviewed them during the course of their sentences.

Unlawful sexual intercourse

There were 23 cases in this group and some details, including the outcomes, are given in Table IV. In all but two of the 23 cases the woman acted as aider and abettor of a man committing a sexual offence. The average age of the offenders was 33 years. Thirteen were married, six were divorced or separated, three single and the marital status of one was unknown. Fourteen had no previous psychiatric contact but there was no information concerning past history on seven. One woman was treated as an out-patient for depression and one had several admissions for immature personality disorder.

In 14 of the 23 cases the victims were under 16 years of age. In four cases the mother was involved in the crime, and in one, the step-mother. In one case both parents were involved and the boy and girl victims were both under 13

TABLE IV
Details of cases in the unlawful sexual intercourse (USI) group

Offences	*No. of cases*	*Age*	*Outcome*
USI	2	19, 19	3 months, Probation × 3 years
AA USI	2	42, 35	3 months, 9 months
AA rape	1	20	1 year
AA USI – victim under 16 years	8	Average 34 years	5 years, 4½ years, 2 years, 1 year (2) 3 months, suspended sentence (2)
AA USI – victim under 13 years	2	59, 29	1 year, 4 years
AA rape – victim under 16 years	2	32, 32	5 years, 4 years
AA buggery and abduction	1	21	6 months
AA incest	2	43, 36	4 years, 18 months
Unlawful administering of drug to AA USI victim under 16 years	1	39	18 months
Procure defective and AA USI	2	31, 25	9 months, 15 months
	23		

AA = aid and abet. Periods of time in the outcome column refer to sentences of imprisonment.

years of age. Some example of reasons given for the behaviour were: prostitution of a child; another woman claimed she was persuaded by her 14-year-old daughter to allow her to have sex to get money; another aided and abetted rape to punish her 14-year-old daughter for being naughty and disobedient.

Case 11

P. was a 51-year-old separated woman. She had a very unhappy marriage because of frequent violent attacks on her by her husband. They separated 10 years prior to the offence and he subsequently became a chronic psychiatric patient. She had six grown-up children and a 13-year-old daughter. For several years prior to the offence she was living with a 65-year-old man. Her 14-year-old daughter knew the man wanted to have sex with her and tried to persuade her mother to agree to this over several weeks. She said they could get money for a holiday that way. P. claimed that she agreed one evening when she was intoxicated. The girl told a visiting social worker and they were arrested. He was convicted of unlawful sexual intercourse and she of aiding and abetting him. She was sentenced to 12 months imprisonment and he to 6 months because of his age.

Discussion

Of those convicted of gross indecency with children and indecent assault on persons under 16, 48% had a psychiatric diagnosis and a history of psychiatric treatment. Little information was available on those convicted of indecent assault and one wonders if these also had a similar high incidence of psychiatric disorder. In the unlawful sexual intercourse group only two were recorded as having had previous psychiatric contact. Only one of this group was examined by the author and she was alcoholic. It is difficult to accept the low rate of psychiatric contact that the case notes suggest in this group.

The first group of offences described were those of indecency. They were minor offences, there was no individual victim and the offences were against accepted standards of social behaviour. Most of the offenders were women with poor coping skills, often of low intelligence and frequently having associated alcohol or psychiatric problems. A large proportion were homeless people.

Two women were convicted of indecent exposure. Of the 21 215 cases of indecent exposure recorded in Home Office Criminal Statistics between 1975 and 1984 all were male. The two women in this study were charged under Section 28 Town Police Clauses Act 1847 and Section 5 Public Order Act 1936 respectively, offences which may not be recorded centrally by the Criminal Records Office. Exhibitionism has generally been thought of as an exclusively male perversion. In men psychodynamic formulations suggest that castration anxiety is relieved by exposing the penis and eliciting a response from a female victim. It is suggested that because females have no penis to lose they have no significant castration anxiety and so have no need to expose their sex organs. Hollender *et al* (1977) describe a case of female genital exhibitionism and they suggest that while male exhibitionism results from castration anxiety, in the female exhibitionism usually stops short of genital exposure and results from the need for attention. Rarely, as in their case, genital exposure may occur in women as a pregenital attention seeking manoeuvre. They comment that males usually desire an aversive response and that the activity is genital (phallic) in psychoanalytic terms.

Zavitzianos (1971) describes a case of female fetishism and exhibitionism and suggests that there is a close relationship between perversion and some psychopathic personalities. Grob (1985) describes a case of a woman whose emotional deprivation led to feelings of rejection and the inability to develop empathic relationships. Her self-esteem relied on success in her profession, and when she lost her position she sought validation through exposing her genitalia while driving alongside large trucks on the highway. The intense need for attention in this case was stressed. Case 3 in the present study has many features of exhibitionism and the need for attention, especially when under stress, was prominent. The other case of indecent exposure was a woman with a history of many convictions for minor crimes and also alcohol abuse. Her offences could not be regarded as sexually motivated.

In this study 39 (63%) of the sex offences with victims involved children and in nine of these the woman was the mother or step-mother. Of interest also is the fact that in all but two of the 23 cases in the unlawful sexual intercourse group the women were convicted of aiding and abetting a male committing the offence. Sexual gratification was never noted as a motivation for the women involved in sex offences with a victim.

Conclusions

Sex offences are rare in females. Offences of indecency are relatively minor offences committed by women of poor social skills and with a high incidence of mental illness, mental handicap and alcoholism. While indecent exposure is a relatively common offence in men, only two women were convicted of this offence in the present study. Women who commit sex offences that have individual victims are a more heterogeneous group and of interest is the number of offences that involve children (63%) and also mothers or step-mothers (nine cases). Of those women convicted of indecent assault on persons under 16 and gross indecency with children, 48% had a past history of psychiatric disorder. Nearly all the women in the unlawful sexual intercourse group were convicted of aiding and abetting a male rather than being the instigator themselves.

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References

Batten, D. A. (1983) Incest—a review of the literature. *Medicine, Science and the Law*, 23, 245–253.

Ciba Foundation (1984) *Sexual Abuse within the Family*. London: Tavistock.

Cross & Jones (1980) *Introduction to Criminal Law*, 9th ed. London: Butterworth.

Davidson, A. T. (1983) Sexual exploitation of children: a call to action. *Journal of the American Medical Association*, 75, 925–927.

Grob, C. S. (1985) Female exhibitionism. *Journal of Nervous and Mental Disease*, 173, 253–256.

Hollender, M. H., Brown, C. W. & Roback, H. B. (1977) Genital exhibitionism in women. *American Journal of Psychiatry*, 134, 436–438.

Home Office *Home Office Criminal Statistics 1975–1984*. London: HMSO.

Howard League For Penal Reform (1985) *Unlawful sex*. London: Waterlow.

Mrazek, P. B., Lynch, M. & Bentovim, A. (1981) In *Sexually Abused Children and Their Families*, (eds P. B. Mrazek & C. H. Kempe). Oxford: Pergamon Press.

Sarrel, P. M. & Masters, W. H. (1982) Sexual molestation of men by women. *Archives of Sexual Behaviour*, II, 117–131.

Zavitzianos, G. (1971) Fetishism and exhibitionism in the female and their relationship to psychopathy and kleptomania. *International Journal of Psychoanalysis*, 52, 297–305.

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No Fixed Abode

A Comparison of Men and Women Admitted to an East London Psychiatric Hospital

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There is little information available on psychiatric disorder in homeless women. This study compares clinical casenote information of all homeless women admitted to an East London psychiatric hospital from 1971–1980 inclusive, with information on a cohort of homeless men admitted during the same period. The results show that the women appear to have been socially more stable than the men prior to admission and yet they have less satisfactory admissions to hospital. Possible reasons for these apparent inconsistencies are discussed.

The relationship between homelessness and psychiatric morbidity has been studied by many people. Although there have always been people who have adopted a wandering lifestyle by choice, some of whom have written about their experiences (Davies, 1908; Orwell, 1933), others, less fortunate, have adopted a nomadic lifestyle as a result of illness. Ødegard (1932) studied the link between emigration and insanity and found that Norwegians in Minnesota were more likely to be suffering from schizophrenia than those in Norway and some of those who had emigrated were ill before they did so. The relationship between high admission rates for schizophrenia and populations living in impoverished inner city areas has been elucidated by Faris & Dunham (1939) in Chicago, Gerard & Houston (1953) in Worcester, Hare (1956*a*,*b*) in Bristol and Dunham (1965) in Detroit.

Research in the UK has been mainly centred on people living in common lodging houses or hostels, people living rough or using soup kitchens and people with no permanent address who are in prison or hospital. This last group are often referred to as of 'no fixed abode' (NFA) for the purposes of record-keeping. Operational definitions of homelessness are often of limited value as the boundaries are unclear. A survey carried out in 1965 of 'homeless single persons' (National Assistance Board, 1966) demonstrated the overlap between the populations of various establishments with those who slept rough. In general, the lower the standard of accommodation, from lodging house, to hostel, to reception centre, to the street, the greater is the proportion of physically and mentally disabled people found.

Surveys carried out in Salvation Army hostels (e.g. Crossley & Denmark, 1969; Lodge Patch, 1970) have generally concentrated on the assessment of men. However, Priest (1971) interviewed men and women in common lodging houses in Edinburgh and found that the residents were typically men in their 50s or 60s, who had resided in lodging houses for several years. There was a preponderance of Catholics from Scotland and Ireland and half the residents were unemployed at the time of interview. Psychiatric morbidity was diagnosed in 58 (78%) of the residents and the most common diagnosis was schizophrenia, in 25 residents (32%). Alcoholism was diagnosed in 14 residents (18%) and personality disorders in a further 14 (18%). Priest (1976) then compared the characteristics of men and women in Edinburgh lodging houses with patients from similar accommodation who were referred to a psychiatric hospital. Both populations were predominantly male, with approximately 18% of women. He showed that the patients presenting to hospital were likely to be younger and have a history of law-breaking, and were more likely to be diagnosed as alcoholic or personality-disordered. The lodging house residents not presenting to hospital were more likely to be older and suffering from schizophrenia. However, these studies did not attempt to compare diagnostic and social data of men and women.

A retrospective survey of patients admitted from NFA to a Birmingham mental hospital from 1961–1965 was made by Berry & Orwin (1966). The figures for 105 men and 40 women were combined and, in all, 49% of patients were diagnosed as suffering from schizophrenia and 28% were diagnosed as having a personality disorder. Some 90% of the patients were unemployed, 75% had been homeless for one year and 37% had a criminal record; 74% of patients had been previously admitted to a psychiatric hospital.

Reception centres were set up by the National Assistance Board after its inception in 1948 and were designed to give temporary shelter to 'persons without a settled way of living'. Surveys by Edwards *et al* (1968) and Tidmarsh and Wood (1972) demonstrated the high degree of social and psychiatric morbidity among the residents of the Camberwell Reception Centre. However, the demand on services there were such that nearly two-thirds of the mentally ill, three-quarters of the alcoholics and half of the physically handicapped new cases did not receive medical treatment at the centre and most were not referred elsewhere (Wood, 1976).

The studies dealing specifically with homeless or destitute women are few, and to date there has been little work delineating the characteristics of destitute women who are admitted to psychiatric hospitals. Higgs & Hayward (1910) described the generally poor working conditions and low wages which employed women endured. Low wages or an unwanted pregnancy could force women into common lodging houses or workhouses, where they lived with prostitutes and the mentally ill. In 'casual wards' for vagrants they were subjected to a dehumanizing regime, and in 1905, 9% of vagrants thus 'relieved' were women. Between the wars there were adequate numbers of lodging house places for women (Menzies, 1927), in which charges were higher than in similar institutions for men and the institutions were generally dreary. It was said that the women were more destructive than the men, were likely to steal the sheets and spent longer indoors, thereby increasing staffing and heating costs. Laidlaw (1956) surveyed women and men in Glasgow lodging houses and found that the women residents were more likely than the men to be or have been married and fewer of the women had been in prison. Few residents of either sex were employed and about 25% of all residents were mentally ill or mentally handicapped.

Digby (1976) in a nation-wide survey of hostels and lodging houses estimated that approximately 2200 women were living in such settings compared with 23 300 men. He found that the women were socially more stable than the men on several criteria including maintaining contact with relatives and holding a job. In contrast, a study of the residents of a hostel for homeless women (Brandon, 1973) describes markedly disturbed behaviour by the residents when the community opened, with violent acting out, overdoses and slashed wrists. Some social and psychiatric characteristics of destitute men and women in Dublin have been delineated by Fernandez (1984) in a survey designed to assess the needs of the homeless mentally ill, but there is a general lack of information about NFA women (Herzberg, 1984).

It is apparent that people of no fixed abode do not form a homogeneous group and rehabilitation strategies will depend on the availability of resources allocated to medical and social services. The situation is further complicated by a general lack of low-cost accommodation. Digby (1976) showed that there was a 17% loss of beds in hostels and lodging houses between 1965 and 1972. Furthermore, the number of reception centres throughout the UK had fallen from 270 in 1948 to 17 in 1970. However, moves have been made to establish smaller reception centres with the aim of providing a better assessment of the needs of referred clients. Leach & Wing (1980) found that these smaller centres were considered more acceptable by both staff and clients. Although the Housing (Homeless Persons) Act 1977 does state that priority for rehousing should be given to the mentally ill, homeless families generally take precedence over the single homeless.

There is evidence that destitute alcoholics can be helped by hospital rehabilitation programmes (Myerson & Mayer, 1966) or through contact made in 'shop fronts' leading on to placement in long term hostel accommodation (Cook, 1975). A flexible attitude to service provision is necessary when dealing with the needs of the destitute. Leach & Wing (1978) and Leach (1979) found that the effectiveness of the St Mungo's service for helping destitute men was improved by the use of 'action research' techniques where research findings were used to modify the original project. They found that the men who were suitable for placement in a rehabilitation scheme were assessed more effectively from a night shelter than a soup run. Similar flexibility in approach was shown to be helpful by Brandon (1973), who found that violence in a hostel for destitute women was reduced by the introduction of encounter groups and relaxation therapy.

The provision of rehabilitation facilities for mentally ill patients in the community is often inadequate, and official estimates of the number of hostel places required have been too low (Christie Brown *et al*, 1977). There is currently much worry about the move of patients from long stay hospital care into a community with a widespread lack of after-care facilities. There is anecdotal evidence that in the USA, where a programme of 'deinstitutionalisation' is well under way, the night shelters are filling with the mentally ill homeless (Bassuk, 1984).

Leach & Wing (1978) describe an 'enabling model' of rehabilitation for the destitute, with contact services on the street, leading on to chains of housing and vocational opportunities at closely spaced levels, with the necessity to provide facilities for those who cannot move upwards from any one level. They state that this policy would require adequate assessment of the projected need for facilities, proper funding and close co-operation between government, local authority and voluntary agencies.

Method

The study was based at the London Hospital (St Clement's), the psychiatric unit of an East London teaching hospital, which offers a catchment area service to Whitechapel, Stepney, Bethnel Green, Poplar and the Isle of Dogs. This is an impoverished Inner London catchment area which at the time of the 1981 census had the highest proportion of unemployed men of any of the Inner London boroughs (Office of Population Censuses and Surveys, 1982). The area contains a large number of hostels and lodging houses for homeless people.

Various studies of the homeless have used different definitions of homelessness. For example, the National Assistance Board (1966) survey included persons sleeping rough, and those using reception centres, lodging houses, shelters and hostels, together with the people seeking financial aid from the Board's offices. The present survey took the form of a retrospective casenote study. The entry of a patient into this study was initially determined by the patient being recorded as homeless on admission and therefore noted as NFA in hospital admission books kept in the medical records department. Thus the type of accommodation previously used did not determine initial entry into this study and neither did the length of time for which the patient had been homeless. However, attempts were made to note the duration of homelessness and previous use of hostels and reception centres in the subjects who were finally included in the study.

When the casenotes were studied in detail it sometimes transpired that patients recorded as NFA in the admission books did in fact have an abode to which they could return on discharge, but this information had not been available at the time of admission. It was decided to study only those patients who were homeless at the time of admission and who had no home to return to on discharge. Furthermore, it was apparent when studying the casenotes that some NFA patients had been transferred to hospital from prison, but otherwise met the criteria for entry into the study (i.e. they were homeless offenders who subsequently were thought to require hospital treatment). These patients, 10 men and 3 women, were included in the study and allowance was made in the analysis of the results where their admission from prison was likely to have affected the data.

Inspection of the admission books showed that between 1971 and 1980 inclusive, 79 NFA women had been admitted. Exclusion of cases not meeting the above criteria gave a final sample of 50 women.

Inspection of the admission books showed that 272 NFA men had been admitted in the same period. A subsample of every third admission during this period was taken to provide a sample of men of approximately equal size to the sample of women. The subsample of men chosen (90) was similar to those not chosen (182) in respect of age ($P<0.4$), number of previous admissions to the index hospital ($P<0.95$) and whether the current admission was on section ($P<0.3$). Exclusion of cases from the subsample not meeting the entry criteria gave a final sample of 63 men.

A schedule was drawn up to record the wide range of information found in the medical and nursing notes. For convenience, information is presented on the whole group of 113 subjects where there is no appreciable difference between the sexes for a specific item. It is acknowledged that information obtained from casenotes retrospectively may be unreliable in detail and that prospective studies are likely to yield the most accurate results. However, retrospective casenote studies of the hospitalised homeless have yielded valuable information in this field (e.g. Whitely, 1955; Berry & Orwin 1966). Information about individual items was not obtained for every subject, and percentages, where stated, refer to the percentages of valid cases (i.e. those cases where the information was available for a given item). Where there are significant differences between the sexes these are delineated and have been subject to non-parametric statistical analysis using a χ^2 test. For items where two by two tables were obtained, a Yates correction was applied. Only one item on the schedule, namely, age of subjects, was analysed by parametric statistics using a *t*-test. Statistical computations were performed using the Statistical Package for the Social Sciences (Nie *et al*, 1973).

Results

Social data

The mean age of all subjects was 38.6 years (s.d. 12.2). The mean age of the women, 36.3 years, was less than that of the men, 40.5 years. This result tended towards, but did not reach, statistical significance ($P<0.07$).

Thirty-eight men (61.3%) were single, compared with 18 women (36.7%). Only 24 men (38.7%) were currently or previously married, compared with 31 (63.3%) of the women. The women were significantly more likely to be married than the men ($P=0.02$). Thirty-two women (72.7%) had one or more children compared with 19 men (34.5%); significantly more women than men had children ($P<0.005$).

Virtually half of all the subjects, 53 (48.6%) were born in England, 17 (15.6%) were born in Scotland and 13 (11.9%) in Eire. Five subjects (4.6%) came from other parts of Ireland. Only 22 subjects originated from London. There were as many subjects registered Catholic, 42 (44.7%) as Church of England, 42 (44.7%).

Forty-five subjects (49.5%) had experienced a separation of greater than 3 years from one or both parents before the age of 15, and further information was available in 41 cases. Of these, in 17 (41.5%) the separation was due to the death of a parent and in 18 (43.9%) it was due to the separation or divorce of the parents. Only in two cases did the separation appear to be attributable to delinquent behaviour by the child. In general, the subjects came from large families with a mean of 3.5 siblings (s.d. 3.1).

The subjects left school at an early mean age, 15.3 years (s.d. 1.9), and left home at a mean age of 18.1 years (s.d. 3.7). It was noted that 21 of the men left home to join the armed services or the merchant navy and the records suggest that four of these men were subsequently dishonourably discharged. Eight women left home to get married or live with a boyfriend and four left for residential jobs as cleaners or nannies. Five women left home to attempt training as student or pupil nurses and another two women subsequently attempted such training. Further information available on six of these trainee nurses suggested that only one actually completed her training.

Nearly two-thirds of all subjects, 60 (64.5%), had been in London for more than 1 year, and relatively few, 12 (12.9%), had arrived in London within a week of their admission to hospital. Twenty-eight subjects (45.2%) had been in accommodation within a week of their admission to hospital and a further 19 subjects (30.6%) had been in accommodation within a month of their admission. Fifty-six subjects had previously used hostels and four subjects, all men, had used facilities for destitute alcoholics. Only one subject was recorded as having used a reception centre, but the index hospital was situated relatively far from reception centres. Only 37 subjects (32.7%) had definitely been living rough prior to admission and 24 of these had lived rough for less than one week. Places used included streets, parks, under bridges, shop doorways and a bombed site.

Fourteen men (29.8%) were in contact with their families compared with 18 women (64.3%). Contact was loosely defined to include any known personal contact or contact by telephone or letter within the previous 12 months. Women were significantly more likely to be in contact with their families than men ($P<0.008$).

Only two subjects were currently in employment at the time of admission. The mean time of unemployment for subjects was 25.6 months (s.d. 41.0). It was noted that 22 men had done casual labouring or building site work and 5 had been kitchen porters. Three men had been barmen, all of whom were diagnosed as alcoholic at the time of admission. Only nine men held a skilled or semi-skilled job when they last worked. Few jobs were coded for women. Seven women had worked in factories, three had done domestic work and three had worked in laundries. The notes suggested that nine of the women had earned money by prostitution.

The mean number of known admissions to other psychiatric hospitals was 2.5 per subject (s.d. 2.9). One subject had at least 20 previous admissions and only in 19 subjects was there no history of previous admission. In addition, 28 subjects (24.8%) had previously been admitted to the hospital under study.

Forty-one subjects (53.9%) had a history of imprisonment and 13 of these subjects (ten men and three women) were transferred to hospital from prison on the index admission. When all 113 subjects were considered, the men had a higher mean number of terms of imprisonment (1.5) than the women (0.6), and women were significantly less likely to have a history of imprisonment than men ($P<0.04$). However, when the data were reanalysed excluding the subjects transferred from prison, this result tended towards, but failed to reach, statistical significance ($P<0.1$).

Referral to hospital

In the following section only data on those subjects admitted to hospital from the community (53 men and 47 women) will be considered. Forty subjects (40.0%) were referred by doctors, who were generally casualty staff, 25 (25.0%) were self referrals and 28 (28.0%) were referred by the police. Overall, seven men (13.2%) were referred by the police compared with 21 women (44.7%). The women were significantly more likely to be referred to hospital by the police than the men ($P<0.001$). It was also apparent that Section 136 was being used more frequently by the police for women than men, although this result could not be subject to statistical analysis due to low cell frequencies in the χ^2 table. However, 16 women (34.0%) were admitted under Section 136 compared with 6 men (11.3%). In addition, women admitted from the community were significantly more likely than men to present via the emergency clinic rather than organised channels of referral ($P<0.02$).

The reasons for admission under Section 136 are noteworthy. Six subjects were walking or running dangerously through traffic five women and one man) and four subjects (all women) were admitted following assaults on people or property. Three subjects (two women and one man) were admitted having been found in varying degrees of nudity in a public place and three other subjects (one woman and two men) had been talking to themselves. Two subjects (both women) had been found on a railway track and two others (one woman and one man) had been fire-setting. One subject (woman) told the police that she was in America and demanded that they take her to India, and another subject (man) was found cutting his wrist. Seven out of the 16 women admitted under Section 136 (43.8%) were subsequently diagnosed as schizophrenic and a similar proportion of the men, three out of six (50.0%) of those admitted under section 136, were also diagnosed as schizophrenic. Two thirds of the non-schizophrenic men and women admitted under Section 136 subsequently received a diagnosis of personality disorder. The patients' diagnoses did not correlate with the behaviour precipitating their referral by the police. Violent, potentially self-destructive and bizarre behaviours seemed equally prevalent among the schizophrenics as the personality-disordered.

Admissions

The mean length of admission for all subjects was 30.7 days, with a large standard deviation (s.d. 35.8). Seven subjects had a stay of 24 hours or less and the maximum length of stay was 170 days by one subject.

Symptom/sign combinations were coded for each subject but were not analysed due to low cell frequencies. A summary of these findings is given in Table I.

Seventeen subjects had physical illnesses needing treatment (Table II) but only two subjects, both women, were transferred to a general hospital. One subject had a previously undiagnosed carcinoma of the breast and the other had a pyrexia of unknown origin which did not require treatment. Four of the 63 men (6.4%) suffered from epilepsy, a condition with a high psychiatric morbidity.

TABLE I
Symptoms and signs by sex[1]

	Men	*Women*
Depression	29 (50.0%)	19 (41.3%)
Suicidal ideation	10 (17.2%)	9 (19.6%)
Delusions/hallucinations	16 (27.6%)	19 (41.3%)

1. Valid cases: 58 men, 46 women.

A diagnosis had been coded in the casenotes of 110 subjects and was recorded for this study, but the casenote diagnosis was not subject to formal validation by standardised measures. Where more than one diagnosis was coded, the primary diagnosis was used. Thirty-six subjects (32.7%) were diagnosed as schizophrenic, 26 (23.6%) as alcoholic and a further 32 subjects (29.1%) were diagnosed as personality-disordered. Relatively few subjects, nine (8.2%), received a primary diagnosis of depression. Of the 13 subjects transferred to hospital from prison, seven (53.9%) were schizophrenic. A breakdown of diagnostic data by sex is given in Table III.

TABLE II
Diagnosis by sex[1]

	Men	*Woman*
Schizophrenia	16 (25.8%)	20 (41.7%)
Depression	3 (4.8%)	6 (12.5%)
Mania	0 (0.0%)	1 (2.1%)
Alcoholism	22 (35.5%)	4 (8.4%)
Personality disorder	16 (25.8%)	16 (33.3%)
Organic	2 (3.2%)	0 (0.0%)
Other	3 (4.8%)	1 (2.1%)

1. Valid cases: 62 men, 48 women

Excluding the use of benzodiazepines for night sedation, 82 subjects (72.6%) were treated with drugs and eight (7.1%) received ECT. The rest received milieu therapy with or without a benzodiazepine night sedative. Of the eight subjects (four women and four men) who received ECT, five had a primary diagnosis of depression and two others (one alcoholic, one personality-disordered) had attempted suicide prior to admission and were suicidal on admission. The other subject had a paranoid psychosis which was not diagnosed as schizophrenia.

Nineteen women (54.3%) were noted in medical or nursing notes as co-operating poorly with treatment, compared with 19 men (31.7%). Women were significantly more likely to co-operate poorly with treatment than men ($P = 0.05$). Furthermore, 23 women (46.0%) took their own discharge, compared with 13 men (21.0%); women were significantly more likely to discharge themselves from hospital than men ($P < 0.02$).

Sixty-three subjects (36.8%) were considered to have an improved mental state on discharge, while 48 subjects (43.2%) had not shown improvement. Follow-up was arranged for 35 subjects, of whom 17 subsequently attended.

In general, the hospital-based social workers attached to each consultant firm were involved in facilitating the after-care arrangements for the subjects who had a planned discharge. During the period covered by this study there was only one small psychiatric after-care hostel in the vicinity of the hospital under study and there were no group homes nearby. Of necessity, most subjects would be referred to voluntary agencies such as the Salvation Army for accommodation after discharge.

TABLE III
Physical illnesses present in the subjects

Sex	*Illness*	*Number of subjects*
Male	Epilepsy	4
	Tuberculosis	2
	Ischaemic heart disease/ chronic bronchitis	1
	Alcoholic gastritis	1
	Deep vein thrombosis	1
	Hypertension	1
Female	Carcinoma breast	1
	Pyrexia of unknown origin	1
	Epilepsy	1
	Buttock abscess	1
	Vaginal discharge/pelvic inflammatory disease	1
	Oedematous legs (? cause)	1
	Diarrhoea	1

Discussion

Despite the limitations imposed by undertaking a retrospective survey from casenotes, the results of this study correlate well with other studies of the destitute. The typical male subject in this study is aged 40, and single with no children. A significant proportion of the subjects are of Irish or Scottish origin. The fact that just under a half of all subjects were separated from their parents in childhood is similar to findings by Edwards *et al* (1966) and Priest (1976). The finding that nearly half the subjects had been in accommodation within a week of admission and only a third had been living rough, substantiates the clinical impression that NFA subjects often present to emergency hospital services when their accommodation has been lost. This is not to say that all homeless people behave in this way, as there is little doubt that many, including those who are ill, avoid all helping agencies completely.

Edwards *et al* (1966) noted that skid row alcoholics have often been in the merchant services and in this study 21 men had been in the armed or merchant services, although the contribution of conscription to this figure cannot be evaluated from the data. Of more interest is the finding that seven women had attempted nurse training. It may be that this group, having experienced emotionally impoverished childhoods, sought a sheltered, hierarchically determined environment similar to that provided by the armed or merchant services for men. Presumably all but one of the women were unable to cope with the emotional and intellectual demands made on them during the training.

Many subjects in this study had a history of previous periods in hospital and/or prison; these findings were similar to those of Berry & Orwin (1966). The use of Section 136 to admit many of the subjects, particularly women, is noteworthy. Szmukler *et al* (1981) reviewed all compulsory admissions in a London borough and found that NFA patients were over-represented among those on sections. They found that Section 136 patients were younger than those on other sections, often male and with a short episode of disturbance prior to admission. These patients often had brief admissions, and presented a nursing problem. They were also likely to abscond, a finding previously described by Sims & Symonds (1975). There are suggestions in the literature that NFA personality-disordered patients often know how to secure an admission (Berry & Orwin, 1966), and this may be particularly true of Section 136 patients (Szmukler, 1981). The finding that most subjects in this study were admitted through the emergency clinic is not a surprising one. Lim (1983) notes in a survey of the Maudsley Hospital emergency clinic that use of emergency services are often the only way that NFA patients can get help.

The actual determinants of the admissions for all patients were not specifically elicited in this study, but a third of all subjects were psychotic (i.e. deluded or hallucinated) and by definition were suffering from severe mental illness. The patients who were admitted on Section 136 had generally shown marked behavioural disturbance prior to admission. Patients fell into three main diagnostic categories, namely, schizophrenia, personality disorder and alcoholism, a feature in common with all other studies in this field.

When comparing the NFA men and women, the findings of this study appear paradoxical. The women appeared socially more stable than the men prior to admission, a finding similar to that of Laidlaw (1956) and of Digby (1976). Women were more likely to have been married, to have had children and to have remained in contact with their families. Furthermore, proportionally fewer women had been in prison than men. However, at the time of admission, women were more likely than men to have been referred by the police on a Section 136, and they co-operated less well with treatment and were more likely to discharge themselves. The comparison of diagnoses of the men and women (Table III) shows that proportionally more women were schizophrenic than men, and that more men were alcoholic, but this does not appear to provide an answer for all these differences. Social and attitudinal explanations should also be considered.

It is possible that people dealing with destitute women are prepared to tolerate a greater degree of behavioural disturbance than they would from a similar group of men. This could explain why the women were more likely to have remained in contact with their families and were less likely to have been in prison. However, this does not explain why significantly more women were referred to hospital by the police and why more use was made of Section 136 in the case of the women, as the men and women admitted on this section seemed equally disturbed, and similar percentages of the men and women admitted on this section were schizophrenic.

It is interesting to question whether the police perceive similar disturbed behaviour by men and women differently, or indeed handle it differently. The study was not designed to answer this question and there certainly seems little difference between the men and women admitted under Section 136. It is possible, however, that the police are more likely to perceive a woman behaving strangely as 'sick' and initiate a hospital disposal in circumstances where a man is likely to be charged and remanded to prison. Indeed, 10 out of 63 men (15.9%) in this study were transferred to hospital from prison, whereas three out of 50 women (6.0%) were similarly transferred. It is therefore possible to suggest that the women who become NFA and get admitted to hospital are a more disturbed group than NFA men who get admitted to hospital, for two reasons. Firstly, in this study, proportionally more women than men were schizophrenic and secondly, many disturbed men may have ended up in prison. These suggestions could explain why the women's admissions appear to have been less satisfactory than those of the men.

The availability of money for rehabilitation facilities is obviously linked to the economic health of the country as well as to the prevailing political climate. The deteriorating economic climate of the 1970s seems not to have affected the numbers of NFA admissions to the index hospital during the period under study. NFA admissions were in double figures from 1971 to 1975 inclusive, but only reached double figures in one subsequent year under study. It is sad to note the dearth of group home and small hostel accommodation during the 1970s, in an Inner London teaching district which has been a traditional haven for many destitute men and women. Unfortunately this situation has not changed in the 1980s.

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References

Bassuk, E. L. (1984) The homelessness problem. *Scientific American*, **251**, 28–33.

Berry, C. & Orwin, A. (1966) No fixed abode: a survey of mental hospital admissions. *British Journal of Psychiatry*, **112**, 1019–1025.

Brandon, D. (1973) Community for homeless women. *Social Work Today*, **4**, 167–170.

Christie Brown, J. R. W., Ebringer, L. & Freedman, L. S. (1977) A survey of a long stay psychiatric population: implications for community services. *Psychological Medicine*, **7**, 113–126.

Cook, T. (1975) *Vagrant Alcoholics*. London: Routledge & Kegan Paul.

Crossley, B. & Denmark, J. C. (1969) Community care: a study of the psychiatric morbidity of a Salvation Army hostel. *British Journal of Sociology*, **20**, 443–449.

Davies, W. H. (1908) *The Autobiography of a Supertramp*. London: Jonathan Cape. (Republished Oxford: Oxford University Press, 1980).

Digby, P. W. (1976) *Hostels and Lodgings for Single People*. Office of Censuses and Surveys. London: HMSO.

Dunham, H. W. (1965) *Community and Schizophrenia*. Detroit: Wayne State University Press.

Edwards, G., Hawker, A., Williamson, V. & Hensman, C. (1966) London's skid row. *Lancet*, **1**, 249–252.

——, Williamson, V., Hawker, A., Hensman, C. & Postoyan, S. (1968) Census of a reception centre. *British Journal of Psychiatry*, **114**, 1031–1039.

Faris, R. E. & Dunham, H. W. (1939) *Mental Disorders in Urban Areas: An Ecological Study of Schizophrenia and Other Psychoses*. Chicago: University of Chicago Press.

Fernandez, J. (1984) "In Dublin's fair city": the mentally ill of 'no fixed abode'. *Bulletin of the Royal College of Psychiatry*, **8**, 187–190.

Gerard, D. L. & Houston, L. G. (1953) Family setting and the social ecology of schizophrenia. *Psychiatric Quarterly*, **27**, 90–101.

Hare, E. H. (1956*a*) Mental illness and social conditions in Britain. *Journal of Mental Science*, **102**, 349–357.

—— (1956*b*) Family setting and the urban distribution of schizophrenia. *Journal of Mental Science*, **102**, 753–760.

Herzberg, J. L. (1984) No fixed abode. *British Journal of Hospital Medicine*, **32**, 24–26.

Higgs, M. & Hayward, E. E. (1910) *Where Shall She Live? The Homelessness of the Woman Worker*. London: P. S. King and Son.

Laidlaw, S. I. A. (1956) *Glasgow Common Lodging Houses and People Living in Them*. Glasgow: Corporation of Glasgow.

Leach, J. (1979) The evaluation of a voluntary organisation attempting to resettle destitute men: action research with the St Mungo Community Trust. In *Vagrancy: Some New Perspectives* (ed. T. Cook). London: Academic Press.

—— & Wing, J. K. (1978) The effectiveness of a service for helping destitute men. *British Journal of Psychiatry*, **113**, 481–492.

—— & —— (1980) *Helping Destitute Men*. London: Tavistock Publications.

Lim, M. H. (1983) A psychiatric emergency clinic: a study of attendances over six months. *British Journal of Psychiatry*, **143**, 460–466.

Lodge Patch, I. C. (1970) Homeless men: a London survey. *Proceedings of the Royal Society of Medicine*, **63**, 437–441.

Menzies, F. N. K. (1927) *Common Lodging Houses and Kindred Institutions*. London: London County Council.

Myerson, D. J. & Mayer, J. (1966) Origins, treatment and destiny of skid row alcoholic men. *New England Journal of Medicine*, **275**, 419–425.

National Assistance Board (1966) *Homeless Single Persons*. London: Her Majesty's Stationery Office.

Nie, N. H., Hull, C. H., Jenkins, J. G., Steinbrenner, K. & Brent, D. H. (1973) *Statistical Package for the Social Sciences* (2nd edn). New York: McGraw Hill.

Ødegaard, Ø. (1932) Emigration and insanity: a study of mental disease among the Norwegian born population of Minnesota. *Acta Psychiatrica et Neurologica*, suppl. 4.

Office Of Population Cenuses And Surveys (1982) *County Monitor: Inner London*. London: Government Statistical Service.

Orwell, G. (1933) *Down and Out in Paris and London*. Gollancz. (Now published Harmondsworth: Penguin Books.)

Priest, R. G. (1971) The Edinburgh homeless. *American Journal of Psychotherapy*, **25**, 194–213.

Priest, R. G. (1976) The homeless person and the psychiatric services: an Edinburgh survey. *British Journal of Psychiatry*, **128**, 128–136.

Sims, A. C. P. & Symonds, R. L. (1975) Psychiatric referrals to the police. *British Journal of Psychiatry*, **127**, 171–178.

Szmukler, G. I. (1981) Compulsory admissions in a London borough: 2—Circumstances surrounding admission: service implications. *Psychological Medicine*, **11**, 825–838.

——, Bird, A. S. & Button, E. J. (1981) Compulsory admissions in a London borough: 1—Social and clinical features and a follow up. *Psychological Medicine*, **11**, 617–636.

Tidmarsh, D. & Wood, S. (1972) Psychiatric aspects of destitution. In *Evaluating a Community Psychiatric Service* (ed. J. K. Wing and A. M. Hailey). Oxford: Oxford University Press.

Whiteley, J. S. (1955) Down and out in London: mental illness in lower social groups. *Lancet*, **2**, 608–610.

Wood, M. (1976) Camberwell Reception Centre: a consideration of the need for health and social services of homeless single men. *Journal of Social Policy*, **5**, 389–399.

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Tics and Heredity
A Study of the Relatives of Child Tiqueurs

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The limited literature on the pedigrees of tiqueurs, including those with Gilles de la Tourette's syndrome, is reviewed. Most statistical analyses have been restricted to affected family members without specifying the unaffected ones. The present statistical analysis of a series of child tiqueurs, including 91 probands and 1293 first- and second-degree relatives, 46 of whom were tiqueurs, predicts the odds on being a tiqueur for individuals, and establishes how those odds are affected by certain explanatory variables using log-linear models. The data do not confirm a familial pattern beyond reasonable doubt, but if the suggested prevalence of tics in the population is 10% then the figure for parents is large enough to support a familial hypothesis. The pedigrees do not indicate a simple mode of genetic transmission. Further research is needed to confirm that there is a connection between childhood tics and Gilles de la Tourette's syndrome, to establish that the predisposition to tics is familial, and, if so, whether there is a complex genetic mechanism involved, or some other environmental aetiology so far undisclosed.

Throughout the literature on tics and Gilles de la Tourette's syndrome (henceforth referred to as Tourette's syndrome) there are innumerable case-reports containing family histories of members with tics, but few large-scale investigations of the pedigrees of child tiqueuers have been published (Mahler *et al*, 1945—34 cases; Zausmer, 1954—96 cases; Torup, 1962—220 cases; Corbett *et al*, 1969—180 cases). The modest literature on the familial nature of Tourette's syndrome and multiple tics is summarised by Kidd *et al* (1980). There are even fewer genetic studies and these relate to patients with Tourette's syndrome (Shapiro *et al*, 1978; Friedhoff & Chase, 1982). It is evident that the debate regarding the inheritance of tics continues. Shapiro *et al* (1972) investigated 34 patients with Tourette's syndrome and found no relationship between this disorder and a family history of tics; they concluded that genetic factors were not involved in the aetiology. This view was later modified (Shapiro *et al*, 1978) and they recorded that the syndrome occurs with increased frequency in relatives of the patients but that there is no discernible pattern of transmission. Moldofsky *et al* (1974) studied 15 patients with Tourette's syndrome and found nine who had at least one first-degree relative with tics; although no specific sex-linked dominant forms of inheritance were seen, a recessive mode of genetic influence was suggested.

A valuable contribution is that of Kidd *et al* (1980), who collected data on Tourette's syndrome and tics for relatives of 75 patients with the syndrome as part of a pilot questionnaire survey. The frequencies of Tourette's syndrome and/or tics among relatives were significantly heterogeneous in a familial pattern. This suggests (a) that the disorder is transmitted, with tics alone being a milder manifestation, and that this severity difference is a threshold phenomenon related to transmission, (b) that the sex difference in prevalence is real because it was found among relatives of both male and female probands, and (c) that the sex difference is related to transmission as a threshold effect since female probands, although less common than male probands, had a higher proportion of affected relatives. These pilot data provided evidence for the transmission of Tourette's syndrome and can be qualitatively explained by a genetic model of transmission. Kondo & Nomura (1982) agreed that these conclusions are fundamentally compatible with their own; however, in an investigation of 43 patients (41 Japanese and 2 Koreans) with Tourette's syndrome they found that among a total of 428 known relatives only nine (2%) showed evidence of Tourette's syndrome or chronic multiple tics. Kidd & Pauls (1982) discussed the genetic hypotheses for Tourette's syndrome and chronic multiple motor tics. They analysed a combined sample containing 421 first-degree relatives of 118 probands using three different models, and concluded that such models can explain the complex transmission pattern observed in families of patients with the syndrome, but that discrimination between the quite different models for the inheritance of a complex trait is extremely difficult if the only information available is the trait phenotype.

Other recent genetic research includes the study of HLA typing and red blood cell choline levels in Tourette's syndrome (Comings *et al*, 1982*a*,*b*). Reporting the former investigation, the authors state that the available data are compatible with a two-locus hypothesis, with a major gene for Tourette's syndrome and a modifying gene which decreases the threshold for penetrance. However, their findings from a preliminary study of HLA A and B antigens in 12 patients with Tourette's syndrome and their family members do not point to a predominant HLA A or B type associated with the disease. In the latter investigation, to determine whether red blood cell choline could be used as a marker for Tourette's syndrome, 16 patients and their relatives were studied. The findings confirmed an earlier observation of raised levels in Tourette's syndrome, which seem to be genetically controlled. The authors conclude that although the elevated levels do not appear to be a direct marker for Tourette's syndrome, they may be a marker for genetic or environmental influences that serve to enhance the expression of the major gene(s) for the syndrome.

There is therefore a diversity of hypotheses to explain the familial predisposition to these disorders and a paucity of data to support a genetic theory. In view of this we carried out further statistical analysis of the first author's research findings relating exclusively to child tiqueurs (Zausmer, 1954), both for its historical interest and because of the hypothesised connection between chronic multiple motor tics and Tourette's syndrome (Kidd & Pauls, 1982).

Historical background

During the period 1952–1954 the first author studied 96 child tiqueurs (63 boys and 33 girls) as a research project at the University of Liverpool. The sample is described elsewhere (Zausmer, 1954). In 32% of the families there were relatives who had had tics. There were no recorded members exhibiting features of Tourette's syndrome. The figure of 32% tallies with that of Torup (1962), who found a family history of tics in 30–40% of close relatives, but not with that of Corbett *et al* (1969) who noted it in only 10% of cases. Interestingly, Shapiro *et al* (1978) found that the percentage of family members with tics in their sample of Tourette patients was 34.5.

In 1965 the first author had the good fortune to interest Dr C. A. Clarke (at that time Reader in Medicine at the University of Liverpool) in the analysis of the pedigrees of the research sample of tiqueurs to determine if it was possible to explain the familial transmission of tics on a genetic basis. Clarke summarised his findings as follows (5 of the 96 pedigrees were omitted from the analysis because of insufficient background information). (a) There was a highly significant sex difference (87 males as against 50 females). (b) The impression of family size was that it did not differ significantly from that of the general Liverpool population. (c) Most of the tiqueurs were not the first or last born in the families (see also Table V). (d) The mean ages of the parents (where the ages of both were known) at the birth of the propositus were fathers 31.3 (s.d. 5.7) and mothers 28.3 (s.d. 5.1) years, which was probably typical for the population.

Clarke concluded that no simple genetic system could explain the pattern of cases, assuming they were a homogeneous group. (If they were clinically heterogeneous, the numbers in each class would be too small for statistical analysis). The nearest approach to simplicity would be to invoke a single dominant gene with incomplete penetrance, but the lack of penetrance would be so great as to make it valueless from a practical point of view (56 out of 91 tiqueurs had no family history of tics). Clarke then considered the possibility of a single dominant gene plus a multifactorial background. One might expect that where the sex of the tiqueur was the unusual one (that is, female) there would be more likelihood of her parents (mother) or offspring being affected than would be the case in a male tiqueur. There was no evidence that this was so. Thus, where the propositus was a female there was a positive family history in 11 cases and a negative one in 18 cases. Where the propositus was a male there was a positive family history in 24 cases and a negative one in 38 cases.

This was not statistically significant and our more recent analysis described below confirms this finding. (For this analysis illegitimate births, adoptions and incomplete family histories were omitted.)

Method

Wider availability of more advanced statistical techniques since 1965 has meant that we are now able to explore the pedigree data in more detail. As mentioned above there are 91 families with sufficient detail for us to be able to include them in the analysis: 62 of these index cases were boys and 29 girls. We have at least some data on 1533 relatives of these tiqueurs: 178 siblings, 36 half-siblings, 178 parents, 342 grandparents, and 799 siblings of the parents. We have used the following potential explanatory variables: subject's own sex, sex of the index case, age (classified into five groups — 0–4, 5–9, 10–14, 15–19 and 20+ years), relationship to the index case (sibling, half-sibling, parent, grandparent, parental sibling), and parental type (whether or not at least one parent of the tiqueur was affected). Examination of the raw data revealed that some of our 1533 cases had missing values on so many of these variables that including them in the analysis would have been impractical, and the results that follow are based on 1293 cases with almost complete data: 178 siblings, 22 half-siblings, 176 parents, 342 grandparents, and 575 parental siblings. As can be seen, the parental siblings are the group for which data are most often missing on the main explanatory variables. Among the 1293 relatives in our main sample there were 46 who were tiqueurs, 25 males and 21 females.

TABLE I

Proportion of tiqueurs classified by sex and degree of relationship to index case

	Sex of index case					
	Male		*Female*		*Total*	
Index cases	62		29		91	
	Sex of relative					
	Male		*Female*			*Proportion of tiqueurs*
	Observed	*Expected*	*Observed*	*Expected*	*Total*	
Sibling						
Tiqueurs	8	5.0	1	4.0	9	0.05
Total	93	—	85	—	178	—
Half-sibling						
Tiqueurs	0	0.0	0	0.0	0	0.00
Total	9	—	13	—	22	—
Parent						
Tiqueurs	11	13.4	14	11.6	25	0.14
Total	88	—	88	—	176	—
Grandparent						
Tiqueurs	2	3.8	5	3.2	7	0.02
Total	171	—	171	—	342	—
Parental sibling						
Tiqueurs	4	2.8	1	2.2	5	0.01
Total	299	—	276	—	575	—
All relatives						
Tiqueurs	25	—	21	—	46	0.04
Total	660	—	633	—	1293	—

Expected frequencies calculated from the model with sex and degree of relationship included

In Table I we present the raw frequencies broken down by degree of relationship and sex.

Clarke's preliminary analysis (reported above) followed the practice of much of the literature on tics by examining the data in terms of number of families affected. As Wilson *et al* (1978) point out, the fact that a certain proportion of families have at least one affected member tells us relatively little unless we know the size of families concerned. We have therefore analysed the family data using the information on individuals; our prevalence figures may thus be compared with population values directly. We have concentrated on predicting the odds on being a tiqueur for individuals, and analysing how those odds are affected by the explanatory variables. We have used log–linear models (Bishop *et al*, 1975) to examine the effects of the explanatory variables on the odds. The main advantage of this more complex analysis is that we are examining all the possible explanatory variables simultaneously. This means that when we claim that a variable is an important predictor of the odds on being affected by a tic, we are examining the effect of that variable over and above the effect of the other explanatory variables in our model, and we are also able to examine interactions between explanatory variables.

Two of the variables give rise to problems in fitting, parental type and age. Both of these variables have a strong but artefactual association with degree of relationship. In the case of parental type it makes no sense to use this variable in the analysis of the parent data, but we have examined it for the other sets. We wished to include age as an explanatory variable, not because of its scientific interest, but to deal with the problem that younger relatives would not have passed completely through the period of risk and so would show a lower prevalence of tics than a group which was beyond a critical age (hence the choice of age strata). For this reason we only considered age as an explanatory variable for the sibling data. In order to deal with the problem of these two variables we conducted four separate preliminary analyses looking at the different degrees of relationship separately, rather than performing one large analysis. In each of these we fitted a model and then iteratively removed terms from the model, starting from the highest order interactions. Only non-significant terms were deleted, and all lower order terms forming a significant interaction were retained. This is essentially the method outlined by Brown (1976). When it became clear in these analyses that neither parental type nor age would need to be included in the final model, the motivation for the separate analyses disappeared, and we then combined all the data and proceeded with a single analysis using the same approach.

The use of log–linear models makes our method comparable with the methods used by Kidd *et al* (1980). We have used the algorithms of Haberman (1973) and Lustbader & Stodola (1981) to perform the fitting.

Results

The preliminary analyses revealed that age and higher order interactions involving age were not significant even for the

TABLE II

Proportion of tiqueurs among siblings classified by age

Age	*Tiqueurs*	*Non-tiqueurs*	*Proportion*
0–4	1	49	0.02
5–9	3	43	0.07
10–14	3	38	0.08
15–19	0	19	0.00
20+	2	19	0.11
All	9	168	0.05

(one sibling omitted—missing age)

siblings. This can perhaps be attributed to the small number of tiqueurs in each age group. There is in fact a deficiency of tiqueurs in the younger groups (Table II) but this fails to reach significance in any of our analyses.

Parental type

Parental type and higher order interactions involving parental type also failed to reach significance in the analyses of siblings, grandparents and parental siblings combined. The results are presented in Table III.

TABLE III

Proportion of tiqueurs classified by parental type

Degree of relationship	*Parent of child affected*	*Neither parent of child affected*
Sibling		
Tiqueurs	4	5
Total	36	142
Half-sibling		
Tiqueurs	0	0
Total	7	13
Grandparent		
Tiqueurs	3	4
Total	90	250
Parental sibling		
Tiqueurs	0	5
Total	119	454

Sex and degree of relationship

Sex of the proband and higher order interactions involving sex of proband failed to reach significance in any of the analyses conducted. The final simplest model which provides a good fit to the data is one in which the probability of developing a tic is seen as dependent on degree of relationship to the index case and possibly on an interaction between degree of relationship and sex of the relative. The dominant effect is that of degree of relationship (χ^2 test for including degree of relationship in the model $= 54.45$; d.f. 4, $P < 0.001$). There are more tiqueurs among the parents of the index cases, and also among the siblings. There is some suggestion in the data of an interaction between sex and degree of relationship (χ^2 test for including the interaction when degree of relationship and sex are already in the model $= 9.05$; d.f. 4, $0.05 < P < 0.1$). From inspection of the expected values calculated from the model with degree of relationship and sex included it is clear that the main cause of this apparent interaction is the different sex ratio for siblings. Just fitting sex and the component of the interaction due to siblings was significant ($\chi^2 = 6.15$, d.f. 2, $P < 0.05$). Our own feeling is that this interaction will probably not turn out to be a substantive finding, but for completeness we present the χ^3 values and the odds in Table IV.

Odds on being a tiqueur for first- and second-degree relatives

Table IV contains a summary of the two final log–linear models which we have considered. The results are in the form of the odds on being a tiqueur (the probability of being a tiqueur divided by the probability of not being a tiqueur). The table gives the odds for each degree of relationship, and also shows the relative odds (relative to siblings) for the other categories. These can then be interpreted as the increase in relative risk for each degree of relationship. So, for example, we see that the odds for parents are more than three times greater than for siblings. (The odds are conditional on the index case having been identified in the family; they cannot be interpreted as estimates of population risk.)

Birth order

In addition to the other variables mentioned we have some information on the birth order of some of the relatives. Many of the cases have missing data on this variable, but we do have usable information on a subset of the data. Table V shows the relationship between birth order and being affected by a tic for the 418 individuals for whom we have sufficient data. Adding this variable to the log-linear model reveals that there is no relationship between birth order and being affected by a tic, and there is no interaction between birth order and the other variables.

Some characteristics of the pedigrees

Direct transmission through three generations was reported in three families. In one case the male proband had an affected mother and maternal grandmother; in the second case the male proband had an affected mother and maternal grandfather; and in the third case the male proband had an affected father and paternal grandfather.

Of the 28 probands with an affected first-degree relative, 11 indicated at least one other relative who was affected. Within the 20 male probands there were eight instances of the disease being transmitted from an affected male to another male, and the affected relatives were both male and female.

TABLE IV

Odds estimated from log-linear models using degree of relationship and degree of relationship and sex

	Odds on being a tiqueur for a given degree of relationship: likelihood ratio $\chi^2=9.33$, d.f. 9	
Degree of relationship	*Odds*	*Odds relative to sibs*
Sibling	0.053	—
Parent	0.165	3.108
Grandparent	0.021	0.392
Parental	0.009	0.165

Degree of relationship	*Odds calculated from model with degree of relationship and sex and the sibling by sex component of the interaction fitted $\chi^2=3.18$, d.f. 3*				
	Male odds			*Female odds*	
	Odds	*Odds relative to sibs*	*Multiplier for females*	*Odds*	*Odds relative to sibs*
Sibling	0.094	—	0.126	0.012	—
Parent	0.150	1.592	1.211	0.023	1.928
Grandparent	0.019	0.200	1.211	0.003	0.243
Parental sibling	0.008	0.085	1.211	0.001	0.103

TABLE V

Proportion of tiqueurs classified by birth order

Birth order	*Non-tiqueurs*	*Tiqueurs*	*Total*
First	91	10	101
Second	91	8	99
Third	77	5	82
Fourth	54	1	55
Fifth or later	73	8	81
Total	386	32	418

There were three cases of tics in both sides of the family.

Among 91 probands there were 21 with one parent affected and two with both parents affected. Only three of the probands with an affected parent had affected siblings. Of the affected siblings, five cases came from a family in which neither parent was a tiqueur.

Discussion

The reliable identification of tiqueurs among relatives of patients is fraught with difficulties. The majority of children's tics disappear before puberty and, as in the case of enuresis, the symptom is frequently forgotten or suppressed when the history is recorded many years later. In spite of careful history-taking it is likely that many relatives who had had tics in childhood were missed. The total familial incidence was probably much higher than the figures obtained.

In the absence of trustworthy estimates of the prevalence of tics in the population it is difficult for us to justify calculating coefficients of hereditability from our data, and indeed we cannot demonstrate beyond reasonable doubt that the data show a familial pattern. If we assume that the prevalence in the population is 10%, as Wilson *et al* (1978) estimate, then the figure for parents is large enough to support the suggestion that tics are familial in origin.

Our results do not appear to indicate any straightforward genetic hypothesis. If the transmission were a simple recessive one we would have expected that the frequency of occurrence of tics would have been higher for siblings than for parents, whereas we found the reverse, even allowing for the fact that the siblings had not yet passed completely through the period of risk.

If there were two sorts of tics, hereditary and environmental, it would seem plausible that we would find differences between the families when we compared members of families where at least one parent of the child was also affected with members of the other families. In fact we found no such differences.

If transmission were multifactorial, with different thresholds for males and females, we would expect that probands of the more rarely affected sex would have more affected relatives. Our current analysis has shown that the relatives (male or female) of the girls are no more likely to be affected, thus confirming for individuals Clarke's original analysis of families. It thus seems unlikely that a single dominant gene with a multifactorial transmission can account for the results.

TABLE VI

Comparison of the numbers of relatives affected in the present study with those in the Tourette syndrome families studied by Shapiro et al (1978)

	Shapiro et al study						*Present study*					
	Total		*Male*		*Female*		*Total*		*Male*		*Female*	
	n	*Ratio*[1]	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*	*n*	*Ratio*
Total number of patients	139	—	—	—	—	—	91	—	—	—	—	—
Patients without Family history of tics	91	65.5	—	—	—	—	56	61.5	—	—	—	—
Patients with Family history of tics	48	34.5	—	—	—	—	35	38.5	—	—	—	—
Family members with tics												
Parents	33	23.7	15	10.8	18	12.9	25	27.5	11	12.1	14	15.4
Grandparents	3	2.2	3	2.1	0	0.0	7	7.7	2	2.2	5	5.5
Siblings (full and half)	8	5.8	6	4.3	2	1.4	9	9.9	8	8.8	1	1.1
Parental siblings	10	7.2	6	4.3	4	2.9	5	5.5	4	4.4	1	1.1
Total relatives	54	—	30	—	24	—	46	—	25	—	21	—

[1] Ratio of number of affected relatives to number of index cases.

The figures do not sum in columns because some patients had more than one affected relative

If there were a dominant single gene transmission (even with a reduced penetrance) we would expect to find many pedigrees with direct transmission through three generations. In fact we found only three instances in our sample. The occurrence of male parents of male probands effectively rules out the possibility of the inheritance of tics as an X-linked characteristic.

In Table VI we present a comparison of our findings with those of Shapiro *et al* (1978). They studied the relatives of 139 Tourette syndrome patients and present their results in terms of number of families with affected relatives. We have already argued that this is not the method of presentation of choice, but for comparative purposes we present our own data in the same format. Exact comparison is difficult without information on the number of relatives not affected in each category, but it is clear that the two sets of figures show good agreement. As is the case with our study, Shapiro and his colleagues found a substantial sex difference among patients, but not among relatives.

A comparison of the significant findings of the present study with those of Kidd *et al* (1980), whose sample included relatives of adults affected by Tourette's syndrome, indicates marked differences. They found own sex, sex of index case and parental type to be significant but we did not. Our own study showed that degree of relationship was significant, and that there was a possible interaction between sex and degree of relationships, but Kidd *et al* did not examine these variables. We have no convincing explanation for the resemblance between our findings and those of Shapiro *et al*, or for their joint dissimilarity from those of Kidd *et al*, other than that there are of course considerable differences between the populations, sampling methods and statistical techniques employed.

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References

BISHOP, Y. M. M., FIENBERG, S. E. & HOLLAND, P. W. (1975) *Discrete Multivariate Analysis*. Cambridge, Ma: MIT Press.

BROWN, M. B. (1976) Screening effects in multidimensional contingency tables. *Applied Statistics*, **25**, 37–46.

COMINGS, D. E., GURSEY, B. T., HECHT, T. & BLUME, K. (1982*a*) HLA typing in Tourette Syndrome. In *Advances in Neurology*, Vol. 35. New York: Raven Press.

——, ——, AVELINO, E., KOPP, U. & HANIN, I. (1982*b*) Red blood cell choline in Tourette Syndrome. In *Advances in Neurology*, Vol. 35. New York: Raven Press.

CORBETT, J. A., MATHEWS, A. M., CONNELL, P. H. & SHAPIRO, D. A. (1969) Tics and Gilles de la Tourette's Syndrome: a follow-up study and critical review. *British Journal of Psychiatry*, **115**, 1229–1241.

FRIEDHOFF, A. J. & CHASE, T. N. (1982) Gilles de la Tourette Syndrome. In *Advances in Neurology*, Vol. 35. New York: Raven Press.

HABERMAN, S. J. (1973) AS51 Log-linear fit for contingency tables. *Applied Statistics*, **22**, 218–225.

KIDD, K. K. & PAULS, D. L. (1982) Genetic hypotheses for Tourette Syndrome. In *Advances in Neurology*, Vol. 35. New York: Raven Press.

——, PRUSOFF, B. A. & COHEN, D. J. (1980) Familial pattern of Gilles de la Tourette Syndrome. *Archives of General Psychiatry*, **37**, 1336–1339.

KONDO, K. & NOMURA, Y. (1982) Tourette Syndrome in Japan: etiologic considerations based on associated factors and familial clusterting. In *Advances in Neurology*, Vol. 35. New York: Raven Press.

LUSTBADER, E. D. & STODOLA, R. K. (1981) AS160 Partial and marginal association in multidimensional tables. *Applied Statistics*, **30**, 97–105.

MAHLER, M. S., LUKE, J. A. & DALTROFF, W. (1945) Clinical and follow-up study of the tic syndrome in children. *American Journal of Orthopsychiatry*, **15**, 631–647.

MOLDOFSKY, H., TULLIS, C. & LAMON, R. (1974) Multiple tic syndrome (Gilles de la Tourette's syndrome): clinical, biological & psychological variables and their influence with haloperidol. *Journal of Nervous and Mental Diseases*, **159**, 282–292.

SHAPIRO, A. K., SHAPIRO, E. & WAYNE, H. (1972) Birth, developmental, and family histories and demographic information in Tourette's Syndrome. *Journal of Nervous and Mental Diseases*, **155**, 335–344.

——, ——, BRUUN, R. D. & SWEET, R. D. (1978) *Gilles de la Tourette Syndrome*. New York: Raven Press.

TORUP, E. (1962) A follow-up study of children with tics. *Acta Paediatrica Scandinavica*, **15**, 261–268.

WILSON, R. S., GARRON, D. C. & KALAWANS, H. L. (1978) Significance of genetic factors in Gilles de la Tourette Syndrome: a review. *Behavior Genetics*, **8**, 503–510.

ZAUSMER, D. M. (1954) The treatment of tics in childhood: a review and a follow-up study. *Archives of Disease in Childhood*, **29**, 537–542.

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Late Paraphrenia or the Paraphrenias?

A Descriptive Study with a 10-year Follow-up

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This study describes 47 cases of paranoid psychosis occurring for the first time after the age of 60. Cases were drawn from the Camberwell catchment area and were followed up for 10 years or until death. The clinical picture of patients showed few differences from those described in earlier studies. Subgroups are described with contrasting clinical features and differing outcome. The findings suggest that late paraphrenia is a heterogenous syndrome giving the appearance of a spectrum of overlapping conditions with paranoid delusions.

Roth (1955) defined late paraphrenia as "a well organised system of paranoid delusions with or without auditory hallucinations existing in the setting of a well preserved personality and affective response". For their later study Kay & Roth (1961) modified the definition of paraphrenia to be "a suitable descriptive term, without prejudice as to aetiology, for all cases with a paranoid symptom complex in which signs of organic dementia or sustained confusion were absent and in which the condition was judged from the content of the delusional and hallucinatory symptoms not to be due to a primary affective disorder".

Post (1966) used wider criteria for his study of *Persistent Persecutory States in the Elderly* in which he included all paranoid states arising after 60 years of age which were unrelated to affective illness. Although up to 30% of his cases had definite or possible signs of organic brain damage, he suggested that "no support was found for the existence of a specifically organic or 'symptomatic' chronic paranoid psychosis". Kay (1972) suggested the existence of symptomatic schizophrenias in the elderly, similar to the cases occurring in the younger patients studied by Davidson & Bagley (1969), and found it likely that these cases with organic changes should make up a proportion of cases of late paraphrenia. In contrast, Grahame (1984), looking at the incidence of first-rank symptoms in a group of paraphrenics, treated the group as homogeneous, concluding that their nature was that of schizophrenia.

In the last two decades, concepts of nosology and aetiology have changed, with a greater awareness of the need to respect the complexity of interaction between organic and functional components in the elderly patient. The American Psychiatric Association's DSM-III (1980) has introduced a radical widening of the concept of organicity with organic delusional syndrome and organic affective syndrome as organic equivalents to functional schizophrenia and affective disorder. The homogeneity of disease entities in the elderly has been eroded, and replaced by the concept of a spectrum of heterogeneity. Studies such as those by Jacoby *et al* (1981), which described a subgroup of elderly depressives with CAT scan changes and a differing prognosis, and by Bergmann (1971), which identified a group of late onset neurotics with a distinct prognosis, are examples of this.

This study was carried out as a fresh look at paranoid states in the elderly, seeking to apply the current concepts of psychiatric nosology to patients clinically recognised as late paraphrenics. The issues of homogeneity and organicity were examined and a description of subgroups in relation to clinical features, aetiology and outcome was attempted.

Method

Using the Camberwell Register and the Maudsley Hospital Case Register, the case notes of all new cases diagnosed as functional paranoid psychoses over the age of 60, presenting in the years 1971 to 1975, from the Camberwell Area, were reviewed. Three cases with a history dating back before the age of 50 were excluded, as were five misdiagnosed cases which were felt to be secondary to dementia or acute toxic confusional states. These diagnoses were made clinically on the basis of the mental state and physical examinations, the history and investigations. Cases with a mental test score of 20 or less (out of 43) on the Gresham Ward Questionnaire (GWQ), (Notes on Eliciting and Recording Clinical Information, 1973), a routinely used questionnaire assessing orientation, general knowledge and memory, were excluded.

Forty-seven cases of persistent paranoid psychosis were identified and were followed up for 10 years or until death, using all available records (hospital, general practice, social services and the Register of Deaths) and by interviewing the next of kin and the survivors. Interview was possible in 14 of the 20 cases alive at 10 years and the remaining six had died between the end of 10 years and follow-up. Information from the clinical records was collected on the patient's background, initial presentation and outcome (including the nature and course of further illnesses). The patient's interview was carried out using an open-ended clinical interview, covering the current mental state, and the history whenever possible.

Reviewing the initial presentation of the 47 cases, five cases were felt to be due to depression with paranoid delusions (an 'affective' group) with clinical features of depression and holothymic delusions. A further five cases formed a 'symptomatic' group in that they appeared to have an association with chronic physical illness (syphilis; myeloproliferative disease; carcinoma of prostate with stilboestrol therapy; chronic heart failure and pernicious anaemia with neurological involvement). This left 37 cases, fulfilling Roth's criteria (1955) for late paraphrenia, as well as Post's (1966) criteria for 'persistent persecutory states in the elderly'. Statistical analysis of the data from this group was carried out to find associations between presentation, background and outcome variables.

These 37 paraphrenics were then further divided on the basis of organicity. Thirteen cases, progressing to dementia within 3 years, according to their clinical state and mental test scores, were incorporated into an 'organic' group, leaving 24 cases with 'functional' paraphrenia, fulfilling Kay & Roth's (1961) criteria.

The data from the 'organic' group was compared with that of the 'functional' group, and a search was made for significant associations between variables within the groups.

This group of 'functional' paraphrenics was retrospectively divided into further subgroups. Five cases formed a 'schizoaffective' group (with at least one subsequent episode being diagnosed as affective disorder), and the remaining 19 cases were divided by the presence or absence of Schneiderian first-rank symptoms into a 'schizophreniform' group (10 cases) or into a 'paranoid' group (nine cases). This created six subgroups of the original 47 cases of paranoid psychosis (schizophreniform, paranoid, schizoaffective, organic, symptomatic and affective). Significant associations and differences between the variables of these subgroups were investigated using the χ^2 test and t-test.

Results

All the cases presented over a 5-year period and were drawn from a population of 28,195 persons over 60 in the Camberwell area. Therefore the incidence for the wider group of paraphrenics ($n=37$) is 26 new cases per 100 000 at risk per year, whilst the incidence for the definitely 'functional' group ($n=24$) is 17 new cases per 100 000 per year.

Comparing the 'functional' group of paraphrenics ($n=24$) with the 'organic' group ($n=13$), there were no significant differences regards the characteristics at presentation except for the presence of lower GWQ scores in the latter group ($P<0.001$). The mean ages were 68.5 years (range 60–79) and 72.5 years (range 60–82) for the 'functional' and 'organic' groups respectively, and the sex ratios, male to female were 1:7 to 1:3. There was a preponderance of patients from the lower socioeconomic groups, and 46% of both groups were socially isolated (no relatives or friends giving regular social contact or support).

A family history of psychiatric illness (a third schizophreniform) was found in 42% of the 'functional group' and 23% of the 'organic' group. Deafness, visual impairment and physical disability (as noted clinically) were found in 25%, 25% and 17%, respectively, of the 'functional' group and 46%, 46% and 15% of the 'organic' group. 42% of the 'functional' group reported major disturbances of their childhood environments (such as separation from their parents by death or divorce, or adverse relationships with one or more parents or step-parents) compared with 31% of the 'organic' group, and precipitants to the illnesses were felt to have been present in 38% and 23% respectively ('precipitants' included all severe stresses temporally related to the onset of the illness).

All patients had, by definition, catathymic delusions which were not felt to originate solely from the prevailing mood. First-rank symptoms were present in 63% of the 'functional' group and 31% of the 'organic' group, whilst auditory, visual and somatic hallucinations were present in 83%, 13% and 8% of the former group, and in 62%, 38% and 8% of the latter group, respectively.

There were significant differences in the outcome of the two groups. After the index admission, 84% of the 'functional' cases returned home, compared with 46% of the 'organic' cases ($P<0.05$), the remainder in each case going to residential care. Of the 'functional' group, 33% had full recovery with insight and 21% without insight, whilst none of the 'organic' cases recovered full insight, and only 8% made a full recovery without insight ($P<0.05$).

By definition, the 'organic' group started dementing within 3 years, and all were institutionalised during the 10-year study period, compared with only 25% of the 'functional' group. In spite of no significant age difference, the survival of the 'functional' group was significantly better than the 'organic' group at 10 years ($P<0.05$) although there was no difference at 5 years. The 'functional' group had an average of 1.6 episodes (range 1–5) whilst the 'organic' group had a mean of 2.4 episodes (range 1–7) (non-significant).

Looking for associations within the 'functional' group, it was found that significantly fewer of those with first-rank symptoms had deafness ($P<0.05$), and that those with major disturbances of their childhood were less likely to have a precipitating stress before their illness ($P<0.05$). Those with childhood disturbances also had an increased chance of institutionalisation and survived less time at home ($P<0.05$). The presence of auditory hallucinations predicted a good response to treatment ($P<0.05$).

In the wider group of paraphrenics ($n=37$) made up of the 'functional' and 'organic' groups combined, it was found that increasing age was significantly associated with increased prevalence of deafness ($P<0.05$) and visual hallucinations ($P<0.05$). Those with a GWQ score of less than 30 had significantly less childhood environmental

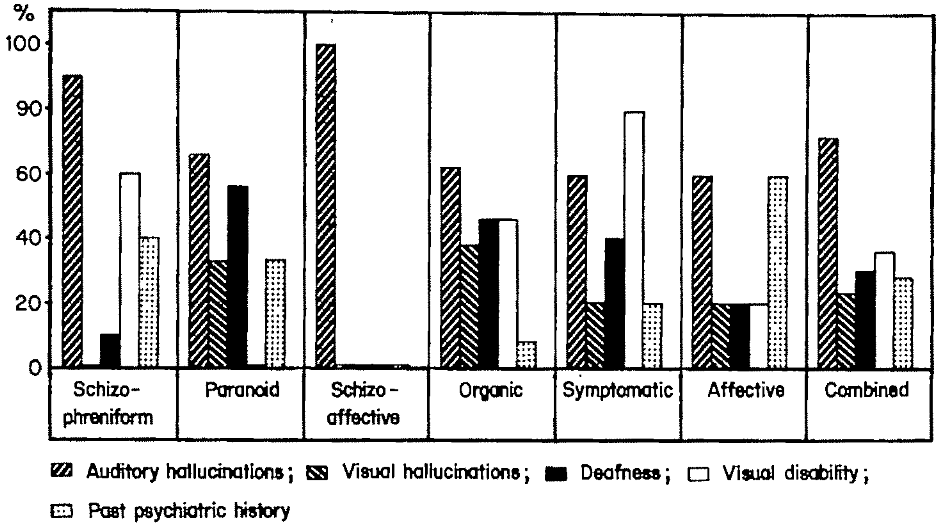

FIG. 1 Characteristics of subgroups at presentation.

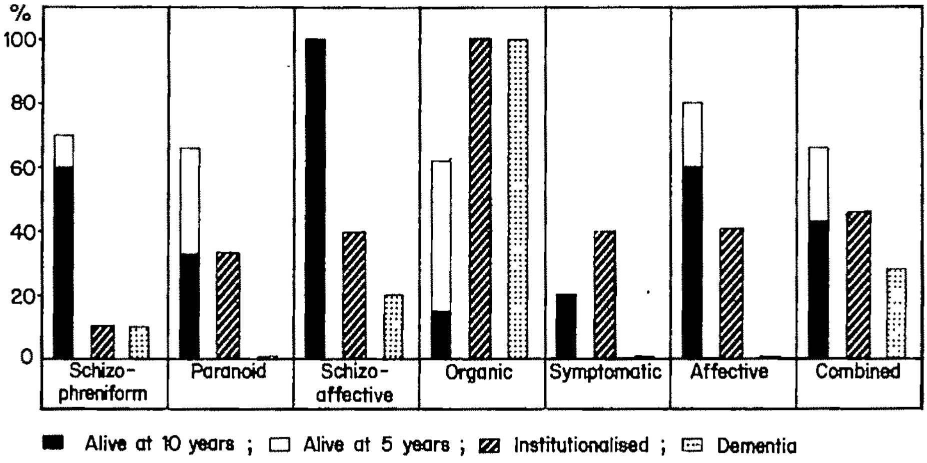

FIG. 2 Outcome of subgroups during the 10-year follow up.

disturbance ($P<0.05$) and significantly fewer males had such childhood upsets ($P<0.05$). Patients with a family history of psychiatric disorder had significantly less visual disability ($P<0.05$). The presence of first-rank symptoms was significantly associated with the absence of deafness ($P<0.01$). Social isolation was associated with increased prevalence of deafness ($P<0.05$).

Older age was associated with an increased rate of death by 10 years ($P<0.01$), increased institutionalisation (especially early) ($P<0.01$) and a worse response to medication ($P<0.05$). A GWQ score between 20 and 30 (out of 43) was associated with dementia ($P<0.001$) and institutionalisation ($P<0.01$) whilst visual hallucinations predicted higher mortality ($P<0.05$). Those with a previous history and those who developed insight following their index admission had better survival at home ($P<0.05$).

Comparing the six subgroupings, (schizophreniform, paranoid, schizoaffective, organic, symptomatic and affective) described above, the differences in characteristics on presentation are illustrated by Fig. 1, a histogram showing the percentage of patients with each symptom. (First-rank symptoms are not included as they are used to define the subgroups.) Similarly the outcome of the subgroups is shown in Fig. 2. The schizophreniform group showed less institutionalisation ($P<0.001$), more visual disability ($P<0.01$), fewer visual hallucinations ($P<0.05$), a better treatment response ($P<0.05$) and a better survival at 10 years ($P<0.05$). The paranoid group showed less visual disability ($P<0.01$), survived longer at home ($P<0.01$), had fewer children ($P<0.05$) and had a trend ($P<0.06$) towards having fewer episodes. The schizoaffective group had better survival to 10 years ($P<0.05$), surviving longer at home ($P<0.01$), had more frequently a good response to treatment ($P<0.05$) and had fewer children ($P<0.05$). The affective group with paranoid depressions had more children ($P<0.05$) and survived longer at home ($P<0.01$).

The organic group was associated with more visual hallucinations ($P<0.05$), a worse response to treatment ($P<0.05$), higher mortality by 10 years ($P<0.05$), and poor survival at home ($P<0.01$) with increased incidence of institutionalisation ($P<0.001$). The symptomatic group had more children ($P<0.05$), more visual hallucinations ($P<0.05$), greater visual disability ($P<0.01$) and higher mortality by 10 years ($P<0.05$).

The schizophreniform group has more first-rank symptoms than the paranoid group by definition, but in addition has significantly more visual disability ($P<0.04$) and less childhood environmental disturbance ($P<0.05$) than the latter group. Institutionalisation was greater in the organic group than the symptomatic group ($P<0.05$).

Discussion

Although this study suffers from the same limitations as other retrospective studies, the quality and thoroughness of the initial assessments were good. The demographic data, clinical features and outcome of the cases are in broad agreement with previous studies, showing that this is a representative sample of paraphrenics. The patients were drawn from the Camberwell catchment area of the Maudsley Hospital and can be expected to be a reasonably complete sample. Community surveys such as that by Kay *et al* (1964) concluded that other than a few 'chronic, mild or incipient forms' most major psychoses were known to the hospital services. The Camberwell area is typical of Inner London, and has a large Afro-Caribbean population. However, most of these are still of a younger age group, and only one West Indian female with paraphrenia was identified. The first admission age-specific rate per 100 000 was found to be between 17 and 26, depending on definitions used; rates similar to those quoted for 1966 by Kay (1972).

Studies of paranoid states in the elderly are partly hampered by the different criteria used in the previous studies. The differences between the concepts of 'late paraphrenia' (Kay & Roth, 1961), which excludes organic paranoid states, and 'persistent persecutory states of the elderly' (Post, 1966) which includes them, have not been clearly resolved. In this study, the narrower, 'functional' group ($n=24$) corresponds to 'late paraphrenia' whilst the wider group, including both 'functional' and 'organic' groups ($n=37$) corresponds to 'persistent persecutory states'.

In comparing the two elements, 'functional' and 'organic' of the wider group, it becomes clear that the two are indistinguishable on clinical characteristics at presentation, except for an increased likelihood of lower mental test scores in the 'organic' group. However, the 'organic' group has a significantly worse outcome in all respects. This is very much in line with the opinions of Kay (1972), but differs from Post's (1966) finding that there was no specific organic chronic paranoid psychosis.

Poor outcome, perhaps as a result of organicity, was predicted in the wider group ($n=37$) by old age, visual hallucinations, the absence of previous history and the failure to regain insight. Within the 'functional' group ($n=24$), with most organic elements excluded, good response to treatment was predicted by the presence of auditory hallucinations and increased institutionalisation was predicted by the presence of childhood environmental disturbances.

Looking at the associations within the wider group, there is a suggestion that there are endogenous, reactive and organic elements contributing to the clinical picture of the condition. A patient with a preponderance of one element, tended to show less evidence of the others. Hence patients with lower mental test scores at presentation (suggesting organicity) have significantly less childhood disturbance, whilst features indicative of endogenous elements, such as a positive family history and the presence of FRS are associated with significantly less visual disability and deafness respectively (possible reactive elements).

Similarly, looking at the associations within the 'functional' group, the reactive and endogenous elements can again be seen. First-rank symptoms remain associated with less deafness, and childhood disturbances are associated with significantly fewer precipitants.

The discussion above of endogenous, reactive and organic elements in the paraphrenic syndrome led to the division of the total sample into the subgroups. In this the schizophreniform group approximates most closely to late-onset endogenous schizophrenia, the paranoid group to reactive forms, whilst organicity plays a major role in the symptomatic and organic groups. These groupings are arbitrary, but nevertheless have some validity according to the significant differences between the groups, not totally explained by their criteria. However, in comparison, when the divisions suggested by Post (1966) and Kay & Roth (1961) were applied to this sample, they gave no significant findings whatsoever.

In short, this study gives some evidence that paraphrenia is not a clinically homogenous entity, and suggests that organicity (as perceived in DSM-III) can play a substantial role, with a different outcome for those who are older, those with slight decrease of mental functioning, and those with visual hallucinations. It also suggests that there may be value in looking at those cases with and without first-rank symptoms, and schizoaffective change. The study suffers from the problems of being partly retrospective, and from the fact that the initial assessment of cases is now 10 years out of date. It needs to be repeated prospectively, using a sample of paraphrenics diagnosed by structured interview assessment, and assessed with computerised tomography for brain density and ventricular-brain ratio. In such a study, heterogeneity might be illustrated more objectively by a cluster analysis revealing distinct clusters at various levels.

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References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) DSM-III. Washington, DC: American Psychiatric Association.

Bergmann, K. (1971) The neurosis of old age. In *Recent Developments in Psychogeriatrics* (ed. D. W. K. Kay & A. Walk). British Journal of Psychiatry Special Publication No. 6.

Davidson, K. & Bagley, C. R. (1969) Schizophrenia-like psychoses associated with organic disorders of the CNS: A review of the literature. In *Current Problems in Neuropsychiatry* (ed. R. N. Herrington). British Journal of Psychiatry Special Publication No. 4.

Grahame, P. S. (1984) Schizophrenia in Old Age (late paraphrenia). *British Journal of Psychiatry*, **145**, 493–495.

Jacoby, R. J., Levy, R. & Bird, J. M. (1981) Computed tomography and the outcome of affective disorder: a follow-up study of elderly patients. *British Journal of Psychiatry*, **139**, 288–292.

Kay, D. W. K. (1972) Schizophrenia and schizophrenia-like states in the elderly. In *Contemporary Psychiatry* (ed. T. Silverstone & B. Barraclough). London: Royal College of Psychiatrists.

—— & Roth, M. (1961) Environmental and hereditary factors in the schizophrenias of old age ('late paraphrenia') and their bearing on the general problem of causation in schizophrenia. *Journal of Mental Science*, **107**, 649–686.

——, Beamish, P. & Roth, M. (1964) Old age mental disorders in Newcastle-upon-Tyne Pt 1, a study of prevalence. *British Journal of Psychiatry*, **110**, 146–158.

Notes On Eliciting and Recording Clinical Information (1973) The Department of Psychiatry Teaching Committee, The Institute of Psychiatry, London. Oxford: Oxford University Press.

Post, F. (1966) *Persistent Persecutory States of the Elderly*. Oxford: Pergamon Press.

Roth, M. (1955) The natural history of mental disorder in old age. *Journal of Mental Science*, **101**, 281–301.

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The Nithsdale Schizophrenia Survey

VI. Relatives' Expressed Emotion: Prevalence, Patterns, and Clinical Assessment

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A review of all known schizophrenics ($n = 142$) in Nithsdale, Scotland, found 50% were living with relatives or friends. Of these, 32% had high contact with a relative showing high expressed emotion (EE); put another way, 87% of all Nithsdale schizophrenics were not living in a high contact/high EE family. Parents were more critical than spouses, and more showed emotional over-involvement. A retrospective review showed a trend, not of statistical significance, towards a higher admission rate to in-patient care in patients from high EE homes. Of relatives believed by a consultant psychiatrist to be neither critical nor hostile, 80% fell into the low EE category, but only 44% thought hostile or critical belonged to the high EE category; that is, the clinician had a wider view of high EE than the Camberwell Family Interview. Relatives' scores on a short patient-rejection scale were higher in high EE than in low EE relatives, but the wide scatter of scores suggested the scale could not be used to identify an individual relative with high EE.

Reports over 25 years from the MRC Social Psychiatry Unit in London have suggested the prognosis of schizophrenic patients discharged from hospital may depend on their living arrangements. Patients living with relatives who show high expressed emotion (EE) are more likely to relapse, especially if face-to-face contact between patient and relative is high and the patient is unprotected by maintenance antipsychotic medication (Brown *et al*, 1962, 1972; Vaughn & Leff, 1976). These findings have become of increasing clinical relevance in recent years as in both the UK and USA family therapy which includes education, relatives' groups and family sessions has been shown to reduce contact and lower EE, and thus substantially reduce relapse rates (Leff *et al*, 1982, 1985; Falloon *et al*, 1982, 1985).

Both the UK and USA studies examined patients in an urban setting. A study in Chandigarh, Northern India found different levels of expressed emotion (Leff, 1986); whether this was due to rural or cultural factors is not certain. The present study is the first to examine schizophrenics living in a rural area in the West. It is also the first to assess the *prevalence* of high EE in a community; in contrast with previous studies it does not confine itself to patients recently admitted to in-patient care. By assessing prevalence the amount of work likely to face a NHS clinician willing to embark on prophylactic family therapy can be estimated. The study also describes patterns of EE in different types of relative, and examines, retrospectively, rates of admission to in-patient care in patients living on their own, and with different categories of relative.

If clinicians are prepared to carry out family therapy they must be able to recognise high EE. EE is currently assessed by the Camberwell Family Interview (CFI), which cannot be used without specialised training and is time consuming; it is therefore not suitable for use in everyday clinical practice. Are there any shortcuts in the assessment of EE? The present study examines two possibilities, firstly an experienced clinician's hunch, and secondly a short 'patient rejection' scale administered to relatives by a community psychiatric nurse.

Method

Patients

Patients were drawn from Nithsdale, a rural, historically ancient and geographically discrete part of Dumfries and Galloway Region, Scotland. Those included were all in-patients, day-patients and out-patients of Crichton Royal Hospital, Dumfries, who on the census date (1 July 1985) had a firm case record diagnosis of schizophrenia and whose home address was in Nithsdale. Also, all general practitioners in the area replied to a questionnaire which identified other schizophrenics known to the practice and receiving treatment but not known to the hospital. The census identified 142 schizophrenics; as the population of Nithsdale is 56 000, this gives a point prevalence of 2.53

per 1000 of the total population. Sixty-eight per cent fulfilled the Feighner criteria for 'definite' or 'probable' schizophrenia (Feighner *et al*, 1972).

At the time of the census 20% were long-stay in-patients, 28% lived alone or in lodgings, and 2% lived with relatives under 18 years of age. Fifty per cent ($n=72$) were living with adult relatives or significant others and it was these on whom the present study focused. Forty nine per cent of such patients were male (mean age 42 years, s.d. 12 years, range 21-66 years; mean length of illness, as estimated from first psychiatric hospital contact, 14 years, s.d. 10 years, range 1-40 years) and 51% female (mean age 50 years, s.d. 13 years, range 27-79 years; mean length of illness 18 years, s.d. 12 years, range 2-48 years).

Scrutiny of case records identified all Nithsdale schizophrenics admitted to psychiatric in-patient care in the year preceding the census, 1 July 1984-30 June 1985.

Relatives

The type of relative with whom the 72 patients were living is shown in Table I. More males than females were living with parents (χ^2 6.75: $P<0.01$). Relatives of 12 patients were not interviewed: six patients had refused to take part in any of the previous Nithsdale surveys; a general practitioner refused access to a seventh patient; three patients refused access to relatives; and two relatives refused to be interviewed. Of the 12 patients, two had had no previous psychiatric admissions and the mean time from the census to the previous admission in the remaining ten was 12 years (s.d. 8 years, range 2-26 years).

TABLE I
Patients' living arrangements

Patient lives with: %	*Total* ($n=72$)	*Male* ($n=35$)	*Female* ($n=37$)
Parents	41	57	24
Spouse	42	34	49
Child(ren)	4	0	8
Other relative	6	6	5
Friend	7	3	14

Assessment

One of us (A.D.T.R.), trained in the use of the CFI by Dr Christine Vaughn, carried out all interviews which on all but three occasions took place in the patients' homes over a 3-month period following the census. Interviews were taped and an assessment made from the recordings of the five main components of EE, namely criticism, hostility, emotional over-involvement, warmth and positive comments. The amount of face-to-face contact between relative and patient during a typical week was also assessed through the construction of a time budget; more than 35 hours per week indicates high contact (Vaughn & Leff, 1976). The husband or wife of a married patient was always seen; where an unmarried patient lived with both parents, both mother and father were interviewed. In all, 70 relatives or friends were seen. A relative fell into the high EE category if he had a score of 6 or more critical comments, any degree of hostility, or a rating of 3 or more on emotional over-involvement (EOI) (Leff *et al*, 1982).

At the same time as the CFI ratings were made the consultant psychiatrist (R.G.McC), responsible for the patients' clinical management and blind to the ratings, made his own assessment of expressed emotion through the use of a simple 4-point global scale:

> I think this relative (i) is probably neither critical nor hostile (group 1 relatives), (ii) is possibly neither critical nor hostile (group 2), (iii) is possibly critical and/or hostile (group 3), (iv) is probably critical and/or hostile (group 4).

To make the assessment as simple as possible, no attempt was made to rate EOI. Over the same period, community psychiatric nurses visited relatives in their homes and asked them to complete a patient rejection scale (PRS) (Kreisman *et al*, 1979*). This is a 24-item questionnaire containing such statements as "I am very disappointed in my . . ."; "I wish my . . . had never been born". The relative is invited to respond to each statement in one of seven ways ranging from "I feel this way 'always'" through 'sometimes' to 'never'. Each item is scored 1-7 and a grand total obtained (range 24-168); a lower score indicates less hostility.

Results

Prevalence and patterns of high EE

At the time of the survey 58% ($n=35$) of the 60 patients were living with low EE relatives, 42% ($n=25$) with at least one high EE relative, and 32% ($n=19$) with at least one high EE relative with whom they were in face-to-face contact for more than 35 hours each week. Thirty relatives showed high EE. The breakdown by type of EE was as follows: criticism, as evidenced by a score of 6 or more critical comments, without EOI, as evidenced by a rating of less than 3, was shown by 10% of relatives ($n=7$), criticism and EOI by 21% ($n=15$) and EOI alone by 11% ($n=8$). Hostility, as evidenced by a score of 1 or more, shown by 8% ($n=6$) was never found without criticism. Sixteen of 21 mothers (76%) interviewed showed high EE, three of six fathers (50%) (that is, 70% of parents showed high EE); one of nine wives (11%), four of 14 husbands (29%) (that is, 22% of spouses); four of nine other first degree relatives (44%); two of three more distant relatives (67%); none of eight friends showed high EE. Parents had significantly higher scores when compared with spouses in mean number of critical comments (5.67 (s.d.4.79) vs 2.82(2.29): t-test, $t=2.60$, $P<0.02$), and also more showed EOI (63% vs 13%: $\chi^2=10.89$; $P<0.001$). There were no statistically significant differences in the percentage showing hostility (11% parents ($n=3$) vs 4% ($n=1$)) but the numbers in each group were very small.

Patients living in high EE homes, in low EE homes or on their own did not differ in: sex distribution, length of illness, and number of previous admissions. Patients living on their own were older than those living in high EE homes

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*The questionnaire has been expanded somewhat since the first version was published (full details on request to Dr D. Kreisman, New York State Psychiatric Institute, New York, 10032) (mean 51 years vs 43 years; t-test: $t=2.65$, $P<0.02$). More patients in high than in low EE homes lived with parents (68% vs 14%; χ^2 15.88; $P<0.001$).

Admissions to in-patient care

Scrutiny of case records showed that all Nithsdale schizophrenics admitted to in-patient care in the year preceding the census ($n=27$, 24% of all non long-stay in-patients) did so as a result of an exacerbation of a chronic schizophrenic illness or a fresh episode. There was no difference between admissions and non-admissions in: age, sex distribution, marital status and length of illness. Admissions had had more previous admissions than non-admissions (mean 7.3 vs 4.9; t-test: $t=2.12$, $P<0.05$).

TABLE II

Admission of Nithsdale schizophrenics to in-patient care in previous year

Living arrangement	*Total number of patients*	*Number of patients admitted*	*Percentage of patients admitted*
Lives alone	42[1]	9	22
No medication	14	4	29
Receiving medication	27	5	18
Low EE family	35	9	26
No medication	14	6	43
Receiving medication	21	3	14
High EE family	25	9	36
No medication	6	1	17
Receiving medication	19	8	42
High EE/high contact family[2]	19	7	37
No medication	4	1	25
Receiving medication	15	6	40

1. Medication unknown in one patient.
2. More than 35 hours per week in face-to-face contact.

Patients were then grouped on the basis of domiciliary status and whether they had received maintenance anti-psychotic medication in the year preceding the census or at least until the time of hospital admission (Table II). Admission rates for the three main groups (patients living on their own, in low EE homes and in high EE homes) ranged from 22% to 36%; the trend, not statistically significant, was towards a higher admission rate in patients from high EE homes. Admission rates for patients in high and low contact with high EE relatives were virtually the same. There were no significant differences between relatives of patients admitted and not admitted in mean number of hours face-to-face contact with patients and mean scores on criticism, hostility, emotional over-involvement, warmth and positive comments sub-scales. A trend in patients living on their own or in low EE homes was that admission rates were lower in patients on anti-psychotic medication (Table II); in contrast the trend in high EE patients was that admission rates were higher in patients on medication. No trend, however, reached statistical significance.

Comparison of CFI with consultant's assessment and PRS

The consultant psychiatrist rated 54 of the 70 relatives; he believed his knowledge of the remainder was so slight that an assessment could not be attempted. He assigned 17 relatives to group 1 ("probably neither critical nor hostile"), 12 to group 2, 14 to group 3 and 11 to group 4 ("probably critical and/or hostile"). When groups 1 and 2 were combined, it was found that 23 out of 29 relatives (80%) fell into the low EE category as assessed by the CFI; when groups 3 and 4 were combined, 11 out of 25 (44%) fell into the high EE category. Concordance rates were only marginally or not improved when relatives in groups 1 and 4 were compared respectively with those in low and high EE categories, and when groups were compared with relatives categorised by the criticism and EOI sub-scales of EE. The number of relatives showing hostility on the CFI ($n=4$) was too small to warrant examination of concordance rates but two fell into group 3 and two into group 4.

Fifty-eight relatives completed the PRS; the remainder were not known to the community psychiatric nurses. Mean scores on the hostility questionnaire for different categories of relative as determined by the CFI are shown in Table III. Although there were statistically significant differences between mean scores in different EE categories, there was such a wide scatter of scores in high EE relatives (see standard deviations in Table III) that it was not possible to determine a cut-off point on the PRS above which most high EE but few low EE relatives would be found.

TABLE III

Scores on patient rejection scale

Type of relative as determined by Camberwell Family Interview	*Number of relatives*	*Rejection score* *Mean score*	*s.d.*	*Student's 't'*	*P*
High expressed emotion	25	68	27	2.05	<0.05
Low expressed emotion	33	56	18		
Hostile[1]	5	93	11	3.61	<0.001
Not hostile	53	58	21		
Critical[2]	17	76	25	3.40	<0.01
Not critical	41	55	19		

1. Any degree of hostility on CFI
2. Six or more critical comments on CFI

Discussion

Relatives of 12 patients were not interviewed and some might belong to the high EE category. However, either patients had no history of admission to a psychiatric hospital or had not been admitted for many years. If there is a relationship between high EE, relapse and subsequent in-patient care it is likely few of these relatives fell into the high EE category.

The study, the first prevalence survey of high EE, found that in a community of schizophrenics 50%

were living with relatives or friends and of these, 32% lived in high contact with high EE relatives, the family structure believed by Leff and colleagues to be most conducive to schizophrenic relapse. However, put another way, 87% of all Nithsdale schizophrenics were *not* living in a high contact/high EE family. In terms of absolute numbers, therefore, it is unlikely that at any given time many Nithsdale schizophrenics have to face this sort of family life. If these results obtain in other parts of the country, and if NHS clinicians wish to embark on prophylactic family therapy with schizophrenics at risk, then the amount of work involved is not likely to be great. It is possible of course that within an individual the level of EE fluctuates over time and that a higher or lower prevalence of high EE might have been found on another date. However, with highly enmeshed families (high EE, high contact), EOI in one study barely changed in relatives where no therapeutic intervention took place, at least over a 9-month period (Leff *et al*, 1982). Criticism appears less stable; a considerable reduction occurred over 9 months in some relatives where patients markedly improved (Brown *et al*, 1972).

A common family structure in Nithsdale is the male schizophrenic living with elderly parents (McCreadie, 1982); such a patient may be at high risk as the present survey found parents were more critical and over-involved than spouses. Why this should be so is not clear. It has previously been suggested that more emotion is expressed by those with low rates of contact with friends and relatives (Brown *et al*, 1972). Perhaps parents are more isolated than spouses, most of whom were born and brought up in Nithsdale and possibly had other supportive contacts more readily available outside the nuclear family.

Comparison of the present findings with other studies is very difficult, firstly because the Nithsdale survey examines prevalence while the others examined relatives of patients recently admitted to in-patient care, and secondly because different cut-off points in the subscales of criticism and EOI were used at different times to determine the presence or absence of high EE. At the suggestion of Dr Leff (pers. comm.), the fairest comparison is to use a cut-off of 6 for criticism and 4 for EOI (although 3 has been used in this and later studies); and to consider only relatives of Nithsdale schizophrenics recently admitted to in-patient care, that is, during the previous year. The recalculation of results suggests the prevalence of high EE in rural Nithsdale (50%) might be similar to that in urban London (57%) but much higher than in Chandigarh, (23%; cut-off of 3 for EOI) and lower than in California (67%).

The results of the retrospective review must be treated very cautiously because, as discussed above, EE may in some cases fluctuate over time and if relatives had been assessed at the time of the patients' admission more might have shown high EE than in the prevalence survey. Nonetheless the retrospective results are of some interest. Although in the year previous to the survey there was a trend towards increased admission rates among schizophrenics living in high EE families there were no statistically significant differences in admission rates between patients living on their own or with relatives, between patients living with high and low EE relatives and between patients having little or much contact with high EE relatives. This contrasts sharply with relapse rates over 9 months in the prospective studies reported by Vaughn & Leff (1976). In these studies, 13% of patients discharged to low EE homes but 51% of patients discharged to high EE homes relapsed. Also, medication in the present study did not appear to exert a prophylactic effect in patients living with high EE relatives; in contrast, in the studies of Vaughn & Leff, 92% of patients discharged on no medication to high contact/high EE homes relapsed. However, the numbers of patients in the different Nithsdale subgroups are perhaps too small to warrant serious statistical analysis (for example there were only four patients in the high contact/high EE/no medication group).

It may be that other more important determinants of relapse swamp the contribution of family life in a community of schizophrenics. Patients admitted over the preceding year had had more previous admissions than patients who were not admitted; this may simply reflect severity of illness. Also, life events which are not assessed by the CFI can affect relapse rates (Leff & Vaughn, 1980). Alternatively, might there be protective factors in a rural community such as Nithsdale which do not exist in an inner-city area such as Camberwell? Most schizophrenics in Nithsdale were born and brought up in the area and many still have some contact with acquaintances, friends and more distant relatives outside the nuclear family home. Also, there is a well developed community psychiatric nursing service, a considerable number of patients are regular attenders at the day hospital, and most attend out-patient clinics. Perhaps such contributions outside the home are more widespread and better developed than in an urban area; advice, encouragement and support from these people may help to protect against the hostility, criticism and over-involvement of close relatives.

Neither the clinician's hunch nor the PRS proved to be an adequate shortcut to the assessment of high EE. If the consultant believed a relative was neither hostile nor critical, then it was highly likely that the relative fell into the low EE category as determined by the CFI. However, he had less success in determining which relatives had high EE. He appeared to have a much wider concept of hostility and criticism than that determined by the CFI and thus assigned many low EE relatives to the high EE category. If on the basis of his clinical hunch he and his team were to embark on family therapy for schizophrenics living with high EE relatives a substantial number of low EE families would find themselves in treatment—perhaps no bad thing.

The PRS completed by relatives certainly distinguished in a statistical sense between high and low EE categories. However, because of the wide scatter of scores on the questionnaire in the high EE category, there was no clear cut-off point above which the clinician could confidently assert most high EE but few low EE relatives would be found. The PRS, therefore, may be of use for research purposes but is not appropriate in an everyday clinical setting where the purpose is to identify an individual relative with high EE. Statistical probability does not yield precise prediction.

As an important by-product of the study, valuable insights were gained into the families of the Nithsdale schizophrenics and we would like to conclude by recording some of our more vivid impressions. Interviewing the family and friends of a given patient was a dialogue about the lives of people who have taken on a considerable burden of care with varying degrees of willingness. The impression left was of people struggling, but often succeeding in coming to terms with a major change in a child or spouse, a change that left that person unpredictable, sometimes dangerous. One father said that his world had shrunk to the small but chaotic world of his daughter. The most contented homes were often the group homes; it is surprising that in place of the expected emotional blunting there was warmth and positive regard. While many experiences were adverse, humour was not lacking, which was to be expected from a couthie people such as live in Nithsdale. Where a schizophrenic was married, the overall impression of the marriages was of stability with the offspring well cared for. Maybe the stigma helps to draw a family together to withstand any external threat. Any adverse effect of caring for schizophrenic children did not seem to be reflected in the mothers either. Two were aged over 90; both gave vivid interviews.

To see people in their homes gives an insight rarely gained in the out-patient department and a willingness by a doctor ('busy people') to take an interest in home life was remarked on favourably many times. Advice is needed for next-of-kin and must be greatly helped by knowing home circumstances (Kuipers & Bebbington, 1985).

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References

BROWN, G. W., MONCK, E. M., CARSTAIRS, G. M. & WING, J. K. (1962) The influence of family life on the course of schizophrenic illness. *British Journal of Preventive and Social Medicine*, 16, 55–68.

——, BIRLEY, J. L. T. & WING, J. K. (1972) Influence of family life on the course of schizophrenic disorders: a replication. *British Journal of Psychiatry*, 121, 241–258.

FALLOON, I. R. H., BOYD, J. L. MCGILL, C. W., RAZANI, J., MOSS, H. B. & GILDERMAN, A. M. (1982) Family management in the prevention of exacerbations of schizophrenia: a controlled study. *New England Journal of Medicine*, 306, 1437–1440.

——, BOYD, J. L., MCGILL, C. W., WILLIAMSON, M., RAZANI, J., MOSS, H. B., GILDERMAN, A. M. & SIMPSON, G. M. (1985) Family management in the prevention of morbidity of schizophrenia. Clinical outcome of a two year longitudinal study. *Archives of General Psychiatry*, 42, 887–896.

FEIGHNER, J. P., RUBINS, E., GUZE, S., WOODRUFF, R. A., WINOKUR, G & MUNOZ, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 26, 57–62.

KREISMAN, D. E., SIMMENS, S. J. & JOY, V. D. (1979) Rejecting the patient; preliminary validation of a self-report scale. *Schizophrenia Bulletin*, 5, 220–222.

KUIPERS, L. & BEBBINGTON, P. (1985) Relatives as a resource in the management of functional illness. *British Journal of Psychiatry*, 147, 465–470.

LEFF, J. (1986) Recent research on relatives' expressed emotion. In *Contemporary Issues in Schizophrenia*, (eds A. Kerr & P. Snaith). London: Gaskell.

—— & VAUGHN, C. (1980) The interaction of life events and relatives' expressed emotion in schizophrenia and depressive neurosis. *British Journal of Psychiatry*, 136, 146–153.

——, KUIPERS, L., BERKOWITZ, R., EBERLEIN-VRIES, R. & STURGEON, D. (1982) A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, 141, 121–134.

——, KUIPERS, L., BERKOWITZ, R. & STURGEON, D. (1985) A controlled trial of social intervention in the families of schizophrenic patients: two year follow up. *British Journal of Psychiatry*, 146, 594–600.

MCCREADIE, R. G. (1982) The Nithsdale schizophrenia survey. I Psychiatric and social handicaps. *British Journal of Psychiatry*, 140, 582–586.

VAUGHN, C. E. & LEFF, J. P. (1976) The influence of family and social factors on the course of psychiatric illness. A comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, 129, 125–137.

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Specific and Non-specific Effects of Educational Intervention with Families Living with a Schizophrenic Relative

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A brief educational intervention with families living with a schizophrenic relative is described and evaluated in terms of benefits for relatives' well-being and patient recovery. Education led to considerable knowledge gains and to reductions in relatives' reported stress symptoms and fear of the patient. There was a trend for relatives to be more optimistic concerning their role in treatment. At 6 months follow-up, only knowledge gains were maintained; however, relatives' perceptions of family burden were significantly reduced. The evidence suggests that family education may be useful both as a cost-effective intervention in its own right and in facilitating a more receptive attitude to subsequent family intervention.

Over the past two decades with the introduction of neuroleptic medication and the emphasis on de-institutionalisation there has been an increased pressure to discharge patients from hospital into the community. However, lack of adequate community-based medical and social provision has inevitably placed the burden of care on the patient's family; it has been suggested that over 50% of schizophrenic patients return to live with families (Goldman, 1980; Holden & Lewine, 1974). Families are therefore taking on much of the burden of care for their relatives particularly concerning the management of disturbed behaviour, often in the absence of sufficient information, skills and support (Carstairs *et al*, 1985). For many families the impact of this responsibility can be very distressing, and can lead to a high level of intra-familial tension and individual stress symptoms (Birchwood, 1983; Gibbons *et al*, 1984).

In addition to the needs of the individual with schizophrenia (not addressed here) there would, therefore, appear to be justification for directing attention to the needs of the supporting family both during the initial crisis and thereafter. There has, until recently, been a general lack of professional awareness and responsiveness to the family's needs in this respect. One significant aspect of this neglect has been the lack of time spent by professionals providing basic information about the illness, its treatment and practical advice concerning home management. Yet the need for, and benefits from, family-education are evident. In addition to meeting family needs, education may have beneficial effects for the schizophrenic member. Non-compliance with medication has been shown to be an important factor in schizophrenic relapse which family- (and patient-) education may help to ameliorate. Family-education has also been a common component in three large-scale family interventions which have reported the successful short-term control of relapse (Goldstein & Kopeikin, 1981; Leff *et al*, 1982; Falloon *et al*, 1982).

The specific role of education in these interventions is unclear. Berkowitz *et al* (1984) reported that in the Leff *et al* (1982) intervention, education led to their high EE (high relapse risk) group 'knowing more about diagnosis and management' and increased their optimism; other long-term attitude changes were confounded with the later intervention. However, comparisons of high and low EE families prior to the intervention had revealed no differences in these characteristics. McGill *et al* (1983) reported that their educational intervention led to parents acquiring knowledge 'approximating that of mental health professionals' and these knowledge gains were maintained 9 months after intervention. Non-specific effects other than knowledge acquisition were not assessed.

Education alone could, therefore, meet many of the needs of families (except the important one of in-depth management advice) – however, its function in the context of a large-scale family intervention remains uncertain. The present study was therefore undertaken to examine the efficacy of education *alone* by observing the specific (knowledge acquisition) and non-specific (stress reduction, attitude change) effects immediately and 6 months after intervention. In order to test whether information *per se* is important as opposed to its delivery by professional personnel, half of the families received information in booklet form through the post. Both groups were required to undertake homework exercises designed to maximise information assimilation and to prompt relatives to apply the information to their own situation.

Method

Subjects

Twenty-three families who had a schizophrenic relative participated in the study. The families were drawn from a random sample of patients known to the hospital. Of this sample, six patients were in hospital at the time of the study. (Of the original sample, nine families refused to participate in the study and six families dropped out prior to intervention; there were no drop-outs during the intervention period.) Criteria for selection of patients were: evidence of one or more 'first-rank' symptoms of schizophrenia (Schneider, 1959) observed at onset in the case notes; living at home or in close contact with the family (5 or more days per week) and stabilised on depot or oral neuroleptic medication. The sample consisted of 40 family members (28 parents, seven spouses and five other relatives) who were all English speaking. Forty-four per cent of the families were from social class level 3, the remainder being evenly distributed across social class groups 1, 2 and 4; however, only one family was from social class group 5. The schizophrenic sample, consisting of 18 males and five females, were a relatively chronic group with a mean age of 36.4 years (s.d. ± 14), mean illness duration of 7.9 (s.d. ± 6.8) years and mean number of 3.7 (s.d. ± 3.8) admissions. The majority were unmarried (15 of 23).

Design

Families were randomly assigned to either a group or a postal intervention condition. The education package was delivered in both intervention conditions at weekly intervals over a 4-week period.

Intervention

The aims of the intervention were to:

(a) improve relatives understanding of schizophrenia, its nature, symptoms and treatment
(b) improve relatives 'cognitive mastery' of their own situation by encouraging relatives to apply the information to their own circumstances (mainly through an instrumental component in the form of homework assignments).
(c) improve the quality of the relationship and reduce alienation between patient and relative by changing relatives' attitudes towards, and improving understanding of, schizophrenia
(d) emphasise the importance of maintaining relatives' personal well-being.

The intervention did not cover issues relating to behavioural management except in very broad terms.

The information given to relatives in both conditions was divided into four separate sections covering:

(a) concepts of schizophrenia, epidemiological data and possible causal factors.
(b) the symptoms of schizophrenia
(c) treatments and outcome
(d) hospital and community resources available and basic management guidelines.

(See Appendix for a more detailed description of the information covered in each of these sections.)

This information was presented in the form of four typed booklets corresponding to the four information sections outlined. The information was written in simple English and tailored as far as possible, to be comprehensible to the average family. A readability analysis (Flesch, 1948) showed that the booklets could be understood by individuals of average IQ (overall reading ease score = 56.75; IQ > 103).

Group condition

Those family members assigned to the group intervention participated in four weekly educational sessions conducted by the primary therapist (J.S.). The sessions were in a semi-structured seminar format involving oral presentation of the information as well as audiovisual aids. A video film entitled *Coping with Schizophrenia* (Birchwood, 1983) was used to illustrate common schizophrenic symptoms and behaviours. The content of the four sessions corresponded to the four information sections previously described. Family participation was encouraged through general question and answer discussion – each session lasting 60–90 minutes. At the end of the session, each family member received a booklet corresponding to the material covered in that particular session and a written homework exercise which they were invited to complete. These were designed to encourage families to apply the information they had received to their own situation.

Postal condition

Family members assigned to the postal intervention received a typed information booklet (corresponding to each of the information sections described previously and to that received in the group intervention condition) through the post at weekly intervals over a 4-week period. A covering letter accompanied each of the booklets which briefly outlined the information booklet and invited the family members to complete the corresponding 'homework' exercise attached.

Measures

All family members were assessed before and immediately after the educational intervention and at 6 months follow-up. The assessment instrument comprised a six-part paper and pencil questionnaire designed to assess:

Knowledge acquisition

This consisted of an 18-item multiple choice questionnaire designed to assess family knowledge and understanding about schizophrenia – in particular it concerned demography, aetiology, symptoms, medication and relevant hospital procedures.

Beliefs about schizophrenia and its treatment

This consisted of four 5-point scales separately assessing the family's beliefs and expectations about their role in treatment, effectiveness of medical treatment, likelihood of improvement and the control that the patient has over his symptoms.

Worry and fear

This consisted of two 5-point scales assessing aspects of the family's perception of the individual in terms of the amount of worry and fear about the 'individual and his behaviour'.

Behavioural disturbance

The symptom-related behavioural disturbance scale (SBDS) developed by Birchwood (1983) was employed. This is a 22-item measure of severity of patient disturbance. The reliability of this measure has been demonstrated in terms of inter-rater reliability ($r = 0.90$) and internal coherence (Chronbach's $\alpha = 0.84$). The scale correlates $r = 0.6$ with scales derived from the PSE.

Stress

This was measured using the symptom rating test (SRT) developed by Kellner & Sheffield (1973), modified and validated by Cochrane (1980), a 30-item measure of stress symptoms in the relatives incorporating anxiety, depression, somatic and inadequacy subscales.

Family distress

The family distress scale (FDS) developed by Pasamanick *et al* (1967), was employed. This is a 22-item scale which measures the impact of having a schizophrenic relative in the family in terms of the extent of disruption on family life, embarrassment and the concern of self and others. (Chronbach's $\alpha = 0.91$ using data from this study).

Results

Three (intervention style × test occasion) factorial analyses of variance with test occasions as a repeated measure were carried out on the pre-education, post-education and 6 months follow-up scores. There were no significant differences in any of the variables measures between group and postal relatives at baseline, except that postal relatives were significantly more worried ($t = 2.47$, d.f. = 38; $P < 0.01$).

Knowledge acquisition

The knowledge scores of both sets of relatives significantly increased from baseline to post-education ($F = 51.80$, d.f. = 1,38, $P < 0.001$). These improvements in knowledge from baseline were maintained at 6 months follow-up ($F = 51.38$, d.f. 1,38, $P < 0.001$). 'Group' relatives acquired significantly more knowledge than 'postal' relatives ($F = 5.22$, d.f. = 1,38, $P < 0.05$). This significant difference in knowledge between the two sets of relatives remained at 6 months follow-up ($F = 14.50$, d.f. = 1,38, $P < 0.01$). (See Table I, Fig. 1).

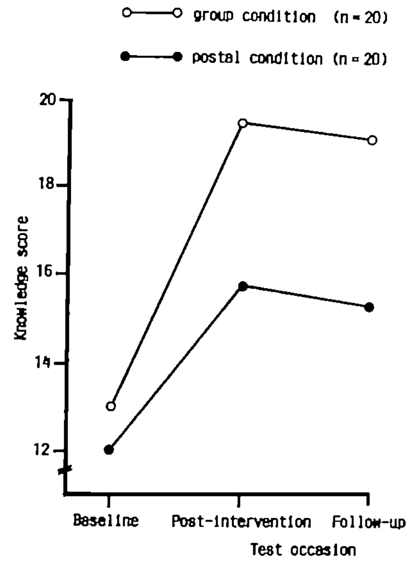

FIG. 1 Mean knowledge score for group and postal relatives across test occasions.

In the group relatives there was a mean improvement in knowledge on the questionnaire of 27% (from 58% to 85% following education) while in the postal group there was a mean improvement in knowledge on the questionnaire of 16.5% (from 52% to 68.5% following education.) These gains were maintained at a 6 month follow-up. Immediately following the educational intervention, 90% of the group relatives possessed relatively 'sophisticated' knowledge about schizophrenia (i.e. more than 70% correct) compared with only 60% of the postal relatives. This difference in degree of knowledge remained at 6 months follow-up.

Information acquired was evenly spread across the five main areas; demographic data, aetiology, illness (course and symptoms) hospital procedures and medication. Interestingly, 75% of families had little understanding about the function of medication prior to education. Post-education, 67% of families possessed a good knowledge about the effects of medication (score >3 out of 4).

Belief systems of relatives about schizophrenia

There were no significant differences between baseline scores and either post-education or follow-up scores for either group on any of their beliefs concerning the effectiveness of treatment, the likelihood of improvement or the control that the patient possessed over his symptoms. However, the two sets of relatives did differ in their belief concerning the family's role in treatment following education which was revealed in a significant interaction effect ($F = 5.37$, d.f. = 1,38, $P < 0.05$) (See Table I, Fig. 2).

TABLE I
Mean scores with standard deviations on each of the variables for group and postal intervention

	Group intervention						*Postal intervention*					
	Before		*After*		*Follow-up*		*Before*		*After*		*Follow-up*	
Variables	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*
Knowledge	13.20	5.24	19.55	3.33	19.10	3.61	12.00	4.08	15.75	3.27	15.30	3.01
Belief:												
family's role	2.55	0.99	3.00	1.03	2.95	0.89	2.95	1.05	2.70	0.92	2.90	0.97
Fear	0.95	0.88	0.70	0.73	1.05	0.88	1.25	1.16	0.90	0.97	0.60	0.75
SRT	14.00	9.78	11.95	7.67	15.35	10.60	15.95	8.50	15.15	8.92	15.40	9.19
FDS	9.20	6.08	8.75	6.56	6.60	6.21	14.05	11.33	11.95	8.48	9.70	8.55
SBDS	22.50	12.45	21.20	11.32	17.35	9.62	24.50	10.90	25.00	11.63	20.00	13.65

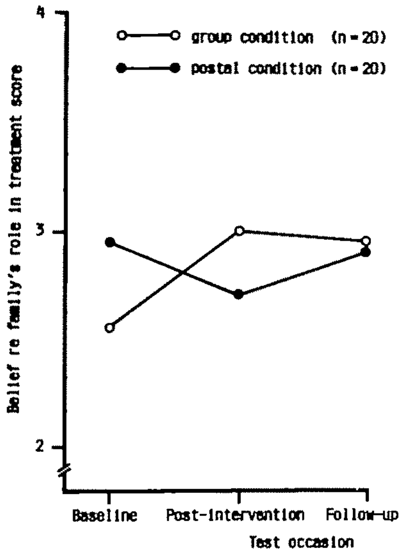

FIG. 2 Mean belief scale score concerning the family's role in treatment for group and postal relatives across test occasions.

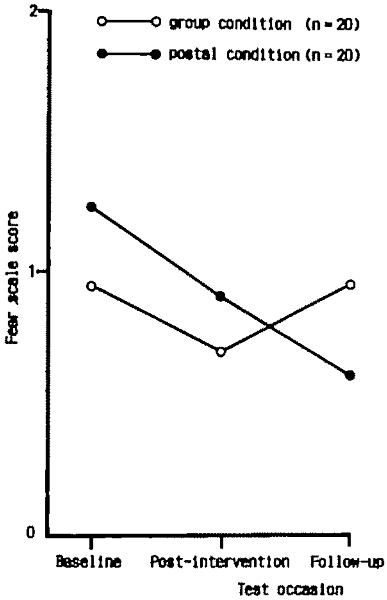

FIG. 3 Mean fear scale score for group and postal relatives across test occasions.

Results of t-tests revealed that there was a trend for group relatives to be more optimistic concerning the family's role in treatment ($t = 1.40$, d.f. = 38, $P < 0.10$). The scores of postal relatives did not significantly differ from baseline. This interaction effect disappeared at 6 month follow-up.

Worry and fear

Fear scores reflecting fear concerning safety of self, others and property were significantly reduced across both sets of relatives from pre to post as a result of education ($F = 5.63$, d.f. = 1,38, $P < 0.05$). Group relatives were not significantly different from postal relatives. However, the sets of relatives differed in their fear scores at 6 months follow-up compared to post-education reflected in a significant interaction effect ($F = 6.4$, d.f. = 1,38, $P < 0.05$). (See Table I, Fig. 3).

Results of t-tests revealed that the postal relatives continued to be significantly less fearful compared to baseline ($F = 6.05$, d.f. = 1,38, $P < 0.05$), while the fear scores for the group relatives returned to baseline levels.

Postal relatives worried more than group relatives at baseline and following education ($F = 5.78$, d.f. = 1,38, $P < 0.05$). However, this significant difference in worry scores between the two sets of relatives was not maintained at 6-month follow-up.

Psychological distress in the relatives

Stress scores on the Symptom Rating Test were significantly reduced across both groups of relatives from baseline to post-education ($F = 5.28$, d.f. = 1,38, $P < 0.05$). Neither set of relatives showed a significantly greater reduction in stress symptoms than the other. Reduction in stress symptoms was consistent across all four symptom rating test subscales: anxiety, depression, somatic and inadequacy. Those relatives who had the highest stress scores at baseline showed the biggest reduction in stress symptoms following education ($r = 0.39$, d.f. $= 38$, $P < 0.01$). The reductions observed at post-education were not maintained at 6 months follow-up where scores had returned to baseline levels. (See Table I, Fig. 4). The return to baseline level was consistent across all four subscales.

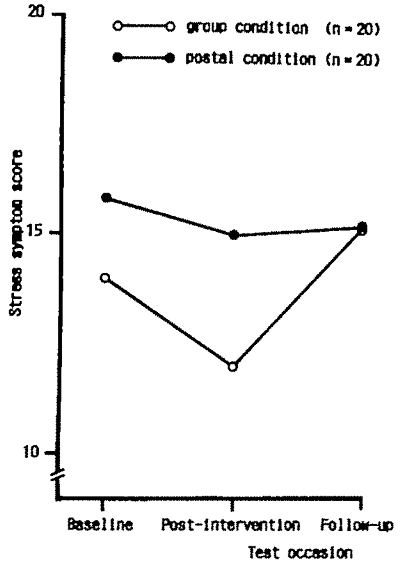

FIG. 4 Mean stress score for group and postal relatives across test occasions.

Burden on the family

There were no significant differences from baseline scores in reported burden (FDS) following education for either set of relatives. However, there were significant reductions in reported burden across both groups from the baseline score to follow-up ($F = 21.08$, d.f. $= 1{,}38$, $P < 0.001$) and post-education scores to follow-up ($F = 7.4$, d.f. $= 1{,}38$, $P < 0.01$). (See Table I, Fig. 5).

Symptom related behavioural disturbance (SBDS)

Where ratings of level of patient disturbance were made by two members of the same family the ratings were found to be significantly correlated ($r = 0.70$, d.f. $= 14$, $P < 0.01$). Ratings for relatives from the same family were therefore combined for the purposes of analysis.

There were no significant differences from baseline scores in level of patients' disturbance following education or at 6 months follow-up for either group. Level of patient disturbance as indexed by scores on the SBDS were found to be significantly correlated with family burden scores on the FDS ($r = 0.72$, d.f. $= 38$, $P < 0.01$).

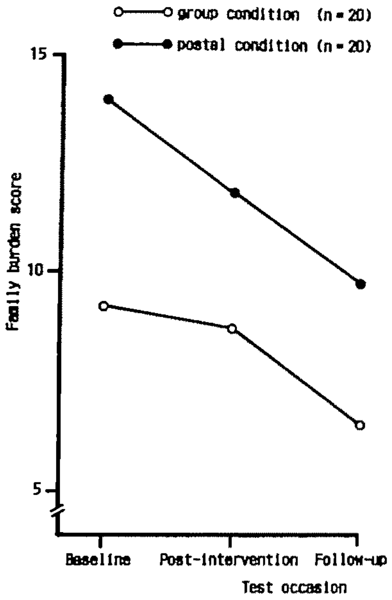

FIG. 5 Mean family burden scores for group and postal relatives across test occasions.

Correlational analyses

Between knowledge acquired and attitude change, stress and burden

The change in knowledge scores was not found to be significantly correlated with either the change in fear scores or the change in stress symptom scores observed following education. The change in knowledge scores was also not found to be significantly correlated with the change in perceived family burden observed at 6 months follow-up.

Between social class and knowledge scores

Social class was found to be significantly negatively correlated with initial knowledge scores ($r = 0.35$, d.f. $= 38$, $P < 0.05$) i.e. relatives from social class groups 1–3 had more knowledge concerning schizophrenia compared with groups 4 and 5, prior to the education course. Social class group was not significantly correlated with any of the specific or non-specific effects observed following education.

Between duration of illness and change in attitude, stress and burden

Correlational analysis revealed that duration of illness was not significantly correlated with any of the specific or non-specific effects observed following education.

Discussion

The four session education course led to considerable acquisition and retention of information across all participants and to actual reductions in relatives' stress symptoms and fear of the patient. Significantly, there was no correlation found between amount of knowledge acquired and these non-specific effects. Group relatives recalled more information about schizophrenia than postal relatives and there was a trend for the former to be more optimistic about the family's role in exerting control over the situation. At 6 months follow-up, only knowledge gains were maintained across all participants; all non-specific effects had disappeared, with the exception that postal relatives remained less fearful. However, it was noted that all relatives were reporting significantly less burden on the family. Similarly, no correlation was found between the amount of knowledge acquired following education and the reduction in perceived burden observed from baseline to follow-up.

The independent variables can be divided up into 'content' and 'context' of delivery. It is important to try to distinguish those effects which are influenced by content and those by contextual variables from a therapeutic viewpoint. The postal delivery of information served as partial control for certain 'placebo' elements in the group education package. In the group condition, there were certain additional elements, specifically, the presence of a therapist, other group members and repetition with clarification of the information being conveyed. The majority of specific and non-specific effects obtained were the *same* across both conditions with the exception that group relatives acquired significantly more knowledge and showed a trend towards being more optimistic concerning the family's role in treatment.

It seems likely that 'context' as well as 'content' contributes to knowledge acquisition. Pure information content common to both groups is clearly the crucial element in knowledge acquisition *per se*. However, contextual factors would appear to have a role in further enhancing knowledge assimilation conceivably through the combined effects of repetition and clarification of information – and the opportunity to listen to and discuss views with the therapist and other group members.

Since all non-specific effects observed were the same across postal and group conditions they could only be accounted for by information content or by placebo elements *common* to both conditions such as relatives' expectations, motivation and attention to problem. (However, group context may have played a significant role in further encouraging optimism through the opportunity provided to meet and listen to a therapist and other group members). It is difficult if not impossible to tease out the specific contributions of information content from these general 'placebo' effects. If contextual factors are crucial to the non-specific effects observed, the question is raised as to whether, aside from academic interest, one should be looking for ways to encourage rather than to eliminate such effects. Given the well-documented lack of information currently given or available to patients and their families (as the pre-knowledge scores demonstrate), information courses would appear to be a useful and cost-effective measure to disseminate information widely and easily. However, it must be borne in mind that these knowledge gains were observed in a general sample drawn from the community containing only six patients at acute admission. It is difficult without further study to assess whether the results are generalisable to families receiving education during the acute phase.

The value of patient education is an important but separate issue (not addressed here). In the authors' experience, it is adviseable that patient and family education are kept separate since the presence of the individual with schizophrenia can inhibit the relative's freedom to air sensitive issues and concerns and can be potentially disruptive in circumstances where the individual lacks insight into the illness. The impact of patient education is currently being addressed in other work by the authors.

It is interesting to note that 75% of the families possessed little understanding about the function of medication prior to education, a factor which may contribute to the widespread problem of medication non-compliance. Thus the provision of information about medication to the patient and his family holds out the prospect of encouraging compliance to medication regimes.

Education, however, is not purely an academic exercise to increase knowledge. In fact, the value of education would not seem to lie solely in knowledge acquisition *per se*, since information gained was not correlated with any of the non-specific effects. It may be argued that since no correlation emerged between information retained and non-specific effects, content is irrelevant as a medium of change. However, what may be important is the subjective effect of *being* or *feeling* more knowledgeable rather than the degree of improvement in knowledge itself. The subjective effect may act to improve relatives 'cognitive mastery' and perceived ability to influence the situation and thereby reduce their feelings of uncertainty and helplessness (cf. reductions in fear and the trend towards increased optimism). There may also be hidden direct effects of being more knowledgeable by filling in idiosyncratic knowledge gaps about their patient which in turn may serve to reduce feelings of stress and increase feelings of control over the situation. For example, in one case the family did not understand the relationship between the individual talking to himself and hearing voices; in another, the patient's refusal to eat meals cooked by his parents was initially viewed as a comment on their cooking rather than a behavioural sequel to a delusion of persecution.

These effects are clinically important because they may serve to reduce alienation of the family from the individual and encourage more positive relative–patient interaction and willingness to help the patient, factors which may mitigate against intra-familial tension. Education also served to improve relatives level of stress and perceived burden which may otherwise constrain the family's ability to provide a supporting rehabilitative environment. Specifically, a reduction in stress may enable relatives to become more tolerant and accepting, characteristics which again have been noted in low risk family environments (Greenley, 1979; Kuipers, 1979). Unfortunately, these effects were transient and their therapeutic impact limited, although the intervention was sufficient to reduce perceived family burden over 6 months. We would hesitate to suggest therefore that a brief educational intervention could substitute for more intensive management-focused interventions since the majority of benefits were short-term and insufficient in themselves to satisfy relatives' needs for specific management advice. (Many relatives requested specific advice about how best to manage problems at home following education.) Overall, the results suggest that a brief educational intervention may serve to 'prepare the ground' in terms of enabling assimilation and adherence to subsequent intervention of a management kind, although the latter must be undertaken promptly to capitalise upon the short-term changes observed (Birchwood & Smith, 1987). It seems likely that the potential contribution of an initial education phase in family intervention programmes may be in its enabling effect, encouraging factors which facilitate and removing practices which constrain effective management and communication (Birchwood & Smith, 1987).

It is possible that education incorporating basic management guidelines might be a sufficient stimulus in itself for certain families of limited experience (i.e. those families of younger acute patients of more recent onset) to develop their own problem-solving skills and resources to cope with and help their schizophrenic relative. This suggestion draws support from the successful, brief (6-week) intervention study conducted by Goldstein and Kopeikin (1981) which combined initial education aimed at improving understanding of schizophrenia with the development of management strategies to deal with stressful situations in the family. Despite its brief nature this intervention appeared to have had a significant effect on relapse, using a sample two-thirds of whom were first episode patients. The Goldstein study suggests that the period immediately after onset may represent the best time to conduct a family intervention before relatively permanent response characteristics set in.

The nature and method of delivery of education packages to date has been fairly arbitrary and varied widely across intervention studies. The experimental manipulation of method of delivery in the present study underlines the need to consider this more carefully. Although the results would seem to indicate that access to written information *per se* is as effective as group education and offers itself as a less costly alternative, one would hesitate to advocate 'bibliotherapy' on its own. The weaknesses of this approach lie in the lack of flexibility for information clarification and adaptation to individual needs and the absence of opportunity to listen to and discuss the information with a therapist and other group members, which would appear to be crucial for the full enabling potential of education to be realised. Work is currently underway by the authors to replicate and develop this study, comparing the effects of different methods of information delivery in combination with the experimental manipulation of an instrumental component in the form of homework assignments.

Appendix

The four information sections were as follows:

Section 1

The first section contained a brief outline as to how the concept of schizophrenia was developed and some epidemiological data about who could develop schizophrenia. Simple explanations of possible causal factors, e.g. the role of genetic and biochemical abnormalities were given, although the lack of conclusive evidence in this area was emphasised. The role of possible environmental stresses including family factors in the development and course of the illness were outlined. Although the families were reassured that they were not responsible for causing the illness, their important role in the recovery process was emphasised. Finally, a guarded but hopeful prognosis for the majority of patients was provided.

Section 2

The second section focused on the nature of schizophrenia, describing common symptoms and behaviours in terms of disturbances in thinking, feelings and behaviour as experienced by patients and observed by relatives. Variability in symptom patterns between patients was emphasised. Various familiar terms e.g. 'acute', 'chronic', 'positive symptoms', 'negative symptoms', 'relapse', 'remission', were elucidated. It was also stressed that negative symptoms should not be regarded as inevitable.

Section 3

The third section described the function of the relevant psychiatric services and the role of neuroleptic medication in acute and maintenance phases. Routine hospital procedures were outlined and difficulties in diagnosis were discussed. There was special emphasis on medication describing the various types of drugs usually prescribed, their purpose and their common side-effects. The effectiveness of drug therapy particularly with positive symptoms of schizophrenia and in preventing relapse was emphasised. Relatives were encouraged to discuss any queries or worries about medication with the prescribing doctor.

Section 4

The fourth section was concerned with helping relatives to identify support services in terms of hospital and community resources available. (Local and national branch addresses of various organisations including self help and family support groups were supplied and a short reference list to encourage further reading was provided). Finally, some very general advice was given to encourage relatives to look after their own needs. This advice encouraged families:

(a) To look after their own needs by carrying on with or taking up activities that they used to do. As part of this, relatives carried out a homework exercise in which they articulated their past and ideal life-styles.
(b) Not to centre their lives around the patient too much.
(c) Not to worry about giving the individual responsibilities around the house e.g. household tasks, but not to overburden him immediately after discharge.
(d) To try to plan and do things with the patient which the whole family enjoyed e.g. trips out.
(e) To map out their social support network and to talk about their problems and difficulties with friends.
(f) To expect bad times when things fail and expect stress, frustration and some unhappiness.

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References

BERKOWITZ, R., EBERLEIN-FRIES, R., KUIPERS, L. & LEFF, J. (1984) Educating relatives about schizophrenia. *Schizophrenia Bulletin*, **10**, 418–429.

BIRCHWOOD, M. (1983) *Family Coping Behaviour and the Course of Schizophrenia*. Unpublished PhD thesis, University of Birmingham.

—— & SMITH, J. (1987) Schizophrenia. In *Coping with Disorder in the Family* (ed. J. Orford). Beckenham, Kent: Croom-Helm.

CARSTAIRS, G., EARLY, D., ROLLIN, H. & WING, J. (1985) Informing relatives about schizophrenia. *Bulletin of the Royal College of Psychiatrists*, **9**, 59–60.

COCHRANE, R. (1980) A comparison of the symptom rating test and the Langner 22 item scale for use in epidemiological surveys. *Psychological Medicine*, **10**, 115–125.

FALLOON, I., BOYD, J., MCGILL, C., RAZANI, J., MOSS, H. & GILDEMAN, A. (1982) Family managment in the prevention and exacerbation of schizophrenia: A controlled study. *New England Journal of Medicine*, **306**, 1437–1440.

FLESCH, R. (1948) A new readability yardstick. *Journal of Applied Psychology*, **32**, 221–233.

GIBBONS, J., HORN, S., POWELL, J. & GIBBONS, J. (1984) Schizophrenic patients and their families. A survey in a psychiatric service based on a DGH Unit. *British Journal of Psychiatry*, **144**, 70–77.

GOLDMAN, H. (1980) The post-hospital mental patient and family therapy: prospects and populations *Journal of Marital and Family Therapy*, **6**, 447–452.

GOLDSTEIN, M. & KOPEIKIN, H. (1981) Short and long term effects of combining drug and family therapy. In *New Developments in Interventions with Families of Schizophrenics* (ed. M. Goldstein). San Francisco: Jossey-Bass.

GREENLEY, J. (1979) Family symptom tolerance and rehospitalisation experiences of psychiatric patients. *Research in Community and Mental Health*, **1**, 357–386.

HOLDEN, D. & LEWINE, R. (1974) How families evaluate mental health professionals, resources and effects of illness *Schizophrenia Bulletin*, **10**, 626–633.

KELLNER, R. & SHEFFIELD, B. (1973) A self rating scale of distress. *Psychological Medicine*, **3**, 88–100.

KUIPERS, L. (1979) Expressed emotion: a review. *British Journal of Social and Clinical Psychology*, **18**, 237–243.

LEFF, J., KUIPERS, L., BERKOWITZ, R., EBERLEIN-FRIES, R. & STURGEON, D. (1982) A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, 141, 121–134.

MCGILL, C., FALLOON, I., BOYD, J. & WOOD-SIVERIO, C. (1983) Family educational intervention in the treatment of schizophrenia. *Hospital and Community Psychiatry*, **34**, 934–938.

PASAMANICK, B., SCARPITTI, F. & DINITZ, S. (1967) *Schizophrenics in the Community*, New York: Appleton-Century-Crofts.

SCHNEIDER, K. (1959) *Clinical Psychopathology*, New York: Grune & Stratton.

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A Comparative Trial of a New Antidepressant, Fluoxetine

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This clinical trial of a new antidepressant, fluoxetine, shows it to be as effective as a standard tricyclic drug, imipramine. It is effective as a single daily dose and is free of any significant side-effects. It is less sedative and appears to cause fewer problems of weight increase. The three scales used to assess efficacy showed a very positive correlation.

While the development of the tricyclic range of antidepressants constituted the main breakthrough in the pharmacotherapy of depressive illness, the search has continued for more effective and safer antidepressant drugs. The main focus of attention in the new generation of antidepressants has been to isolate molecules which have a specific effect on different parts of the monoamine system of neurotransmission. Studies have shown that 5HT deficiency plays an important role in the psychobiology of depressive illness (Asberg *et al*, 1976; Van Praag, 1977).

Fluoxetine has a biochemical and pharmacological profile which is characterised by its selectivity of action in blocking the re-uptake of 5HT without influencing the noradrenaline or acetylcholine systems. So the efficacy of this drug would confirm the importance of this neurotransmittor in the aetiology of depressive illness and such specificity of action would avoid the unpleasant anticholinergic side-effects of the tricyclic drugs.

Pharmacokinetic studies in both humans and animals show fluoxetine to be rapidly absorbed after oral administration, with peak plasma concentrations occurring 6 h after administration. A plasma half-life of 3–4 days makes a single daily dose possible. It is detoxified mainly in the renal system. Radioactive assays showed 65% is recovered in the urine and about 12% from faeces. Blood concentrations and half-life are the same in elderly patients and the drug has no influence on ethanol metabolism (Lemberger *et al*, 1985).

A double-blind clinical trial was undertaken to evaluate clinical efficacy and safety of fluoxetine in comparison with a standard tricyclic antidepressant, imipramine, in the treatment of depressive illness.

The opportunity was taken to compare the relationship of some standard measures of depressive illness, both observer based – Hamilton Depression Rating Scale (Hamilton, 1960) and Montgomery Asberg Depression Scale (Montgomery & Asberg, 1979) – and a self-rating scale – Levine Pilowsky Depression questionnaire (Pilowsky *et al*, 1969).

Method

Patients

All 60 patients participating in this study fulfilled the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1975) for major depressive illness with a minimum score of 17 on the Hamilton Depression Rating Scale. Patients were excluded from the study if they suffered from significant physical illness, had any history of drug abuse or schizophrenia, or had a duration of illness of more than one year. Table I describes the characteristics of the cohort and the type of depression as allocated by the LPD questionnaire, which separates cases of endogenous depression without observer bias (Pilowsky & Boulton, 1970).

All patients had been drug-free for at least 1 week and on entering the trial they were randomly allocated to either fluoxetine or imipramine according to a double-blind parallel group experimental design. Signed informed consent was obtained from all patients after the trial procedure had been fully explained.

The trial was undertaken in two clinical centres. Of the patients who completed the trial 22 received fluoxetine and 28 received imipramine and both groups were highly comparable for sex, age, type of depression, duration of illness and previous history of depressive illness as described in Table I.

On each occasion when a patient attended, enquiry was made about the development of side-effects. This was done for all patients entering the trial. Ten patients withdrew from the trial before completion: either because they did not attend (four of these were on fluoxetine, one on imipramine) or because they did not feel they were benefiting (two on fluoxetine, one on imipramine), and (two taking fluoxetine) because they developed minor side-effects, namely palpitations, tremor, headache, and peculiar taste.

Treatment

Capsules were taken three times per day and contained either fluoxetine (40 mg) as a single morning dose, or imipramine (75 mg) in three divided doses. After the first

TABLE I
Patient characteristics

	Fluoxetine	*Imipramine*
Out-patients	22	21
In-patients	8	9
Female	22	20
Male	8	10
Age: years		
Mean	46.1	45.4
Range	22–70	24–70
Endogenous depression	14	15
Non-endogenous depression	16	15
Previous episode	14	17
Duration of current illness:		
less than 3 months	3	2
3–6 months	22	24
6–12 months	5	3

week, flexibility of dosage was permitted and the dose could be increased to fluoxetine (60 mg) again as a single morning dose or imipramine (150 mg) in three divided doses. Nineteen patients received the higher dose of fluoxetine and 15 patients the higher dose of imipramine. Temazepam was permitted for night sedation.

Assessments

Response to treatment was assessed by the Hamilton Depression Rating Scale (HRS), Montgomery Asberg Depression Rating Scale (MADRS) and by a self-rating questionnaire, the Levine Pilowsky Depression questionnaire (LPD), after 1, 2, 4 and 6 weeks.

Pulse rate, blood pressure and weight were assessed at the beginning, after 2 weeks and after 6 weeks of the trial. Full haemotological and biochemical screening was also carried out at these times.

Results

Clinical efficacy

Table III shows the initial severity and response as measured on the three scales, for all cases and for cases of endogenous depression as categorised by the LPD scale. Table II shows the number of patients who reached a satisfactory end point as measured on each scale.

TABLE II
Response to treatment—patients with good responses at end of trial

	HRS<6	*MADRS<9*	*LPD<7*
Fluoxetine $n=22$	10 (44.4%)	11 (50%)	12 (54.4%)
Imipramine	17 (60.7%)	15 (53.6%)	12 (42.8%)

Fluoxetine vs imipramine: χ^2 test = NS

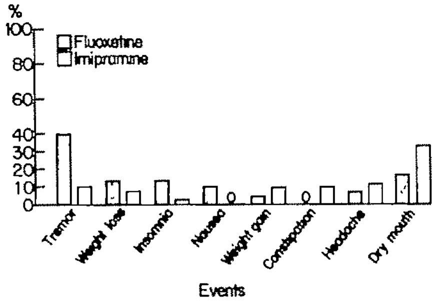

FIG. 1 Percentages of patients with less serious events.

TABLE III
Response to treatment
(mean scores)

	Scale	*Pre-treatment*	*Week 1*	*Week 2*	*Week 4*	*Week 6*
All cases completing study[1]	HRS	26.5	20.6	16.4	12.1	9.8
Fluoxetine	MADRS	35.8	28.8	23.8	17.6	13.5
$n=22$	LPD	14.5	13.0	11.6	9.7	7.9
	HRS	28.8	19.8	16.1	10.1	6.4
Imipramine	MADRS	37.1	27.7	21.6	13.3	9.4
$n=28$	LPD	14.2	12.2	11.1	9.3	7.5
Endogenous cases completing study	HRS	27.7	20.4	17.1	13.4	11.1
Fluoxetine	MADRS	36.8	27.9	23.9	19.1	15.3
$n=14$	LPD	14.1	12.9	11.1	9.2	7.5
	HRS	28.9	19.5	17.0	10.5	6.5
Imipramine	MADRS	37.1	27.0	22.4	13.6	9.4
$n=15$	LPD	14.3	11.9	11.3	9.3	7.7

1. Fluoxetine vs imipramine t-test (Wilcoxon) = NS

Side-effects

There were no major side-effects and Fig. 1 shows the profile of minor side-effects present in the total sample. None of these were sufficiently troublesome to influence compliance, except in the two cases who withdrew. The only difference was in the existence of tremor, which was four times more common with fluoxetine than with imipramine ($P<0.01$).

Discussion

The results show that fluoxetine is as effective an antidepressant as imipramine and that the rate of response is very similar. Although most clinical trials of antidepressant drugs report improvement in 70–80% of patients, effective cures as measured by achieving low end-point scores on the scales used are about 40–50% of cases treated. This trial produced effective cures in the dosages used in only such a proportion of cases suggesting that 5HT deficiency is not the whole story in the biochemistry of depressive illness. It would be of interest to carry out a cross-over trial in non-responding patients to elucidate whether a subgroup of pure 5HT deficient cases exists.

There were no major side-effects with fluoxetine, and haematological and biochemical screening showed no significant abnormalities. The minor side-effects profile is entirely different from imipramine and confirms that fluoxetine neither produces significant anticholinergic side-effects nor any significant drowsiness, or impairment of function. On the contrary, it appears to produce a slight alerting effect as assessed by the increased feelings of tremor, insomnia and nervousness.

There has been a suggestion that fluoxetine causes weight loss due to suppression of appetite. It was noted that 65% of patients on fluoxetine lost weight, compared with 20% on imipramine, and conversely 65% of patients on imipramine gained weight while only 25% on fluoxetine did so. The mean loss on fluoxetine was 0.63 kg and the mean gain on imipramine was 0.82 kg ($P<0.05$). This finding may be of importance in long-term therapy.

The three methods of clinical assessment were subjected to rank correlation coefficient analysis and Table IV shows the results confirming a very strong positive linear correlation and, therefore, the close comparability not only of the observer rating scales, but also of the self-rating scale with both of them. Previous studies have shown the comparability of MADRS and LPD with HRS and other scales (Kearns *et al*, 1982; Levine, 1975), but this is the first comparison of the LPD scale with MADRS. The suggestion that self-rating scales and observer-based rating scales may be measuring different variables (Prusoff *et al*, 1972) does not seem to be borne out by this study.

TABLE IV
Comparability of rating scales

	(Pearson's Test)
HRS vs MADRS	0.95 ($P=0.0001$)
LPD vs MADRS	0.83 ($P=0.0001$)
LPD vs HRS	0.78 ($P=0.0001$)

References

ASBERG, M., THOREN, P., TRASKMAN, L., BERTILSSON, L. & RINGBERGER, W. (1976) Serotonin depression: a biochemical subgroup within the affective disorders. *Science*, **191**, 478–481.

HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology*, Neurosurgery and Psychiatry, **23**, 56–62.

KEARNS, N. P., CRUICKSHANK, C. A., MCGUIGAN, K. J., RILEY, S. A., SHAW, S. P. & SNAITH, R. P. (1982) A comparison of depression rating scales. *British Journal of Psychiatry*, **141**, 45–49.

LEMBERGER, L., BERGSTROM, R. F., WOLEN, R. L., FARID, N. A., ENAS, G. G. & ARNOFF, G. R. (1985) Fluoxetine: clinical pharmacology and physiological disposition. *Journal of Clinical Psychiatry*, **46**, 3, 14–19.

LEVINE, S. (1975) A controlled comparison of Maprotiline (Ludiomil) with imipramine avoiding observer bias. *Journal of International Medical Resesearch*, **3** (Suppl. 2), 75.

MONTGOMERY, S. A. & ASBERG, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, **134**, 382–389.

PILOWSKY, I. & BOULTON, D. M. (1970) Development of a questionnaire-based decision rule for classifying depressed patients. *British Journal of Psychiatry*, **117**, 685–688.

PILOWSKY, I. & SPALDING, D. (1972) A method for measuring depression: validity studies on a depression questionnaire. *British Journal of Psychiatry*, **121**, 411–416.

——, LEVINE, S. & BOULTON, D. M. (1969) The classification of depression by numerical taxonomy. *British Journal of Psychiatry*, **115**, 937–945.

PRUSOFF, B. A., KLERMAN, G. L. & PAYKEL, E. S. (1972) Concordance between clinical assessments and patients' self-report in depression. *Archives of General Psychiatry*, **26**, 546.

SPITZER, R., ENDICOTT, J. & ROBINS, E. (1975) *Research Diagnostic Criteria*. New York: New York State Psychiatric Institute.

VAN PRAAG, H. M. (1977) Indoleamines in depression. In *Handbook of Studies in Depression* (ed. G. Burrows) Amsterdam: Elsevier.

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Further Studies of Vanadium in Depressive Psychosis

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Three studies are reported. In study 1, vanadium concentration was estimated by neutron activation analysis in hair, whole blood, serum and urine from 13 patients suffering from depressive psychosis and then when on recovery. Vanadium concentration of hair, whole blood and serum decreased significantly with recovery, but there was no significant change in 24-h urinary excretion or in renal clearance of vanadium. In study 2, vanadium concentration was estimated by neutron activation analysis in serum and urine of 31 patients with depressive psychosis and of 27 normal controls. Mean renal clearance of vanadium was significantly lower and mean serum vanadium concentration significantly higher in depressed patients than in controls. Mean 24-h excretion of vanadium did not differ between the two groups. Vanadium excretion did not correlate with urine volume, with serum concentration or with age. In study 3, erythrocyte Na-K ATPase activity and serum vanadium concentrations were estimated in 58 patients. There was a strong negative correlation between the two, supporting the suggestion that changes in tissue vanadium concentration may explain the changes in sodium transport which occur in depressive psychosis.

In depressive psychosis intracellular sodium is raised (Coppen & Shaw, 1963), probably secondary to reduced active transport of sodium across membranes, e.g. plasma to CSF (Coppen, 1960), from saliva (Glen *et al*, 1969), out of erythrocytes (Naylor *et al*, 1973) and across the rectal mucosa (Peet, 1975). In the erythrocyte the reduction in active transport of sodium is probably secondary to reduced activity of Na-K ATPase (sodium potassium sensitive adenosine triphosphatase) (Naylor *et al*, 1973) not to reduction in number of Na-K ATPase molecules (Naylor *et al*, 1980).

It has been suggested that this change in sodium transport is secondary to raised tissue concentrations of vanadium (vanadate ion inhibits Na-K ATPase) in depressive illness (Naylor & Smith, 1981*a*). High vanadium concentrations seem to be of aetiological importance since methods designed to reduce them are antidepressant, e.g. ascorbic acid (Naylor & Smith, 1981*a*), methylene blue (Naylor & Smith, 1981*b*, Naylor *et al*, 1986) and EDTA (Naylor & Smith, 1981*a*; Kay *et al*, 1984) and some drugs used in depressive psychosis (e.g. lithium, carbamazepine) reverse the inhibition of Na-K ATPase by vanadate (Naylor *et al*, 1981; Naylor, 1984). Assay of tissue concentrations of vanadium presents considerable problems primarily because of the very low concentrations present in tissues and hence values reported have varied widely. Many techniques have been used, even enzyme methods, but the two techniques now most widely in use are atomic absorption spectrophotometry and neutron activation analysis. There is also uncertainty about which tissues are best sampled for estimation of trace element status: e.g. some suggest hair but others disagree.

Dick *et al* (1982) reported that plasma vanadium concentration, estimated by atomic absorption spectrophotometry, was significantly higher in depressed patients than in normal controls. Naylor *et al* (1984), using neutron activation analysis to assay vanadium, reported that serum and whole blood vanadium concentrations were higher in a depressed group than in a recovered or normal group. Similarly Conri *et al* (1986) found higher serum vanadium concentrations in depressed patients than in normal controls. In contrast, Ali *et al* (1985) using neutron activation analysis found no difference between whole blood vanadium of depressed, recovered and control subjects. None of these studies used the same patients when ill and on recovery.

Although it has been suggested that changes in vanadium concentration may account for the changes in sodium transport, the only experimental evidence for this is the report of a significant negative correlation between the erythrocyte Na-K ATPase activity and serum vanadium in a small group of short cycle manic depressive patients (Dick *et al*, 1982).

The present paper reports three separate studies.

(1) Change in tissue vanadium concentrations on recovery from depression, using the same patients when depressed and on recovery.

(2) Urinary excretion of vanadium in depressed patients compared with that of normal controls.
(3) Correlation of serum vanadium with erythrocyte Na-K ATPase in depressed patients.

Method

Study 1: Change in tissue vanadium concentrations with recovery from depression

Shortly after admission, physically healthy female patients suffering from a depressive psychosis were admitted to a metabolic ward for at least 4 days before samples were taken. During the time in the metabolic ward patients had a fixed intake of Na, K and calories but a free, though recorded, fluid intake. The only drug they received was nitrazepam 10 mg as night sedation. After the samples were taken, patients returned to the admission ward, where they were treated as necessary by a variety of antidepressant regimes. When they had fully recovered they were again admitted to the metabolic ward for a further 4 days when the regime, including diet, drug treatment, etc was the same as on the first occasion.

Each patient was seen separately for diagnosis by two psychiatrists. All patients fulfilled the Feighner criteria (Feighner *et al*, 1972) for depression and all were given an ICD diagnosis of manic depressive psychosis—depressed type by both psychiatrists. Patients who did not fulfil these diagnostic criteria were excluded. During the time in the metabolic ward, they were rated twice daily by trained nursing staff on a 7-point global scale and on the day of blood sampling they also completed the Beck self rating inventory (Beck *et al*, 1961).

Two samples of fasting venous blood were taken at 09:00, one into sodium heparin and the other into a plain tube. Vacutainers were used to minimise risk of contamination. After the blood in the plain tube had clotted, it was centrifuged and the serum removed and saved. A sample of hair, about the size of the small finger, was taken from the nape of the neck close to the scalp, placed in a polythene bag and saved. A 24-h urine collection was made (patients only had access to a dry toilet), the volume noted, the sample well mixed and aliquots taken. All samples were stored at −20° C until the study was complete and then transported to the laboratory for analysis (i.e. depressed and recovered patient samples were analysed together as one batch).

Study 2: Urinary excretion of vanadium

The patients were severely depressed in-patients either at Royal Dundee Liff Hospital or Murray Royal Hospital, Perth. Patients were interviewed by one of the authors. All patients were suffering from manic depressive psychosis—depressed type (ICD 296.1). All were rated above 6 on the Newcastle Scale (Carney *et al*, 1965) and all fulfilled the Feighner Criteria for Depressive Illness (Feighner *et al*, 1972). Patients with physical illness were excluded from the study. Patients received only nitrazepam for at least 3 days before urine and blood sampling.

The controls were physically healthy, drug-free subjects who were not related to the patients but were mainly, though not only, hospital staff.

A 10 ml sample of venous blood was taken into a plain glass container with no anticoagulant. After clotting, the sample was centrifuged, the serum removed and stored at −20° C for later analysis.

A 24-h collection of urine was made on each subject: urine was collected in polythene bottles with 20 ml of 5% EDTA as preservative. The collection was well mixed, the volume measured and aliquots taken and saved at −20° C for later analysis. Samples from patients and controls were accumulated over the same time. Serum and urine samples were saved until the study was complete. Patient and control samples were analysed together in one batch.

Study 3: Correlation of erythrocyte Na-K ATPase activity and plasma vanadium concentration

A 10 ml sample of venous blood was taken at 09:00 into sodium heparin from each of 58 physically healthy female in-patients. The diagnostic procedure was the same as in the other two studies—all patients fulfilled the Feighner criteria and all were suffering from manic depressive psychosis—depressed type. As in study 1, all patients were maintained in a metabolic ward for at least 4 days before blood sampling and the only drug they received was nitrazepam. In contrast to study 1, only one sample was taken from each patient but patients were at different phases of the illness, i.e. not only acutely depressed and recovered but many were partially recovered.

Blood was centrifuged, plasma removed and Na-K ATPase assayed on the erythrocytes. Plasma was stored at −20° C for later estimation of vanadium concentration: again these were saved until the study was complete and analysed together as one batch.

Biochemical method

Estimation of vanadium concentration

A 5–10 ml urine sample was 'concentrated' to 1 ml under an i.r. lamp (12–24 h) enclosed in a dust-free partial vacuum chamber, placed within a controlled fumehood (Clean Room Construction Ltd) environment. An adequate proof of this technique is shown by the analysis of National Bureau of Standards, Standard Reference Material Bovine Liver (NBS-SRM, 1577) reported elsewhere (Ward *et al*, 1979; Ward, 1983). The 'concentrated' urine or blood serum sample underwent wet destruction of the organic content using a teflon bomb with subsequent ion-exchange separation using hydrated antimony pentoxide (Ward & Ryan, 1979). Neutron activation analysis measurements for vanadium were made after 300-s irradiation, 60-s decay and 300-s count using the ^{52}V nuclide 3.8 min half-life at 1434 keV gamma-ray energy. The detection limit for this method is 0.0002 ug/g. All samples were analysed blind to the clinical classification.

TABLE I

Concentration of vanadium in tissues of patients when ill and on recovery (probability by two-tail 't' test for correlated means)

	Depressed			Recovered			
	n	*m*	*s.d.*	*n*	*m*	*s.d.*	*P*
Hair ng/g	13	48.2	10.0	13	27.2	13.2	<0.002
Whole blood µg/litre	13	7.96	1.62	13	6.38	1.32	<0.02
Serum µg/litre	13	5.20	1.13	13	4.11	0.48	<0.01
24-h urine volume ml	13	1579	796	13	1739	560	NS
Urine concentration µg/litre	13	6.10	1.24	13	5.04	1.05	<0.05
24-h vanadium excretion µg/24-h	13	9.08	3.13	13	8.34	1.81	NS
Renal clearance ml	13	1800	440	13	2060	470	NS

TABLE II

Urine vanadium results in the 31 patients and 27 controls (probability by two-tail 't' test for uncorrelated means)

	Patients			Controls			
	n	*m*	*s.d.*	*n*	*m*	*s.d.*	*P*
24-h urine volume ml	31	1590	694	27	1772	700	NS
Urine vanadium concentration µg/litre	31	5.93	1.50	27	6.24	3.00	NS
24-h vanadium excretion µg	31	8.73	2.45	27	9.82	2.49	NS
Serum vanadium µg/litre	31	5.01	1.16	26	4.30	1.13	<0.05
Clearance (litre)	30	1.82	0.52	26	2.43	0.79	<0.002
Age	31	54.7	12.9	27	50.5	11.2	NS

Erythrocyte Na-K ATPase Activity

Erythrocyte membranes were prepared from washed cells by haemolysis in 10 mmol/litre tris-HCl buffer, pH 7.4 (Dick *et al*, 1969) for estimation of Na-K ATPase activity and were then assayed by a modification of the method of Bonting (1970), whereby the phosphotungstate formed was estimated by extraction into ethylacetate and measured at 310 nm with a UV spectrophotometer. The whole procedure was carried out in duplicate.

Results

Study 1: Change in tissue vanadium concentrations with recovery from depression

Samples were collected from 13 female patients (seven unipolar, six bipolar) mean age 57.6-13.3 years. In all three tissues, (hair, whole blood and serum) the concentration of vanadium decreased signficantly with recovery (Table I). Concentration of vanadium in hair decreased in 11 of the 13 patients, in whole blood in 10 of the 13 patients, and in serum in 12 of the 13 patients. Results were similar in both the unipolar and bipolar group.

The concentration of vanadium in urine on recovery was significantly lower ($P<0.05$) than that when depressed but there was no statistically significant change in urine volume, 24-h urinary excretion of vanadium or renal clearance of vanadium.

There was no significant correlation between measures of depression, e.g. Beck self rating inventory and any of the vanadium estimations.

Study 2. Urinary excretion of vanadium

Samples were obtained from 31 patients (29 females, two males) with mean age 54.7 ± 12.9 years and from 27 controls (24 female, three males) with mean age 50.5 ± 11.2 years.

The results of patients and controls are shown in Table II. There was no significant difference between urine volumes of patients and controls. Mean urinary vanadium concentration of the depressed group (5.93 ± 1.50 ng/ml) was not significantly different from that of the control group (6.24 ± 3.00 ng/ml). Similarly, the total vanadium excreted in 24-h did not differ significantly between the two groups. In contrast, mean serum vanadium concentration was significantly higher in the depressed group (5.01 ± 1.16 ng/ml) than in the control group (4.30 ± 1.13 ng/ml: $P<0.05$). The 24-h vanadium clearance was calculated: there was a highly significant difference between that of patients (1820 ± 520 ml) and that of controls (2430 ± 790 ml: $P<0.002$).

Eleven of the patients were bipolar and 20 unipolar. There was no significant difference between any of the urinary values of the bipolar and unipolar patients.

There was no significant correlation between serum vanadium concentration and urine concentration or 24-h vanadium excretion. There was, however, a strong negative correlation between 24-h urine volume and urine vanadium concentration (normals, $r=-0.61$, $n=27$, $P<0.001$; patients, $r=-0.69$, $n=31$, $P<0.001$).

Study 3. Correlation of erythrocyte Na-K ATPase activity and plasma vanadium concentration

Samples were obtained from 58 female depressed patients and erythrocyte Na-K ATPase activity and plasma vanadium estimated. Mean erythrocyte Na-K ATPase activity was 1.26±0.37 mmol PO4/litre RBC/h and mean plasma vanadium 4.62±1.30 ug/litre. There was a strong negative correlation between plasma vanadium concentration and erythrocyte Na-K ATPase activity ($n=58$, $r=-0.41$, $P<0.002$).

Discussion

The first study confirms earlier studies which had suggested that tissue concentrations of vanadium are raised in depression compared with recovery. All patients were in a metabolic ward for at least 4 days before the assays. Published data on vanadium content of food is conflicting and appears to depend, in part, on where it is grown. It was not possible, therefore, to give patients a known vanadium intake but since patients ate exactly the same diet for 4 days before each assay, the intake of vanadium over that time was the same on each occasion. It is possible, however, that 4 days of controlled diet was not long enough for patients to reach equilibrium. Depressed patients prior to admission are likely to have a reduced dietary intake and moderate weight loss which is reversed on recovery. Whether weight loss can bring about an increase in tissue vanadium concentration and therefore explain the findings in the first study (e.g. by loss of muscle but without excretion.of the released vanadium) is not known but seems unlikely in view of the urinary excretion of vanadium. It is more probable that reduced food intake leading to weight loss is likely to be accompanied by reduced vanadium intake with consequent reduction in tissue concentration.

Previous drug intake of patients also complicates the interpretation of the results. Although patients were drug free, apart from benzodiazepines, for at least 4 days prior to assessment, this may have been inadequate to obviate drug effects. In animals, the biochemical changes induced by psychotropics often persist for 2 weeks or more after administration and it is known that some psychotropic drugs affect vanadium metabolism, e.g. lithium and carbamazepine inhibit vanadium induced inhibition of Na-K ATPase. However, there are ethical difficulties in withholding drugs from severely depressed patients for prolonged periods for the sake of research. Since the first assay was carried out shortly after admission to hospital, the drugs the patient took before admission may be important but it is not only difficult to establish what drugs they were prescribed, it is impossible to determine with certainty which drugs they actually swallowed. As with virtually all clinically based research of this nature it must, therefore, remain a possibility that some of the changes found could be drug-induced. However, it must also remain a possibility that such putative drug-induced changes are the means by which the drugs exert their therapeutic effects.

The changes in tissue concentrations were shown in a tissue which reflects short-term changes, i.e. serum and also a tissue which reflects long-term changes, i.e. hair. Changes in hair must have occurred early after admission since the sample from the recovered patients must have been growing for several weeks before full recovery. It was of interest that, although tissue concentrations changed, there was no significant change in 24-h renal excretion of vanadium. However, if patients were stable and presumably in balance on each occasion, one would not expect the 24-h urine excretion to differ if the intake was the same. The renal clearance did show a non-significant ($P<0.1$) increase with recovery.

In estimating 24-h excretion, accuracy of urine collection is essential. It seems unlikely in the second study that the collections of the patients were any less complete than those of the normals; there was no significant difference between 24-h urine volumes; correlation of vanadium concentration with urine volume was strongly negative in both groups; it was more difficult for normal controls to make a full collection, e.g. while at work, than for patients who only had access to a dry toilet. More important, concentrations of many other elements were estimated (e.g. K, Al, Ba, etc) in the urine and there was no evidence of a consistent trend for patients to have a reduced 24-h urinary excretion of these, i.e. no evidence that 24-h urine collections in the patients were less complete than those of controls. Studies, like the present one, which compare patients with controls must be interpreted with some caution because of the uncertainty of the comparability of the two groups, i.e. controls differ in several ways other than mood from patients, e.g. activity, diet, sleep pattern, etc. However, mean serum concentrations of vanadium in this study (patients 5.01±1.16 ng/ml, controls 4.30±1.13 ng/ml) were remarkably similar to those obtained in a previous separate study (patients 5.09±1.80 ng/ml, controls 4.13±1.13 ng/ml) (Naylor *et al*, 1984).

The results of study 2 suggest that the renal clearance of vanadium is reduced in depressive psychosis, compared with normal controls but no significant change in renal clearance occurred in the first study with recovery although tissue concentrations fell. It is possible that the results of the first study simply failed to reach statistical significance ($P<0.1$) but it appears that renal clearance in the recovered patients was still lower than that of controls. It is possible that the differences were due to differences in diet between patients and controls, since it is not possible to control dietary vanadium but this would more likely affect tissue concentrations than renal clearance: serum concentrations of recovered and control groups were similar although renal clearance still differed. Without further research on the mode of renal excretion of vanadium the present results can only be taken as suggestive that changes in renal excretion of vanadium may occur in manic depressive illness and warrant further investigation. However, there are other possible ways for vanadium concentration of tissues to change, e.g. by change in absorption from the gut, change in dietary intake, change in tissue binding, etc. These possibilities also require investigation.

There is now reasonable agreement that the active sodium transport (measured as transport or Na-K ATPase activity) of erythrocytes is reduced in depressive psychosis and increases with recovery. The suggestion, that changes in vanadium concentrations are involved in this, is strengthened by the finding of a strong negative correlation between plasma vanadium concentration and Na-K ATPase activity. The relatively high correlation with plasma levels was perhaps surprising since vanadate acts on the inside of a cell membrane, not the outside, most intracellular vanadium is in the form of vanadyl which is much less inhibitory of sodium transport and the correlations were on inter- not intra-patient measures.

It is possible that changes in vanadium in depressive psychosis are an unimportant secondary phenomenon but this is unlikely since methods which reduce vanadate concentration appear to be antidepressant (Naylor & Smith, 1981*a*; Kay *et al*, 1984). How vanadium relates to symptoms is uncertain but there are several ways in which it is known to interrelate with neuronal function. Vanadate ion is a potent inhibitor of the sodium pump (Cantley *et al*, 1977) and the distribution of sodium and potassium is involved in the conduction of the nerve impulse and is involved in both release and uptake of amines at synapses. Vanadate is bound directly by noradrenaline and noradrenaline will reverse the vanadium-induced inhibition of the sodium pump. Vanadate ion also directly affects amine metabolism: large doses of vanadium given to rats produce changes in cerebral catecholamines and indolamines (Witkowska & Brzyzwiski, 1979), and vanadium compounds, both pentavalent and tetravalent, directly catalyse oxidation of some catecholamines and of 5-hydroxytryptamine. Depression or melancholia is often given as one of the symptoms of vanadium poisoning (Witkowska & Brzyzwiski, 1979). However, it is not an inevitable symptom of vanadium poisoning and therefore patients with depressive psychosis must have some other abnormality to account for their vulnerability to raised vanadate levels. There have been reports of genetic abnormalities in β-adrenoceptors (Wright *et al*, 1984) in acetyl choline receptors (Nadi *et al*, 1984) and in Na-K ATPase (Naylor & Smith, 1981*c*) in manic depressives which may be of relevance.

This paper offers further evidence that vanadium metabolism is altered in depressive psychosis. The results confirm the suggestion that tissue levels of vanadium fall with recovery from depression and support the suggestion that changes in vanadium concentrations are responsible for the change in sodium transport reported in depressive illness. The results suggest that changes in renal excretion of vanadium may occur. This area of investigation warrants further study.

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References

Ali, S. A., Peet, M. & Ward, N. I. (1985) Blood levels of vanadium, caesium and other elements in depressive patients. *Journal of Affective Disorders*, 9, 187–191.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

Bonting, S. L. (1970) Sodium-potassium activated adenosinetriphosphatase and cation transport. In *Membranes and Ion Transport*, Vol. 1, (ed. E. E. Bittar). New York: Wiley Interscience.

Cantley, L. C., Josephson, L., Warner, R., Yanagisawa, M., Lechene, C. & Guidotti, G. (1977). Vanadate is a potent (Na-K) ATPase inhibitor found in ATP derived from muscle. *Journal of Biological Chemistry*, **252**, 7421–7423.

Carney, M. W. P., Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

Conri, C., Simonoff, M., Fleury, B. & Moreau, F. (1986) Does vanadium play a role in depressive states? *Biological Psychiatry*, **21**, 546–548.

Coppen, A. J. (1960) Abnormality of the blood cerebrospinal fluid barrier of patients suffering from a depressive illness. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 156–161.

—— & Shaw, D. M. (1963) Mineral metabolism in melancholia. *British Medical Journal*, ii, 1439–1444.

Dick, D. A. T., Dick, E. G. & Tosteson, D. L. (1969) Inhibition of adenosine triphosphatase in sheep red cell membranes by oxidised glutathione. *Journal of General Physiology*, **54**, 123–133.

——, Naylor, G. J. & Dick, E. G. (1982) Plasma vanadium concentration in manic depressive illness. *Psychological Medicine*, **12**, 533–537.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

GLEN, A. I. M., ONGLEY, G. C. & ROBINSON, K. (1969) Diminished membrane transport in manic depressive psychosis and recurrent depression. *Lancet*, **ii**, 241–243.

KAY, D. S. G., NAYLOR, G. J., SMITH, A. H. W. & GREENWOOD, C. (1984) The therapeutic effect of ascorbic acid and EDTA in manic depressive psychosis: double blind comparisons with standard treatments. *Psychological Medicine*, **14**, 533–539.

NADI, N. S., NURNBERGER, J. I. & GERSHON, E. S. (1984) Muscarinic cholinergic receptors on skin fibroblasts in familial affective disorder. *The New England Journal of Medicine*, **11**, 225–230.

NAYLOR, G. J. (1984) Reversal of Vanadate Induced Inhibition of Na-K ATPase. *Journal of Affective Disorder*, **8**, 91–93.

—— & SMITH, A. H. W. (1981*a*) Vanadium, a possible aetiological factor in manic depressive illness. *Psychological Medicine*, **11**, 249–256.

—— & —— (1981*b*) Methylene blue. A new treatment for manic depressive psychosis. *IRCS Med Sci*, **9**, 1154–1155.

—— & —— (1981*c*) Defective genetic control of sodium pump density in manic depressive psychosis. *Psychological Medicine*, **11**, 257–263.

——, MCNAMEE, H. B. & MOODY, J. P. (1971) Changes of erythrocyte sodium and potassium on recovery from a depressive illness. *British Journal of Psychiatry*, **118**, 219–223.

——, DICK, D. A. T., DICK, E. G., LE POIDEVIN, D. & WHYTE, S. F. (1973) Erythrocyte membrane cation carrier in manic depressive illness. *Psychological Medicine*, **3**, 502–508.

——, SMITH, A. H. W., DICK, E. G., DICK, D. A. T., MCHARG, A. M. & CHAMBERS, C. A. (1980) Erythrocyte membrane cation carrier in manic depressive psychosis. *Psychological Medicine*, **10**, 521–525.

——, DICK, D. A. T., JOHNSTON, B. B., HOPWOOD, S. E., DICK, E. G., SMITH, A. H. W. & KAY, D. S. G. (1981) Possible explanation for therapeutic action of lithium and a possible substitute—(Methylene blue). *Lancet*, **ii**, 1175–1176.

——, SMITH, A. H. W., BRYCE-SMITH, D. & WARD, N. I. (1984) Tissue vanadium levels in manic depressive psychosis. *Psychological Medicine*, **14**, 767–772.

PEET, M. (1975) The potential difference across the rectal mucosa during depressive illness and lithium therapy. *British Journal of Psychiatry*, **127**, 144–148.

WARD, N. I. (1983) Multi-element analysis of urine by neutron activation analysis. *ICRC Report*, (Available ICRC, Silwood Park, Ascot, Berkshire SL5 7PY).

WARD, N. I. & RYAN, D. E. (1979) Trace analysis of blood by neutron activation analysis. *Analytics Chimica Acta*, **105**, 185–197.

WARD, N. I., STEPHENS, R. & RYAN, D. E. (1979) Comparison of three analytical methods for the determination of trace elements in whole blood. *Analytica Chimica Acta*, **110**, 9–19.

WITKOWSKA, D. & BRZYZWISKI, J. (1979) Alteration in brain noradrenaline, dopamine and 5-hydroxytryptamine levels during vanadium poisoning. *Polish Journal of Pharmacology*, **31**, 393–398.

WRIGHT, A. F., CRIGHTON, D. N., LOUDEN, J. B., MORTON, J. E. N. & STEEL, C. M. (1984) β-adrenoceptor binding defects in cell lines from families with manic depressive disorder. *Annals of Human Genetics*, **48**, 201–214.

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Epidemiology of Puerperal Psychoses

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Computer linkage of an obstetric register and a psychiatric case register made it possible to investigate the temporal relationship between childbirth and psychiatric contact in a population of 470 000 people over a 12-year period resulted in 54 087 births: 120 psychiatric admissions within 90 days of parturition. The 'relative risk' of admission to a psychiatric hospital with a psychotic illness was extremely high in the first 30 days after childbirth, particularly in primiparae, suggesting that metabolic factors are involved in the genesis of puerperal psychoses. However, being unmarried, having a first baby, Caesarian section and perinatal death were all associated with an increased risk of psychiatric admission or contact, or both, suggesting that psychological stresses also contribute to this high psychiatric morbidity. Women with a history of manic depressive illness, manic or depressive, had a much higher risk of psychiatric admission in the puerperium than those with a history of schizophrenia or depressive neuroses, and the majority of puerperal admissions met Research Diagnostic Criteria for manic or depressive disorder. Probably, therefore, puerperal psychoses are manic depressive illnesses and unrelated to schizophrenia.

The effect of childbirth on psychiatric morbidity has been studied many times, and it is well established that the incidence of psychotic illnesses rises dramatically in the first few weeks or months after childbirth, and that such illnesses are commoner in women with a previous history of psychiatric illness, and commoner in primiparae than in multiparae. The magnitude of this increased risk of psychosis, and its duration, have, however, not yet been quantified with any accuracy, nor is it yet clear whether obstetric and demographic variables other than parity influence the risk. Three requirements have to be met before these issues can be settled. First, the puerperal psychoses studied must be drawn from a geographically defined population. It is not sufficient simply to study all women admitted to a particular hospital or mother-and-baby unit unless that hospital or unit has a precisely defined catchment area which it does not share with any other hospital or unit. Secondly, obstetric and other risk factors cannot be identified with any confidence unless accurate information is available about the incidence of these factors in the same geographically defined population and time period in women who do not develop psychiatric disorders. Thirdly, because some of the most important potential risk factors, like perinatal death, are fairly rare events, large populations may need to be studied.

A computer linkage of separate psychiatric and obstetric registers provides an effective means of meeting these three requirements. We have previously reported such a study, involving linkage of the Edinburgh Psychiatric Case Register (EPCR) and Scottish maternity discharge data, based on the 35 729 episodes of childbirth taking place in a geographically defined population of 470 000 over a 7-year period (Kendell *et al*, 1981*b*). The present investigation is an extension and amplification of that 1981 study, based on the 54 087 episodes of childbirth occurring in the same population over an 11-year period. It involves four main improvements or innovations. The larger numbers make it easier to estimate the risks associated with comparatively rare events like perinatal death and twin delivery. The longer time period makes it possible to estimate the 'relative risk' associated with childbirth much more accurately. The combination of larger numbers and a longer time period makes it possible to compare the risk of a puerperal psychosis in women with earlier admissions, unrelated to childbirth, with psychiatric disorders of different kinds. And lastly, because the EPCR was expanded in 1978 to cover all psychiatric contacts as well as all admissions, it is now possible to examine risk factors for out-patient or general hospital contact as well as for psychiatric admission.

Method

The catchment area of the Royal Edinburgh Hospital includes the whole of the city of Edinburgh (population 470 000). The EPCR has recorded data on all admissions from this population since 1 January 1970 and on all psychiatric contacts (as out-patient, domiciliary visit or general hospital ward referral) since 1 January 1978. The Information Services Division of the Scottish Health Service has collected extensive information on all hospital deliveries in Scotland (which in Edinburgh are over 98% of all deliveries) since 1 January 1971. The study covered the 12-year period from 1 January 1970 to 31 December 1981. Information was extracted from the EPCR on all women with a psychiatric admission or other psychiatric contact during this 12-year period who were aged 15–44 inclusive and living within the city at the time of admission or contact. This file was then linked to a similar file extracted from the maternity discharge records, containing data on all episodes of childbirth in any of the five obstetric units in or near Edinburgh during the 11-year period from 1 January 1971 to 31 December 1981 in which the mother's home address was within the city. In this way all Edinburgh women with both a psychiatric contact and an episode of childbirth between 1 January 1970 and 31 December 1981 were identified. The record-linkage system used was the same as that used in the original 1981 study and is described by Heasman & Clarke (1979). It uses surname, maiden name, initials, sex and date of birth as identifying characteristics and the Russell Soundex Code, which makes allowance for common misspellings, for matching. The program actually assigns each pair of women an estimate of the likelihood that the two individuals – the mother and the psychiatric patient – are the same person. There were 154 doubtful matches. These were checked by reference to the original psychiatric and obstetric case-notes and in most cases it was possible to say confidently whether the two belonged to the same individual. Seventy of the 154 were correct matches, 79 were incorrect and 5 remained doubtful. The 70 correct matches were retained and the other 84 discarded.

TABLE I

Temporal relationship between psychiatric admission and childbirth

Time period (successive 90-day periods)		*Number of psychiatric admissions in the three successive 30-day periods*					
		All diagnoses			*Functional psychoses*[1]		
	−8	12	9	9	3	2	2
	−7	14	6	16	4	0	0
	−6	6	16	11	0	3	1
	−5	8	8	6	4	1	3
	−4	11	12	6	2	3	3
	−3	8	5	5	3	0	0
Pregnancy	−2	7	7	8	2	2	2
	−1	8	10	6	2	3	4
	+1	68	39	23	51	25	13
	+2	17	12	12	9	6	2
	+3	17	17	9	10	7	4
	+4	12	17	14	2	4	4
	+5	14	9	19	7	1	7
	+6	15	12	14	4	3	5
	+7	8	11	10	4	3	2
	+8	16	12	6	3	5	0

1. Defined as a discharge diagnosis in ICD-8 and ICD-9 categories 295, 296, 297 and 298 or ICD-8 category 294.4

Temporal relationship between childbirth and psychiatric admission

In order to assess the effect of childbirth on admission rates we examined the distribution of psychiatric admissions in the 2 years before and the 2 years after parturition. As details of psychiatric admissions were available for the 12-year period 1970–1981 inclusive this meant examining the distribution of admissions in women with an episode of childbirth in the 8-year period from 1972–1979. The distribution of the psychiatric admissions of these women was calculated for 24 successive 30-day periods both before and after parturition, counting each episode of childbirth within that 8-year period separately. The results are shown in Table I and Fig. 1(a). As most other investigators have found, and as we ourselves found in our earlier study, there is a dramatic rise in the admission rate in the first few months after childbirth. The average number of admissions a month in the fifteen 30-day periods before the onset of pregnancy is 10.0 and the average number a month in the nine 30-day periods during pregnancy is 7.11. The latter is significantly lower than the former (Mann Whitney $U=33$ and $P<0.05$); that is, as Pugh *et al* (1963) found in Massachusetts, pregnancy is associated with a significant reduction in the admission rate. In the first 30 days after childbirth there are nearly seven times as many admissions as the average monthly admission rate before pregnancy (68 *v.* 10). Although this very high admission rate is not sustained, the admission rate after childbirth remains higher than it was before the onset of pregnancy for at least 2 years. Even if the first 90 days after childbirth are discounted, the average admission rate thereafter (i.e. in the last twenty-one 30-day periods) is significantly higher than that in the fifteen 30-day periods before the onset of pregnancy (13.0/month *v.* 10.0/month; Mann Whitney $U=82.5$ and $P=0.016$). It is also worth noting that the 'excess' of admissions in the first 90 days after childbirth is far greater than the 'deficit' of admissions during pregnancy. In other words, the excess after childbirth is far too great to be explained by the postponement of a proportion of pregnancy admissions.

Table I and Fig. 1b show the number of admissions with a hospital discharge diagnosis (ICD-9 criteria) of some kind of functional psychosis. The effect of childbirth on the psychosis admission rate is even more dramatic than on the overall admission rate. The average rate of psychosis admissions before pregnancy is 2.07/month and the average during pregnancy is 2.0/month, virtually identical. There are then 51 psychosis admissions in the first 30 days after parturition, more than in the whole of the previous 2 years. Although the admission rate falls fairly rapidly thereafter it remains significantly higher throughout the 2-year period after childbirth than it had been before. Even if the first

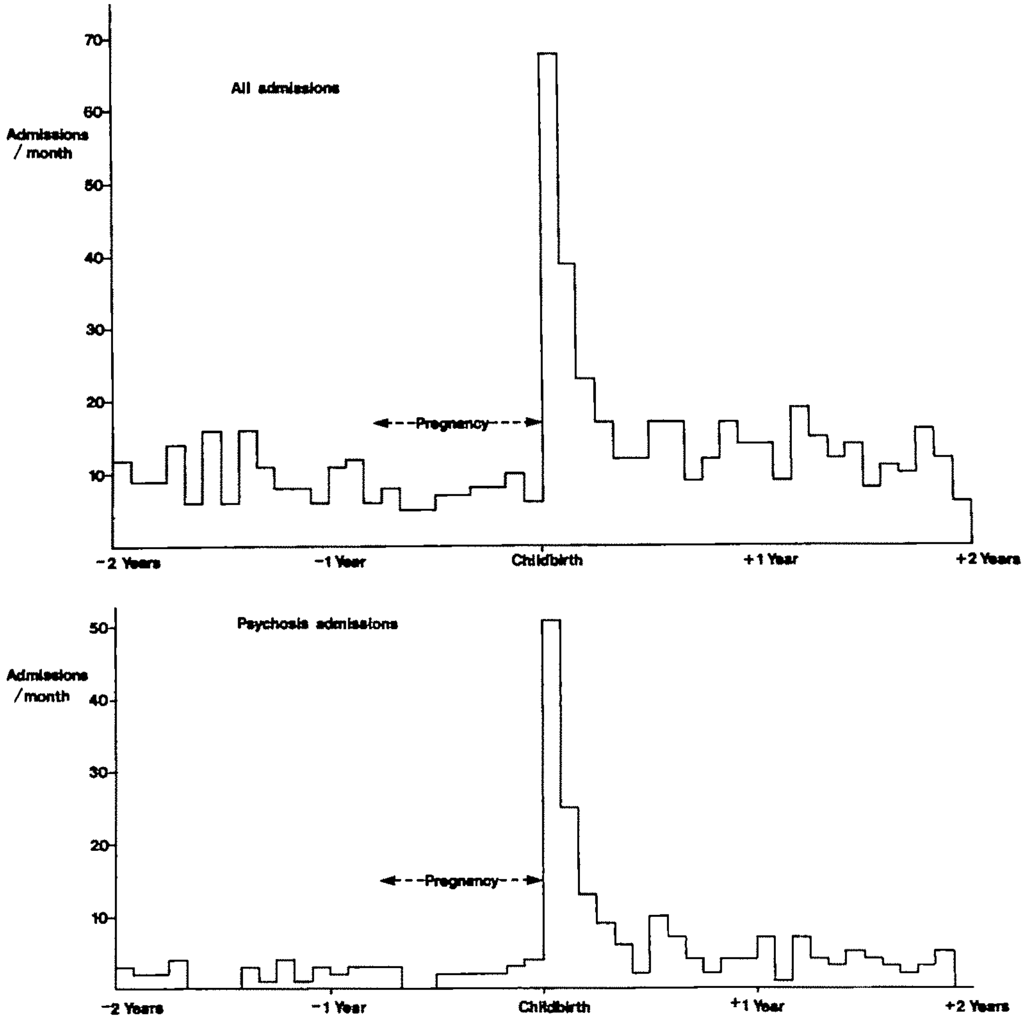

FIG. 1 Temporal relationship between psychiatric admission and childbirth: (a) all admissions; (b) psychosis admissions.

90 days after childbirth are excluded, the average admission rate in the following twenty-one 30 day periods is 4.38/month, compared with 2.04/month in the twenty-four 30-day periods before childbirth (Mann Whitney $U=107$ and $P=0.001$).

The relationship between age and psychiatric admission rate is considered in Appendix 1.

Relative risk associated with childbirth

The ratio of the number of admissions immediately after childbirth to the average admission rate beforehand is a measure of the 'relative risk' associated with childbirth and its magnitude is of considerable importance. In fact the relative risk varies considerably depending on the length of time over which it is measured, on whether one considers all psychiatric admissions or only psychoses, and on whether one considers all mothers or only primiparae. For psychiatric admission the relative risk is $43.3/10.0=4.3$ over the first 90 days, and $68/10.0=6.8$ over the first 30 days. For admission with a psychosis the relative risk is $29.7/2.07=14.3$ over 90 days, and no less than $51/2.07=24.6$ over the first 30 days. These estimates are all somewhat inflated, however, because not all the women living in the register catchment area at the time of delivery would have done so throughout the preceding (or the following) 2 years, and any psychiatric admissions they might have had while living elsewhere would not have been recorded. As a result the average admission rates calculated for the 15 months before pregnancy – the denominator of the ratio from which the relative risk is derived – will be too low. We know from a follow-up study of the women concerned that 18% (21 of the 120, with three untraced) had left the catchment area

TABLE II

Increased risk of psychiatric admission associated with pregnancy and childbirth[1]

	Relative risk[2]	*Attributable proportion*[3]
Admission during pregnancy	0.65	—
Admission with a psychosis during pregnancy	0.88	—
Admission within 90 days of childbirth	3.8	0.74
As above – primiparae only	5.8	0.83
Admission within 30 days of childbirth	6.0	0.83
As above – primiparae only	10.9	0.91
Admission with a psychosis within 90 days of childbirth	12.7	0.92
As above – primiparae only	17.8	0.94
Admission with a psychosis within 30 days of childbirth	21.7	0.95
As above – primiparae only	35.0	0.97
Admission within 2 years of childbirth, excluding the first 90 days	1.3	0.10
As above – primiparae only	1.1	0.09
As above – multiparae only	1.4	0.28
Admission with a psychosis within 2 years of childbirth, excluding the first 90 days	2.1	0.52
As above – primiparae only	1.8	0.46
As above – multiparae only	2.3	0.56
Admission within 2 years of childbirth (including the first 90 days)	1.6	0.38
Admission with a psychosis within 2 years of childbirth (including the first 90 days)	3.5	0.72

1. The base rates, calculated from the average admission rate/month in the 15 months before the beginning of pregnancy and corrected as described in the text were 29.48 admissions per 100 000 women per month and 6.10 psychotic admissions per 100 000 women per month.
2. Ratio of the admission rate in the time period in question to the admission rate of the same population of women before pregnancy.
3. Proportion of admissions in the defined period after childbirth which are directly attributable to the effect of childbirth.

within 2 years of childbirth. If we assume that the same proportion entered the catchment area within the 2 years before childbirth, and did so at a constant rate, both the denominator and the numerator can be corrected for this source of error. This correction reduces the relative risk of admission calculated over the first 30 days by about 12%, and over the first 90 days by about 11%.

The relative risks listed in Table II have all been corrected in this way. Pregnancy is associated with a relative risk of less than 1, and this reduction is more substantial for all admissions than for psychotic admissions. The relative risk of admission is high in the first 90 days after childbirth and higher still in the first 30 days. In this immediate post-partum period it is increased further if only psychotic admissions are considered, and is also considerably higher in primiparae than in multiparae. The effects of shortening the time period from 90 days to 30, of focusing upon primiparae, and of focusing on psychotic admissions are additive, so that the highest relative risk of all – the remarkable value of 35.0 – is the risk for a woman with no previous children of being admitted with a psychotic illness in the first 30 days after childbirth.

These remarkably high relative risks are shortlived, as Fig. 1 illustrates. However, the relative risk remains raised for at least 2 years after parturition. Even if the first 90 days after childbirth are excluded, the relative risk of psychiatric admission is 1.3 (i.e. 30% above the risk before the onset of pregnancy) throughout this 2-year period and the relative risk of admission with a psychotic illness is higher still at 2.1 (i.e. 110% above the risk before pregnancy). And at this stage the relative risk is higher in multiparae than in primiparae rather than the other way about, both for admission and for admission with a psychosis (see Table II).

(All these calculations of relative risk ignore the fact that some women would have had more than one episode of childbirth during the 4-year period of study. Second episodes of this kind would be equally common before and after the index episode and would tend to elevate the admission rate, particularly in the following 3 months. The overall effect of this complication would be to reduce the before/after difference; that is, the true relative risks will be somewhat *higher* than the values in Table II suggest.)

Temporal relationship between childbirth and psychiatric contact

Because the EPCR only started to record psychiatric contacts other than admissions (i.e. out-patient contacts, general hospital ward referrals and domiciliary visits) on 1 January 1978, data were only available for the 4 years 1978–1981. For this reason we only examined the distribution of 'new contacts' (defined as any psychiatric contact other than admission without any other psychiatric contact in the previous 90 days) over the 12 months before and the 12 months after childbirth. Even this limited analysis could only be based on 2 years' births; that is, episodes of childbirth in 1979 and 1980. The results are shown in Table III and the general pattern is the same as it was for admissions. The average number of new contacts a month in the twelve 30-day periods before parturition is 7.08. The monthly average is higher before pregnancy than during pregnancy (8.67 *v.* 6.56) but the difference is not significant. The number of new contacts a month rises sharply after childbirth. The rise is not so dramatic as for psychiatric admissions, but even so there are more new contacts in the first 90 days after childbirth than in the whole 9 months of pregnancy. Furthermore, as for admissions, the new contact rate remains higher than it was before childbirth even after this first 90 days. The average number of new contacts a month in the last nine 30-day periods

TABLE III
Temporal relationship between new psychiatric contacts (other than admissions) and childbirth

Time period (successive 90-day periods)		*Number of new contacts in the three successive 30-day periods*		
	−4	12	7	7
	−3	6	10	7
Pregnancy	−2	3	5	10
	−1	6	6	6
	+1	25	19	16
	+2	7	9	9
	+3	16	10	10
	+4	8	7	8

is 9.33, compared with 7.08 in the twelve 30-day periods before childbirth (Mann Whitney $U=25$ and $P<0.05$).

Risk factors for psychiatric admission or contact in the puerperium

At present there is no consensus on the maximum interval between childbirth and psychiatric presentation during which an illness should be designated as puerperal. Some authors, like Tetlow (1955) and Paffenbarger (1964), accepted as puerperal any illness leading to hospital admission within 6 months of delivery. Others, like Brockington *et al* (1982), have argued that the term should be restricted to illnesses starting within 2 or 3 weeks of delivery. The evidence presented in Tables I and III and Fig. 1 strongly suggests that 90 days is the most appropriate cut-off point if the time of psychiatric admission or contact is to be the criterion. Although the admission rate is highest of all in the first 30 days after childbirth and falls steeply thereafter, it is still higher between 60 and 90 days than at any time during the 2 years before childbirth and any time in the subsequent 21 months. Moreover, this is so not only for psychiatric admissions as a whole, but for psychoses on their own, and for psychiatric contacts as well. Between 90 and 120 days, however, the admission rate is no higher than in several later months, and again this is so for psychoses and psychiatric contacts as well. For this reason, as in our 1981 study, we define the puerperium as lasting for 90 days and therefore looked for risk factors for admission within that 90-day period.

In the 11-year period 1971–1981 inclusive, 120 episodes of childbirth were followed within 90 days by the mother's admission to the Royal Edinburgh Hospital without that woman having been a psychiatric patient within the last 90 days of her pregnancy. (These 120 episodes of childbirth involved 116 different mothers; four had two puerperal admissions within this 11-year period. For 74 of the 116 it was their first psychiatric admission; 34 had had previous non-puerperal admissions, three had had previous puerperal admissions and four had had both puerperal and non-puerperal admissions previously.) These 120 puerperal admissions came from the 54 087 births to women living in the city of Edinburgh within that 11-year period. The overall risk of puerperal admission is therefore 2.2 per 1000 births.

The RDC diagnoses of these 120 admission are shown in Table IV. As most others who have used RDC (Spitzer *et al*, 1978) have found, the majority of patients have affective disorders, either depressive or manic, and patients fulfilling criteria for schizophrenia are rare. There are, however, a substantial number of patients with illnesses which are psychotic but which do not fulfil criteria for any recognised disorder, and so have to be assigned to the residual category of unspecified functional psychosis.

The time of onset of the symptoms of these 120 patients relative to childbirth is shown in Table V. In early half the onset was within the first week after childbirth and in nine the onset had occurred before childbirth, though in most cases with a further exacerbation afterwards. There was no strong tendency for the onset to occur on any particular post-partum day. The third day (12 patients) and the seventh (13 patients) were the commonest, so our previous suggestion (Kendell *et al*, 1981*a*) that the mood disturbance on the fifth post-partum day coincides with the peak time of onset of puerperal psychoses is not supported. Nor was a 'lucid interval' always present; in eight patients the onset was on the first or second post-partum day. (The 71 patients in this series who were admitted to hospital in the 7-year period 1971–1977 inclusive have been described in detail previously (Dean & Kendell, 1981).)

TABLE IV
RDC[1] *diagnoses of the 120 puerperal admissions*

Major depressive disorder (28 definite, 18 probable)	46
Minor depressive disorder (16 definite, 4 probable)	20
Manic disorder (all definite)	22
Schizoaffective disorder (5 manic, 3 depressed)	8
Schizophrenia (3 definite, 1 probable)	4
Unspecified functional psychosis	13
Personality disorder (1 definite, 2 probable)	3
Other or unknown	4

1. Research Diagnostic Criteria of Spitzer *et al* (1978)

TABLE V
Time of onset of symptoms relative to childbirth

During pregnancy	9
1–7 days after childbirth	54
8–15 days after childbirth	12
16–30 days after childbirth	21
31–60 days after childbirth	17
61–90 days after childbirth	7

TABLE VI

Social and obstetric variables associated with a significantly increased or decreased risk of psychiatric morbidity

	Controls 1971–1981 (n = 54 087)	*All admissions (n = 120)*	*Psychotics (n = 63)*	*Onset within 15 days (n = 66)*	*Cycloid psychoses (n = 16)*	*Controls 1978–1981 (n = 18 358)*	*Contacts (n = 50)*
Marital state							
Single	4033	13	7	11**	4	1430	4
	7.5%	10.8%	11.1%	16.7%	25.0%	7.8%	8.0%
Widowed, separated or divorced	1328	10**	3	2	0	687	5*
	2.5%	8.3%	4.8%	3.0%	0%	3.7%	10.0%
Not currently married	5361	23**	10	13*	4	2117	9
	9.9%	19.2%	15.9%	19.7%	25.0%	11.5%	18.0%
Past obstetric history							
History of spontaneous abortion[1]	5179	9	2*	2*	2	3016	11
	16.3%	10.7%	4.1%	4.3%	18.2%	16.4%	22.0%
No previous pregnancies	21122	56	31	42**	11*	7031	21
	39.1%	46.7%	49.2%	63.6%	68.8%	38.3%	42.0%
No living children	25392	69*	39*	47**	12*	8676	26
	46.9%	57.5%	61.9%	71.2%	75.0%	47.3%	52.0%
This delivery							
Female child	26378	64	41*	34	10	8991	27
	48.8%	53.3%	65.1%	51.5%	62.5%	49.0%	54.0%
Caesarian section	5212	17	11	9	1	2230	15**
	9.6%	14.2%	17.5%	13.6%	9.1%	12.1%	30.0%
Perinatal death	839	6*	1	1	0	216	3*
	1.6%	5.0%	1.6%	1.5%	0%	1.2%	6%

*$P<0.05$ and **$P<0.01$. All P values are two-tailed and based on the χ^2 test (with Yates' modification) or, if $E<5$, on Fisher's exact test.

[1] Only recorded in 1975–81.

As extensive obstetric and demographic data (known locally as SMR2 data) are recorded on the maternity discharge sheet for all episodes of childbirth, it was possible to compare the incidence of a wide range of potential risk factors in the 120 puerperal admissions and the remaining 53 967 births which were not followed by psychiatric admission. The results are presented in Tables VI and VII. As can be seen from Table VI, parallel comparisons were also carried out for three subgroups of the 120 admissions: (a) those with psychotic symptoms (defined as hallucinations, delusions, incoherent or grossly thought-disordered speech, mutism or consistently bizarre behaviour); (b) those whose illness started within the first 15 days after childbirth; and (c) those who fulfilled the criteria of Perris & Brockington (1981) for a cycloid psychosis. These three subgroups were not mutually exclusive. In fact 40 women had psychotic symptoms as well as an onset within 15 days of childbirth, and all the cycloid women were also psychotic by definition. Membership of these subgroups was determined – as were the RDC diagnoses and the timing of the onset of symptoms – by detailed examination of the original medical and nursing case-notes. This means, of course, that some women who perhaps should have been assigned to one or more of these subgroups were not because the relevant information was not recorded in their notes.

During the 4 years 1978–1981 inclusive there were 50 psychiatric contacts by women with a home address in the city of Edinburgh which occurred within 90 days of childbirth, were not followed by admission to hospital and had not been preceded by any psychiatric contact within the last 90 days of pregnancy. The role of the potential risk factors was examined in these 50 women in the same way as in the 120 in-patients, except that the control population was restricted to the 18 358 births to Edinburgh women occurring in the 4-year period 1978–1981.

Table VI lists all the statistically significant differences obtained between any of these five patient groups and the controls. Several of them involve marital status. There is an excess of widowed, separated or divorced mothers both in admissions and in contacts, and an excess of single mothers in admissions with an onset of symptoms within 15 days of childbirth. Mainly as a consequence of these differences, currently married women are significantly under-represented in the 'all admissions' and 'onset within 15 days' groups. Strangely, the obstetric register does not record parity as such, though it does record the number of living children and the number of previous pregnancies. The group of all admissions and each of its three subgroups show a significant excess of mothers with no living children. The cycloid psychosis and onset within 15 days subgroups also contain a significant excess of women with no

TABLE VII

Social and obstetric variables not associated with a significantly increased or decreased risk of psychiatric morbidity

Variable	*Controls 1971–1981 (n = 54 087)*	*All admissions (n = 120)*	*Controls 1978–1981 (n = 18 358)*	*Contacts (n = 50)*
Age of mother	25.77 (±5.34)	26.4 (±5.04)	26.23 (±5.29)	27.02 (±4.94)
Past obstetric history				
Therapeutic abortion[1]	2213 (6.9%)	8 (9.5%)	1400 (7.6%)	3 (6%)
Caesarian section	2049 (3.8%)	4 (3.3%)	888 (4.8%)	5 (10%)
Perinatal death[1]	1674 (3.1%)	7 (5.8%)	423 (2.3%)	1 (2%)
Gestation period (weeks)	39.38	39.36	39.34	38.92
Outcome of pregnancy				
Birth weight of baby (kg)	3.30 (±0.58)	3.31 (±0.46)	3.33 (±0.58)	3.22 (±0.64)
Multiple births	551 (1.0%)	1 (0.8%)	379 (2.1%)	0
Breech delivery[1]	422 (1.3%)	0	206 (1.1%)	1 (2%)
Forceps delivery	9496 (17.6%)	18 (15.0%)	2819 (15.4%)	6 (12%)
Sterilisation afterwards[1]	522 (1.6%)	1 (1.2%)	203 (1.1%)	2 (4%)
Complications of pregnancy[2]				
Ante-partum haemorrhage	1448 (3.2%)	4 (3.9%)	—	—
Anaemia of pregnancy	1259 (2.8%)	0	—	—
Malposition *in utero*	365 (0.8%)	2 (1.9%)	—	—
Hydramnios	387 (0.9%)	1 (1.0%)	—	—
Pyelitis/pyelonephritis	1130 (2.5%)	3 (2.9%)	—	—
Pre-eclampsia	4480 (10.0%)	7 (6.8%)	—	—
Complications of delivery[2]				
Post-partum haemorrhage	2015 (4.5%)	3 (2.9%)	—	—
Malpresentation of foetus	1032 (2.3%)	2 (1.9%)	—	—
Prolonged labour	1731 (3.9%)	4 (3.9%)	—	—
Perineal laceration	1321 (3.0%)	2 (1.9%)	—	—
Puerperal sepsis	1454 (3.3%)	2 (1.9%)	—	—
Puerperal anaemia	919 (2.1%)	2 (1.9%)	—	—

1. These items were only recorded in 1975–1981.
2. These items were only recorded in 1971–1979. For this reason it was not feasible to compare 1978–1981 contacts with controls.

The three subgroups of admissions (those with psychotic symptoms, those with an onset within 15 days, and cycloid psychoses) were also compared with the controls on all the variables shown here but none of the differences was statistically significant.

previous pregnancies. A strikingly high proportion of the psychiatric contacts followed Caesarian section. In our 1981 study, psychiatric admissions had also shown a statistically significant excess of Caesarian sections. This time they failed to do so, though the psychotic subgroup came close ($\chi^2 = 3.58$ and $0.1 > P > 0.05$ with 1 d.f.). Both elective sections and operations after a trial of labour contributed to the observed excess. There was a significant excess of perinatal deaths (i.e. stillbirths and neonatal deaths combined) in both admissions and contacts. There were also two quite unexpected findings. There was a significant excess of female babies in the psychotic admissions, and an incidence of prior spontaneous abortion significantly *below* chance expectation in both the psychotic and onset within 15 days subgroups of admissions.

Obstetric and other variables for which there was no statistically significant difference between the controls and any of the five patient groups are listed in Table VII. The mother's age, the length of gestation, the baby's birth weight, most aspects of the mother's obstetric history and a wide range of complications of pregnancy and delivery appear to have no influence on the risk of psychiatric morbidity in the puerperium. Nor do twin births or sterilisation after delivery.

Influence of previous psychiatric illness

The linkage of the EPCR and the maternity discharge (SMR2) data described above identified 486 women who between 1 January 1971 and 31 December 1981 had had a psychiatric admission before their first episode of childbirth (though they might have had other children before the obstetric register came into being on 1 January 1971). The existence of this large number of women with psychiatric illnesses severe enough to warrant hospital admission before childbirth made it possible to estimate the risk of developing a puerperal psychosis (defined as a psychiatric admission within 90 days of parturition) after subsequent pregnancies and to compare that risk in different diagnostic categories. The results are shown in Table VIII. It is clear that different diagnostic categories are associated with quite different risks, and that these are highest for manic/depressive illnesses (21.4% of deliveries in women with prior manic or circular illnesses being followed by a

TABLE VIII
Incidence of psychiatric admission in the puerperium in women with a prior history of psychiatric disorders of different kinds

Diagnostic category	*ICD-9 code*	*Number of women*	*Episodes of childbirth*	*Puerperal admissions*	*Risk of admission (%)*
Schizophrenia	295	22	29	1	3.4
Manic depressive: depressed	296.1	23	30	4	13.3
Manic depressive: manic or circular	296.0 296.2–5	10	14	3	21.4
Depressive neurosis	300.4	79	103	2	1.9
All other diagnosis	—	352	462	19	4.1
Totals	—	486	638	29	4.5

psychiatric admission, and 13.3% of deliveries in women with previous depressive illnesses). The difference between the risk conferred by a previous manic depressive illness (ICD-9 296.0–5) and that conferred by other diagnostic categories is highly significant ($P = 0.00008$). So too is the difference between the risks conferred by manic depressive and schizophrenic illnesses ($P = 0.026$), and between those conferred by manic depressive illnesses and depressive neuroses ($P = 0.0005$). (All probabilities are two-tailed and derived by Fisher's Exact Test.) On the other hand the risks associated with schizophrenia and with depressive neuroses do not differ appreciably from those associated with the residual group of 'other diagnoses' (mainly neurotic illnesses, personality disorders and adjustment reactions). Even so, the risk associated with a prior psychiatric admission for any disorder other than a manic depressive illness (22 of 594, or 3.7%) is many times higher than the risk of 2.2 per 1000 births associated with childbirth as a whole. (Note that all these risks are based on the assumption that no-one either entered or left the register catchment area between their original psychiatric admission and their subsequent deliveries. They therefore provide only rough estimates of absolute risks, but should still give accurate estimates of comparative risks.)

Discussion

One of the main purposes of this study was to identify obstetric and social factors associated with an increased, or decreased, risk of psychiatric illness in the puerperium in the hope that the nature of these risk factors would shed light on the aetiology of puerperal disorders. It is already well established that primiparous women are at increased risk of puerperal psychoses and we demonstrated in our 1981 study that this finding could not be attributed simply to the avoidance of further pregnancies by women who had suffered a puerperal psychosis after their first. This heightened risk in primiparae is confirmed once more. All admissions and each of the three subgroups of these (i.e. psychotic illnesses, illnesses with an onset within 15 days of childbirth and cycloid psychoses) showed a statistically significant excess of mothers with no living children. Psychiatric contacts, on the other hand, did not.

There are also important relationships with marital status. Many investigators from Esquirol (1845) onwards have reported that puerperal psychoses are commoner in unmarried mothers, and our 120 psychiatric admissions contained a highly significant excess of women not currently married, though mainly because of an excess of widowed, divorced or separated women rather than of single women. The fact that the subgroup of admissions with psychotic illnesses does not contain any significant excess of unmarried women suggests that this might simply be a nosocomial effect; that is, that puerperal admissions contain a high proportion of unmarried mothers simply because women living alone with no husband to support them are more likely to be admitted to hospital if they become psychiatrically disturbed in the puerperium. However, the fact that psychiatric contacts also show a significant excess of widowed, divorced or separated women makes this unlikely. If women without husbands are admitted to hospital preferentially, psychiatric contacts should, if anything, show a deficit rather than an excess of mothers not currently married. All admissions, and each of their three subgroups, also contain a higher proportion of single women than the controls, though this is only statistically significant for those with an onset within 15 days. None of these relationships between psychiatric morbidity and marital status can be explained as a secondary consequence of age differences between patients and controls, for there were no age differences.

In our 1981 study we found Caesarian section to be a risk factor for psychiatric admission and for the most severely ill patients this relationship was significant at the 1% level. The fact that this relationship disappears with the larger numbers available here suggests that it may have been a chance finding. This is unlikely to be so, however, because there is, quite independently of that earlier finding, a highly significant excess of Caesarian sections in the psychiatric contacts. Moreover, the excess of Caesarian sections in the psychotic subgroup of admissions only just fails to achieve statistical significance ($P<0.10$).

The significant excess of perinatal deaths in both admissions and contacts is an important finding which did not emerge in our previous analysis, almost certainly because at that stage we had insufficient numbers for significant relationships with rare events like perinatal death to emerge. The relationship has, however, been reported before, by both Vislie (1956) and Paffenbarger (1964). Twin delivery, though, still fails to emerge as a risk factor. The disproportionate number of female children in all five patient groups is puzzling. Despite the fact that it reaches statistical significance in the psychotic subgroup of admissions, this is almost certainly a chance finding, for it has not been found in previous studies of puerperal psychoses. The significant *deficit* of mothers with a past history of spontaneous abortions in the psychotic and onset within 15 days subgroups of admissions is also unexpected, but cannot be dismissed so easily. It may, of course, be a chance finding, for it has not been reported before and the relationships are only significant at the 5% level. However, previous investigators have not had sufficiently accurate control data to be capable of identifying any such relationship, so the possibility that a hormonal environment which protects against abortion somehow increases the risk of puerperal psychosis, or vice versa, should not be dismissed.

We conclude, therefore, partly on the basis of these results and partly on the basis of the literature (reviewed by Kendell, 1985) that having a first baby, not having a husband at the time of childbirth, perinatal death and Caesarian section all increase the risk of serious psychiatric morbidity in the puerperium. All four of these situations were confidently identified by a representative group of 85 normal postpartum women as increasing the psychological stress of childbirth (see Kendell *et al*, 1981*b*), which suggests that psychological stresses of readily understandable kinds play a part in the genesis of all or most of these puerperal disorders. We deliberately refer to puerperal psychiatric disorders in general because the data summarised in Table VI do not suggest that the correlates of psychiatric admission are particularly different from those of psychiatric contact, or that in-patients with psychotic symptoms or with an onset within 15 days of childbirth are obviously different from in-patients as a whole. Although there is no evidence from these data that the incidence of perinatal death is raised in women with psychotic illnesses, or that primiparae are at increased risk of psychiatric contact, the general pattern of relationships is much the same across all five patient groupings. However, in addition to these indications that psychological influences are important, there is very substantial evidence that constitutional factors are involved in the genesis of puerperal psychoses, and probably also of puerperal depressions. Almost everyone who has studied the issue has found that a previous personal history or a family history of psychotic illness increases the risk of puerperal psychosis, and also that the occurrence of one puerperal psychosis increases the risk of a second – from 1 in 500 to 1 in 7 or even less (see Kendell (1985) for a summary of this evidence). The evidence is less clear-cut in the case of milder puerperal depressions, but here too most investigators have reported that a past history of psychiatric disorder increases the risk.

The large number of births and the comparatively long time period studied here has made it possible to calculate fairly accurate admission rates before and during pregnancy and at various intervals after childbirth. From these rates reasonably accurate estimates of the 'relative risk' associated with pregnancy and childbirth can therefore be calculated. As Pugh *et al* (1963) found in Massachusetts, the admission rate during pregnancy is significantly lower than the admission rate over the previous 15 months, with a relative risk of only 0.65. However, there is little fall during pregnancy in the admission rate for psychotic illnesses. This suggests that the fall in the overall admission rate may be a nosocomial effect rather than the result of any real reduction in psychiatric morbidity. That is, it seems likely that, whereas women who develop psychotic illnesses during pregnancy are still admitted to hospital, those with milder disorders are not, perhaps because the patient herself or her doctor hopes that she will recover spontaneously after delivery. Certainly prospective clinical studies suggest that overall psychiatric morbidity rises rather than falls during pregnancy (e.g. Kumar & Robson, 1984).

The 'relative risks' for psychiatric admission after childbirth and the corresponding 'attributable proportions' listed in Table II are far higher than those calculated in other settings for the contribution of psychological stresses to the genesis of psychiatric disorders. The relative risks of various events for depressive, neurotic and schizophrenic illnesses reviewed by Paykel (1978) varied from 1.6 to 7.6, and those calculated more recently for affective disorders in the community by Surtees *et al* (1986) were within the same range. Parasuicide is the only psychiatric phenomenon for which psychological stresses have been shown to have a higher relative risk – of 10.0 by Paykel *et al* (1975) – and even this is quite modest compared with the values of up to 35 obtained here. The most likely explanation of this difference is that childbirth is not acting primarily as a psychological stress. The relative risk is far higher, and the duration of that increased risk more shortlived, because the mechanism responsible is metabolic rather than psychological. Other explanations are possible, however. The magnitude of any estimate of relative risk will be influenced by the length of time over which it is estimated, and in general the shorter the time period the higher the relative risk. Most of the estimates reviewed by Paykel were based on periods of 3–9 months, considerably longer than the 30- and 90-day periods considered here. Perhaps significantly, the high relative risk of 10.0 for the role of life events in the genesis of parasuicide obtained by Paykel *et al* (1975) was based on a time period of only one month. Childbirth also differs from most of the other events known, or suspected, to contribute to the aetiology of psychiatric disorders in occurring at a precise time, in affecting only one person, and in having its occurrence routinely recorded for the population as a whole. These unique advantages obviously make it easier to calculate relative risks accurately than is possible for other events such as bereavement or becoming unemployed. It is possible, therefore, though we ourselves do not think it likely, that technical differences of this kind may be responsible for the striking difference in the magnitude of the relative risks associated with childbirth and other stressors.

This evidence of a dramatic increase in the incidence of psychiatric disorder in the first 90 days after childbirth should not lead us to overlook the fact that the psychiatric admission rate remains significantly higher than it was before pregnancy for at least 2 years after childbirth. The psychiatric contact rate also remains significantly raised for at least 1 year. Because only 4 years' data were available for analysis it was not possible to calculate accurate relative risks for psychiatric contacts, but the relative risk for psychiatric admission in the 2 years after childbirth was 1.6 and the relative risk for psychotic admission 3.5 (see Table II). This means that childbirth makes an appreciable impact on the long-term as well as on the short-term risk of psychiatric admission. If a woman has three children during the 20 years of her active reproductive life (and assuming that her 'base rate' remains stable during that period), her risk of having a psychiatric admission during this 20-year period relative to her risk had she remained childless is at least $[(1\times14)+(1.6\times2\times3)]/20=1.18$, and the relative risk of her being admitted with a psychotic illness is at least $[(1\times14)+(3.5\times2\times3)]/20=1.75$. This means that having three children increases her risk of being admitted to a psychiatric hospital at some time in this 20-year period by at least 18%, and her risk of being admitted with a psychotic illness by at least 75%, and these are minimum values because the period of increased risk may well last for longer than 2 years after each birth.

It is not clear why the risk of psychiatric admission should remain elevated for so long after childbirth. Relapses of puerperal illnesses will make some contribution, but only in the early months after delivery. The birth of a child is, of course, often associated with other important changes in a woman's life. These may involve the loss of a job, of financial independence, of social contacts and of freedom, and a changed marital relationship, and any or all of these may be important stresses. However, such changes are much more likely to occur after the birth of the first than after the birth of later children and, as the relative risks listed in Table II show, after the first 90 days the persisting risk of admission is greater for multiparae than primiparae. If equally extensive data had existed for all psychiatric contacts (i.e. as out-patients or general hospital ward referrals) as were available for psychiatric admissions it would have been possible to calculate how great a contribution having two or three children made to a woman's risk of having any kind of psychiatric contact during her reproductive years. It is a pity this was not possible, for in most discussions of the reasons for women having a higher psychiatric morbidity than men, and for married women having a higher morbidity than single women, surprisingly little attention is usually given to the effects of childbirth. In a previous study based on a small cohort of 2257 women, 'new psychiatric contacts', three-quarters of which were as out-patients, were actually more frequent in the second year after childbirth than in the first, suggesting that the risk of psychiatric contact may remain raised for as long, and be raised just as much, as that of psychiatric admission (Kendell *et al*, 1976).

Social factors are usually invoked to explain the difference in psychiatric morbidity between married and unmarried women, but the findings discussed above suggest that much of the excess morbidity in married women may be attributable to childbirth (and its secondary social consequences). This possibility is supported by the findings of a recent study in north Edinburgh of the prevalence of psychiatric symptoms in the community (Surtees *et al*, 1983). As the prevalence rates set out in

TABLE IX

Distribution of psychiatric cases in a population sample of Edinburgh women of childbearing age[1]

Marital status	*Psychiatric cases* *Number of children* *None*	*One*	*Two*	*Three*	*Total*
Single	8/95 (8%)	0/7	0/2	—	8/104 (8%)
Married	5/78 (6%)	8/59 (14%)	9/66 (14%)	3/19 (16%)	25/222 (11%)
Widowed, separated divorced or co-habiting	5/28 (18%)	7/13 (54%)	4/9 (44%)	4/9 (44%)	20/59 (34%)
Total	18/201 (9%)	15/79 (19%)	13/77 (17%)	7/28 (25%)	53/385 (14%)

[1] These 385 women are a representative sample of women aged 18–45 in a female population of 91 000 in north Edinburgh (see Surtees *et al*, 1983). Psychiatric cases were defined by the Research Diagnostic Criteria of Spitzer *et al* (1978).

Table IX show, the higher morbidity of married women than of single women is entirely due to those with children, for the morbidity of childless married women is actually slightly lower than that of single women. There is, however, no evidence from these data that having two or three children is associated with any higher morbidity than having one.

Although the findings presented here cannot settle the vexed question of how puerperal psychosis should best be defined, they can help to clarify the issues involved. If puerperal psychoses are to be defined in terms of the time interval between parturition and admission to a psychiatric hospital, cogent evidence is presented here that the maximum interval between the two should be 3 months. Brockington *et al* (1982) have argued that the interval between parturition and the onset of symptoms is more important, and that this should not be longer than 2 or 3 weeks. There is no doubt that its time of onset is a more fundamental characteristic of an illness than the time at which it results in admission to hospital. The time of onset is, however, not always easy to determine. Cernik and Brockington achieved an inter-rater reliability of only 0.61 (Cohen's Kappa) in a series of over 100 patients with puerperal illnesses (Brockington *et al*, 1982). Moreover, it is not uncommon for women to become mildly depressed or anxious in late pregnancy and then to deteriorate suddenly and develop psychotic symptoms a week or two after delivery. Their psychosis is puerperal but their original mood disturbance was not.

In general, the time of onset of symptoms will probably be the better criterion in clinical studies, while the timing of psychiatric admission or contact may be the best or the only criterion available in epidemiological studies. In practice, however, there may be little difference between the two criteria discussed here, namely an *onset* within 3 weeks of childbirth and *admission* within 90 days of childbirth. Only 45 (38%) of the 120 patients admitted within 90 days of childbirth described here had an onset more than 15 days after childbirth, and only 24 (20%) an onset more than 30 days afterwards.

The finding that the increase in the risk of psychiatric admission in the puerperium associated with a history of a previous psychiatric admission varies considerably with the diagnosis made at the time of that admission is one of the most interesting and novel findings of this study. A previous manic depressive illness, either depressive or manic, involves a much higher risk than a previous schizophrenic illness or a previous depression diagnosed as a depressive neurosis (ICD-9 300.4). Indeed, these latter two diagnoses carry no greater risk than earlier admissions with a diagnosis of personality disorder or adjustment reaction. Other investigators (e.g. Bratfos & Haug, 1966; Reich & Winokur, 1970) have already shown that a previous manic depressive illness greatly increases the risk of a puerperal psychosis, but as this was demonstrated in the context of a follow-up study of manic depressive patients it was not possible for these researchers to determine whether the risk was restricted to manic depressive illnesses or shared with other disorders. The results obtained here show that the risk is considerably greater after a manic depressive illness than after any other psychiatric disorder. This adds further weight to the existing clinical evidence (the fact that most puerperal psychoses meet research criteria for affective disorders) and genetic evidence (e.g. Whalley *et al*, 1982) that the majority of puerperal psychoses are simply manic depressive illnesses precipitated by childbirth. Conversely, the fact that an earlier schizophrenic illness was not associated with an increased risk of puerperal admission, coupled with the fact that only four of the 120 puerperal admissions met RDC for schizophrenia, adds to the evidence that, despite their sometimes floridly psychotic presentation, the great majority of puerperal psychoses are unrelated to schizophrenia. It is important to appreciate, too, that childbirth need not have played any role in the aetiology of those four schizophrenic illnesses despite the fact that they resulted in admission to hospital within 90 days of parturition. As the 'attributable proportion' listed in Table II shows, 26% of admissions within 90 days of childbirth (100% minus 74%) are not attributable to that event, and four patients constitute only 3.3% of 120. In fact, two of the four mothers with schizophrenic illnesses were already chronic schizophrenics before their index pregnancy, and the illness of the third did not begin until 57 days after delivery. Only one of the four developed a schizophrenic illness *de novo* within a few days of childbirth and she eventually made a full recovery with no recurrence in the next 6 years.

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Appendix 1

Relationship between age and psychiatric admission rate

A test was made of the possibility that the unexpected long-term difference in admissions before and after childbirth might be due to a steadily increasing psychiatric admission rate in women of childbearing age unrelated to childbirth; that is, these women might have had higher admission rates in the last 21 months of the 4-year period of study than in the first 15 months simply because they were by then 2½ years older. The total numbers of admissions of married women between 1970 and 1981 were obtained from the EPCR for six consecutive age groups from 15–19 years to 40–44 years, and admission rates per 1000 population were calculated from these, using the mean of 1971 and 1981 census figures. There was indeed a steady increase in the admission rate over this age range, but the rate of increase calculated from a linear regression equation was only 1.6% per year. Even over 2½ years, therefore, the increase would be only 4%, which is trivial compared with the observed increase of 30%.

References

Bratfos, O. & Haug, J. O. (1966) Puerperal mental disorders in manic depressive females. *Acta Psychiatrica Scandinavica*, **42**, 285–294.

Brockington, I. F., Winokur, G. & Dean, C. (1982) Puerperal psychoses. In *Motherhood and Mental Illness* (eds I. F. Brockington & R. Kumar). London: Academic Press.

Dean, C. & Kendell, R. E. (1981) The symptomatology of puerperal illnesses. *British Journal of Psychiatry*, **139**, 128–133.

Esquirol, E. (1845) *Mental Maladies: a Treatise on Insanity*. (trans. E. K. Hunt). Philadelphia: Lea & Blanchard.

Heasman, M. A. & Clarke, J. A. (1979) Medical record linkage in Scotland. *Health Bulletin*, **37**, 97–103.

Kendell, R. E. (1985) Emotional and physical factors in the genesis of puerperal mental disorders. *Journal of Psychosomatic Research*, **29**, 3–11.

——, Wainwright, S., Hailey, A. & Shannon, B. (1976) The influence of childbirth on psychiatric morbidity. *Psychological Medicine*, **6**, 297–302.

——, McGuire, R. J., Connor, Y. & Cox, J. L. (1981*a*) Mood changes in the first three months after childbirth. *Journal of Affective Disorders*, **3**, 317–326.

——, Rennie, D., Clarke, J. A. & Dean, C. (1981*b*) The social and obstetric correlates of psychiatric admission in the puerperium. *Psychological Medicine*, **11**, 341–350.

Kumar, R. & Robson, K. M. (1984) A prospective study of emotional disorders in childbearing women. *British Journal of Psychiatry*, **144**, 35–47.

Paffenbarger, R. S. (1964) Epidemiological aspects of parapartum mental illness. *British Journal of Preventive and Social Medicine*, **18**, 189–195.

Paykel, E. S. (1978) Contribution of life events to causation of psychiatric illness. *Psychological Medicine*, **8**, 245–253.

——, Prusoff, B. A. & Myers, J. K. (1975) Suicide attempts and recent life events: a controlled comparison. *Archives of General Psychiatry*, **32**, 327–333.

Perris, C. & Brockington, I. F. (1981) Cycloid psychoses and their relation to the major psychoses. In *Biological Psychiatry 1981* (eds C. Perris, G. Struwe & B. Jansson). Amsterdam: Elsevier/North Holland Biomedical Press.

Pugh, T. F., Jerath, B. K., Schmidt, W. M. & Reed, R. B. (1963) Rates of mental disease related to childbearing. *New England Journal of Medicine*, **268**, 1224–1228.

Reich, T. & Winokur, G. (1970) Postpartum psychoses in patients with manic depressive disease. *Journal of Nervous and Mental Disease*, **151**, 60–68.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) *Research Diagnostic Criteria for a Selected Group of Functional Disorders*, 3rd edn. New York State Psychiatric Institute.

Surtees, P. G., Dean, C., Ingham, J. G., Kreitman, N. B., Miller, P.McC. & Sashidharan, S. P. (1983) Psychiatric disorder in women from an Edinburgh community: associations with demographic factors. *British Journal of Psychiatry*, **142**, 238–246.

——, Miller, P.McC., Ingham, J. G., Kreitman, N. B., Rennie, D. & Sashidharan, S. P. (1986) Life events and the onset of affective disorder. *Journal of Affective Disorders*, **10**, 37–50.

Tetlow, C. (1955) Psychoses of childbearing. *Journal of Mental Science*, **101**, 629–639.

Vislie, H. (1956) Puerperal mental disorders. *Acta Psychiatrica et Neurologica Scandinavica*, suppl. 111.

Whalley, L. J., Roberts, D. F., Wentzel, J. & Wright, A. F. (1982) Genetic factors in puerperal affective psychoses. *Acta Psychiatrica Scandinavica*, **65**, 180–193.

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Characteristic Plasma Hormone Changes in Alzheimer's Disease

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A systematic endocrine investigation in dementia, depression and control subjects showed that plasma growth hormone (GH) was higher in the morning and plasma TSH concentrations were higher throughout the day in Alzheimer-type dementia (ATD) than in age-matched depressed patients (MDD), and plasma TSH concentrations were also higher throughout the day in female ATD compared with age-matched female control subjects. The increased plasma TSH concentrations could not be due to reduced negative feedback because plasma T_3, T_4 and rT_3 were in the normal range. Plasma concentrations of oestrogen-stimulated neurophysin (ESN) were lower throughout the day in ATD compared with MDD and controls and lower in the morning compared with other dementias. The high plasma GH and TSH concentrations in ATD may reflect the reduced hypothalamic content of somatostatin in ATD, and the reduced concentrations of ESN may reflect reduced cholinergic activity in ATD brain. These selective hormonal changes provide a useful diagnostic test for Alzheimer's disease.

The neuropathological (Ishii, 1965) and some of the neurochemical features of Alzheimer-type dementia (ATD) affect not only the cerebral cortex but also subcortical structures, including the hypothalamus, in which there is a reduction of choline acetyltransferase (Davies, 1979), noradrenaline (Yates *et al*, 1981) and of somatostatin concentrations (Davies *et al*, 1982). Somatostatin is also reduced in CSF in patients with ATD (Wood *et al*, 1982). Acetylcholine, noradrenaline and somatostatin are involved in the control of the secretion of pituitary hormones (Reichlin, 1985), and the aim of the present study was to determine whether in ATD the central changes in these neurotransmitters are reflected in the basal secretion of pituitary hormones.

There have been few previous neuroendocrine studies in ATD. The majority of the studies have focused on the dexamethasone suppression test (DST), and a high incidence of early escape from dexamethasone suppression has been reported in patients with presenile (Raskind *et al*, 1982; Balldin *et al*, 1983) and senile ATD (Spar & Gerner, 1982; Coppen *et al*, 1983). Samorajski *et al* (1982) reported raised plasma prolactin concentrations in patients in hospital with senile dementia, but this finding was not confirmed in presenile ATD (Balldin *et al*, 1983); and in a study of a small number of patients, Hyyppa *et al* (1980) found the CSF prolactin concentrations were lower in patients with cerebral atrophy. Reduced sleep-related secretion of growth hormone (GH) was described in three ATD patients as compared with three age-matched control subjects (Davis *et al*, 1982).

The present study was designed to test hypotheses that plasma concentrations of pituitary hormones would (a) reflect changes in central neurotransmitters that occur in ATD and (b) prove useful in the differential diagnosis of ATD from other causes of dementia and depression, which is sometimes difficult, especially in elderly patients. The study compared hormone profiles in patients with presenile onset of ATD with those of major depressive disorders (MDD), other types of dementia and control subjects. ATD of presenile onset was studied because although the reduction in choline acetyltransferase is similar in older compared with younger patients with ATD (Yates *et al*, 1983*b*), younger patients with ATD have a more extensive loss of noradrenaline (Yates *et al*, 1983*b*; Rossor *et al*, 1984) and somatostatin (Pierotti *et al*, 1986).

Method

Alzheimer's type dementia

Patients who had developed symptoms suggestive of dementia before the age of 65 were admitted to hospital for investigation. The diagnosis of ATD was based on:

(a) onset of symptoms under the age of 65 with dysmnesia as the initial feature and with steadily progressing dementia
(b) absence of a history suggestive of another type of dementia or focal neurological signs or hypertension
(c) CT scan within normal limits or showing cerebral atrophy and showing no additional pathology
(d) EEG showing no focal abnormalities
(e) normal ECG, haematological, biochemical and CSF clinical investigations.

The 18 patients who met these criteria for ATD and were admitted to the study were all free of psychotropic drugs for at least 6 months before and throughout the duration of the study. They were assessed using the following psychological tests:

(a) orientation (Wechsler, 1955)
(b) short-term memory, forward and backward digit-span (Wechsler, 1955)
(c) long-term memory, Logical Memory (Wechsler, 1955) and Paired Associates Learning (Isaacs & Walky, 1964)
(d) agnosia – common objects; pictures; colours; auditory agnosia; tactile agnosia; naming of body parts (Eisenson, 1954)
(e) apraxia – hand position; finger flexion; use of objects; intrasensitive gestures (Kimura & Archibald, 1974).

Severely demented hospitalised patients were excluded but a wide range of severity of dementia was represented within the ATD population. Two patients were still in regular employment, three others were living alone while the remainder required the close support of relatives to remain in the community. The patients were reassessed 12 months later and only if the diagnosis remained unchanged were they included in the study.

Other dementias

Demented patients who after full investigation, failed to meet the criteria for ATD were included in the study and formed a dementia group who were unlikely to have ATD. Five of the nine patients had focal changes on the CT scan suggestive of vascular disease and were given a diagnosis of multi-infarct dementia. Two patients presented with personality and behavioural changes which were more pronounced than memory impairment. Both showed frontal lobe patterns of response in psychological tests and the provisional diagnosis was Pick's disease. One patient had the typical features of Huntington's chorea and a positive family history, while another patient had dementia associated with bilateral spasticity in the lower limbs, with a diagnosis thought to be disseminated sclerosis. All nine patients were free of psychotropic drugs for at least 6 months before the study.

Depressed patients

Patients with symptoms of a depressive illness, who had been free of antidepressant and neuroleptic drugs for at least 6 months, were selected from routine admissions to the Royal Edinburgh Hospital. The 16 patients admitted to the study remained free of antidepressant and neuroleptic drugs for the duration of the study. Eight patients were totally drug-free, seven patients received night sedation in the form of a benzodiazepine or chloral hydrate and one patient had received a recent course of ampicillin.

The depressed patients were all interviewed using the Present State Examination (PSE) Wing *et al*, 1974) within a few days of admission to hospital and were also assessed using the Hamilton Depression Rating Scale (Hamilton, 1960). The data from the PSE and clinical case records were assessed independently by two psychiatrists, and patients were classified according to the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1978). All 16 patients met the criteria for major depressive disorder (MDD), 14 endogenous and two probable endogenous subtype. Depressive delusions or hallucinations were present in ten patients, who therefore met criteria for MDD psychotic subtype.

Control subjects

The 37 control subjects – hospital staff and non-hospital-associated volunteers – were in good health and had not been taking any medication, including oral contraceptives, for at least 6 months.

Age and sex

Table I shows the mean age, age range and male/female composition of the patient groups and control subjects. Seven depressed patients and 11 control subjects were of similar age to the ATD patients. In the instances when plasma concentrations of a hormone showed a significant correlation with age within the total control population, only the age-related groups were used in statistical comparisons. Comparisons of plasma concentrations of luteinising hormone (LH) between ATD and control subjects were made only in post-menopausal women.

TABLE I
Patient and control populations

				Age (years)	
	n	*Male*	*Female*	*Mean ± s.d.*	*Range*
Alzheimer's disease (ATD)	18	5	13	61.0 ± 5.4	53–70
Major depressive disorder (MDD)	16	8	8	45.5 ± 12.6	25–72
MDD patients of similar age to ATD patients	7	3	4	56.6 ± 7.7	49–72
Other dementias	9	4	5	64.6 ± 8.7	44–72
Control subjects	37	14	23	39.9 ± 13.4	18–63
Control subjects of similar age to ATD patients	11	2	9	56.6 ± 5.6	48–63

Informed consent

All patients and control subjects consented to the study procedures, which they understood were undertaken only for research purposes. In cases where there was any doubt about a patient's understanding, the agreement of a close relative was sought before the patient was included.

Blood sampling

An in-dwelling cannula was inserted at approximately 06:40, kept patent by heparinised saline and left in place until 24:00. Blood samples were taken at 07:00, 07:30 and 08:00 (morning), 15:00, 15:30 and 16:00 (afternoon), and 23:00, 23:30 and 24:00 (evening). This blood sampling technique, designed to take account of the diurnal and pulsatile pattern of hormone secretion, allowed calculation of mean values for morning, afternoon and evening periods. In 15 ATD, 13 MDD and 9 other dementia patients a dexamethasone suppression test was carried out after the initial sampling day. Dexamethasone, 1 mg in a syrup, was given at 24:00 and blood samples were taken the following day at 08:00, 16:00 and 23:00. Blood samples were collected in lithium heparin-coated tubes containing 100 k IU Trasylol, and kept at 4°C before and during centrifugation. Plasma was stored at −40°C until assay.

Hormonal assays

Plasma hormone concentrations of GH, TSH, prolactin (PRL), LH and oestrogen-stimulated neurophysin (ESN) were determined using the NIADDK radioimmunoassay kits. The reference preparations were as follows: for GH, HS2243E; for TSH, WHO 1st IRP 68/38; for PRL, NIAMDD-HPRL-RP-1 (AFP-2312C); and for LH, LER-907. The reference preparation for ESN was that given to NIADDK by Dr A. Robinson, University of Pittsburg. Anti-rabbit gamma globulin (Scottish Antibody Production Unit (SAPU), Carluke, Scotland) at a final dilution of 1/100 was used as a second antibody. All samples were measured in duplicate. The sensitivity of the assays (90% B/Bo) were 1.6 mU/litre for GH (100 μl sample); 0.8 mU/litre for TSH (200 μl sample); 70 mU/litre for PRL (50 μl sample); 0.60 U/litre for LH (100 μl sample); and 85 pmol/litre for ESN (100 μl sample). The intra-assay coefficients of variation (COV) were 7.1% (low pool - 5.3 mU/litre and 7.4% (high pool - 40.0 mU/litre) for GH; 6.2% (low pool - 4.1 mU/litre) and 6.0% (high pool - 13.8 mU/litre) for TSH; 6.6% (low pool - 182 mU/litre) and 5.4% (high pool - 1.31 U/litre) for PRL; 7.4% (low pool - 1.34 U/litre) and 3.4% (high pool - 3.35 U/litre for LH; and 5.8% (low pool - 332 pmol/litre) and 4.9% (high pool - 672 pmol/litre) for ESN. The inter-assay COV were 8.4% (low pool) and 7.4% (high pool) for GH; 7.0% (low pool) and 9.1% (high pool) for TSH; 9.9% (low pool) and 8.4% (high pool) forPRL; 10.3% (low pool) and 8.9% (high pool) for LH; and 8.4% (low pool) and 7.8% (high pool) for ESN.

Total cortisol concentrations were determined by radioimmunoassay using a modification of the method of Seth & Brown (1978) with an antiserum (SAPU), raised in sheep against cortisol-3-O-carboxymethyloxime-bovine serum albumin. Anti-sheep/goat serum (SAPU) at a final tube dilution of 1/83 was used as the second antibody. The sensitivity of the assay (90% B/Bo) was 11.6 nmol/litre cortisol. The intra-assay COV were 2.2% (low pool - 153 nmol/litre), 2.8% (middle pool - 310 nmol/litre) and 3.2% (high pool - 560 nmol/litre). The interassay COV were 4.6% (low pool). 2.68% (middle pool) and 6.34% (high pool).

Statistical analysis

The distributions of the mean morning (07:00, 07:30 and 08:00), afternoon (15:00, 15:30 and 16:00) and evening (23:00, 23:30 and 24:00) plasma concentrations of cortisol, GH, TSH, PRL, LH and ESN in patients and control subjects were significantly skewed. The cortisol data were log-transformed to approximate normality, and the significance of the differences in the mean plasma cortisol concentrations between ATD, MDD, other dementias and control subjects was determined by one-way analysis of variance and the multiple range test of Duncan (1961). The distributions of the other hormones, however, remained skewed despite transformation, and the Mann–Whitney *U* test was used to determine the significance of differences between the values. In all 96 comparisons were made between the groups; thus by chance alone up to five significant differences would be expected (four at $P<0.05$ and one at $P<0.02$). In the ATD group, correlations between the mean morning, afternoon and evening plasma GH, TSH, cortisol, PRL and ESN concentrations were made using Spearman's Rank Correlation coefficient (30 comparisons). Also, within the ATD group, mean morning, afternoon and evening concentrations of GH, TSH, cortisol and ESN were correlated with psychological test scores (60 comparisons).

Results

Relationship between age, sex and plasma hormone concentrations in control subjects

Plasma cortisol and plasma ESN concentrations were not significantly correlated with age. Afternoon plasma GH concentrations decreased with age (morning, $r=-0.24$, NS: afternoon, $r=-0.30$, $P<0.05$; evening, $r=-0.23$, NS). Morning and afternoon plasma TSH concentrations (morning, $r=0.36$, $P<0.02$; afternoon, $r=0.31$, $P<0.02$; evening, $r=0.24$, NS) increased with age, and afternoon plasma prolactin concentrations (morning, $r=-0.02$; afternoon, $r=0.35$, $P<0.02$; evening, $r=0.16$) also increased with age. We concluded that plasma GH, TSH and prolactin concentrations correlated with age and that comparisons between ATD and other groups would require age-matching.

Plasma concentrations of TSH, cortisol and ESN were similar in male and female control subjects at all three time periods. Plasma GH concentrations were higher in female as compared with male control subjects in the evening only ($P<0.01$) and plasma prolactin concentrations were higher in female compared with male control subjects in the afternoon ($P<0.02$). When the female control population was divided into pre- and post-menopausal groups, the post-menopausal women, as expected, had higher plasma LH concentrations at all three times of day ($P<0.02$) than the pre-menopausal women.

Growth hormone

Table II shows that plasma GH concentrations in the morning were significantly higher in ATD as compared with age-related control subjects ($P<0.02$) but not different from age-related MDD. Female ATD patients had higher morning ($P<0.02$) and afternoon ($P<0.05$) plasma GH concentrations than age-related female control subjects. The other dementia group had GH concentrations similar to those in control subjects.

TABLE II

Plasma GH and TSH concentrations in Alzheimer presenile dementia, other dementias and age-matched control subjects

		Growth hormone (mU/l) (mean ± s.e.)				*TSH (mU/l) (mean ± s.e.)*		
	n	*Morning*	*Afternoon*	*Evening*	*n*	*Morning*	*Afternoon*	*Evening*
Alzheimer	17	7.7 ± 1.0*	7.4 ± 1.3	5.2 ± 0.7	18	12.2 ± 6.6†††	7.5 ± 3.0††	12.2 ± 6.1†
Depression	7	9.3 ± 2.8	7.5 ± 1.5	4.8 ± 0.7	7	3.3 ± 0.2	3.1 ± 0.2	3.7 ± 0.5
Other dementias	9	5.1 ± 0.9	4.5 ± 0.8	6.0 ± 1.1	9	4.7 ± 0.6	4.0 ± 0.4	5.1 ± 0.5
Controls	11	4.1 ± 0.5	3.9 ± 0.3	6.9 ± 2.3	11	4.4 ± 0.2	3.9 ± 0.2	4.9 ± 0.5
Alzheimer (female)	12	8.8 ± 1.1**	8.6 ± 1.6*	5.6 ± 0.8	13	15.4 ± 9.0*	8.9 ± 4.1*	15.3 ± 8.4**
Controls (female)	9	4.1 ± 0.5	4.0 ± 0.4	7.7 ± 2.8	9	4.2 ± 0.1	3.8 ± 0.2	4.5 ± 0.2

ATD *v.* controls: $^{*}P<0.05$; $^{**}P<0.02$.
ATD *v.* MDD: $^{\dagger}P<0.05$; $^{\dagger\dagger}P<0.02$; $^{\dagger\dagger\dagger}P<0.002$.

Thyroid-stimulating hormone

Table II shows that the plasma TSH concentrations were higher at all three time periods in ATD compared with age-related MDD (morning, $P<0.002$; afternoon, $P<0.02$; evening, $P<0.05$). Female ATD patients had significantly higher plasma TSH concentrations throughout the day compared with age-related female control subjects (morning, $P<0.05$; afternoon, $P<0.05$; evening, $P<0.02$), and morning TSH concentrations were greater than 6 mU/litre in seven of 13 (54%) ATD women but only in one female other dementia patient and no female MDD patients or control subjects. The other dementia patients had plasma TSH concentrations similar to those in control subjects. Plasma triiodothyronine (T_3) and thyroxine (T_4) and reverse triiodothyronine (rT_3) concentrations were similar in ATD and age-related control subjects (ATD, mean ± s.e., $T_4 = 96.4 \pm 5.7$ nmol/litre, $T_3 = 1.58 \pm 0.09$ nmol/litre, $rT_3 = 0.30 \pm 0.05$ nmol/litre; control subjects, mean ± s.e., $T_4 = 99.5 \pm 4.5$ nmol/litre, $T_3 = 1.73 \pm 0.07$ nmol/litre, $rT_3 = 0.22 \pm 0.01$ nmol/litre). All subjects had T_3 and T_4 concentrations within the normal laboratory range. One ATD patient had very high plasma TSH concentrations (mean for the day 98.5 mU/litre) but normal T_3, T_4 and rT_3 concentrations and no clinical features of hypothyroidism. This patient's thyroid status was reassessed over a 4-year period, during which she developed no features of hypothyroidism, her T_3 and T_4 remained within the normal range and her plasma TSH concentrations gradually declined to a value of 10.9 mU/litre.

Cortisol

At all three time periods, MDD had higher plasma cortisol concentrations than control subjects ($P<0.05$) but not significantly greater than ATD (Table III). In the evening, ATD patients and also the other dementia patients had significantly higher plasma cortisol concentrations than control subjects ($P<0.05$).

After the administration of dexamethasone, three of 15 ATD patients (20%) had a 16:00 cortisol concentration greater than 138 nmol/litre and the 23:00 post-dexamethasone cortisol was also raised in these three patients. In patients with MDD, six of 13 (46%) had 16:00 cortisol concentrations greater than 138 nmol/litre. In the other dementia group three of 9 patients (two Picks and one Huntington's) had 16:00 cortisol concentrations greater than 138 nmol/litre. The ATD patients had a lower ($P<0.05$) 16:00 post-dexamethasone cortisol concentration (115 ± 57 nmol/litre) than the depressed patients (167 ± 36 nmol/litre).

Oestrogen-stimulated neurophysin

Plasma ESN concentrations (Table III) were significantly lower at all three time periods in ATD compared with MDD patients (morning, $P<0.01$; afternoon, $P<0.01$; evening, $P<0.05$) and with control subjects (morning, $P<0.001$; afternoon, $P<0.01$; evening, $P<0.001$). They were also lower in the morning ($P<0.05$) in ATD as compared with other dementia patients. The nine values for each patient or control subject were averaged and the plasma ESN concentration for the day was significantly lower in ATD compared with MDD ($P<0.001$), other dementias ($P<0.05$) and control subjects ($P<0.001$) (Fig. 1). All ATD patients had a mean plasma ESN concentration < 170 pmol/litre. The averages of the nine plasma ESN concentrations for the day for the ATD patients were also compared with the average ESN concentrations of 37 patients with psychosis, who were within the age range 50–72 years but not all of whom met the drug-free criteria. The elderly psychotic group had higher plasma ESN concentrations – 161 ± 18 pmol/litre – compared with the ATD patients – 105 ± 6 pmol/litre ($P<0.001$).

Prolactin and luteinising hormone concentrations

Plasma prolactin concentrations (mean ± s.e.) were similar in ATD (morning 493 ± 123 mU/litre, afternoon 355 ± 27 mU/litre, evening 360 ± 26 mU/litre), age-related MDD (morning 687 ± 304 mU/litre, afternoon 678 ± 313 mU/litre, evening 527 ± 158 mU/litre), other dementias (morning 403 ± 57 mU/litre, afternoon 318 ± 30 mU/litre, evening 425 ± 36 mU/litre) and control subjects (morning 344 ± 36 mU/litre, afternoon 291 ± 20 mU/litre, evening 335 ± 28 mU/litre).

Plasma LH concentrations (mean ± s.e.) did not differ significantly in post-menopausal women with ATD ($n = 11$; morning 16.4 ± 1.6 U/litre, afternoon 14.3 ± 1.1 U/litre, evening 15.0 ± 1.4 U/litre) compared with female patients with other dementia ($n = 5$; morning 11.9 ± 2.3 U/litre, afternoon 12.0 ± 1.6 U/litre, evening 12.7 ± 1.6 U/litre) and post-menopausal female control subjects ($n = 9$; morning

TABLE III
Plasma cortisol and oestrogen-stimulated neurophysin concentrations

		Cortisol (nmol/l)[1] *(mean ± s.e.)*				*Oestrogen-stimulated neurophysin (pmol/l)*[2] *(mean ± s.e.)*		
	n	*Morning*	*Afternoon*	*Evening*	*n*	*Morning*	*Afternoon*	*Evening*
Alzheimer	17	469±23	244±22	210±25*	16	105± 6***	102± 6**	107± 9***
Depression	16	511±31*	301±39*	214±24*	14	230±41††	239±45††	202±37†
Other dementias	9	530±41*	274±41	249±34*	9	192±44§	173±36	163±34
Controls	37	436±25	230±13	154±16	36	191±24	185±25	239±38

1. Conversion, SI to traditional units, cortisol: 1 nmol/litre ≃ 0.36 μg/litre.
2. Conversion, SI to traditional units, ESN: 1 pmol/litre ≃ 0.012 μg/litre.

**v.* control subjects $P<0.05$: ATD *v.* controls: **$P<0.01$, ***$P<0.001$; ATD *v.* MDD, †$P<0.05$, ††$P<0.01$; ATD *v.* other dementias, §$P<0.05$.

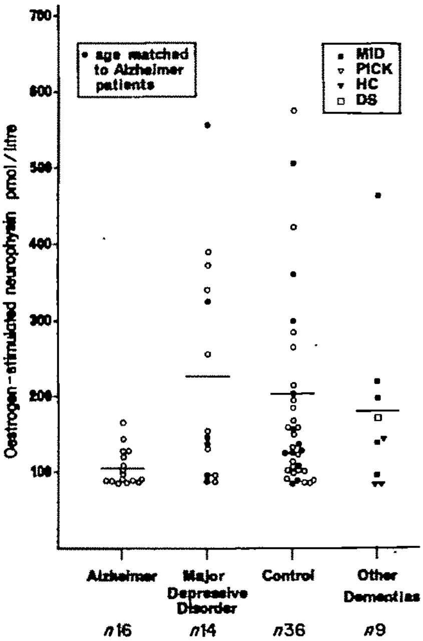

FIG. 1 Mean plasma oestrogen-stimulated neurophysin concentrations based on nine estimations in 'neuroendocrine day' studies carried out in patients with Alzheimer-type dementia (ATD) major depressive disorder (MDD) and other dementias (MID = multi-infarct dementia, Pick = Pick's disease, HC = Huntington's chorea, DS = disseminated sclerosis) and in normal control subjects: ATD *v.* controls – $P<0.001$; ATD *v.* MDD – $P<0.001$; ATD *v.* other dementias – $P<0.05$ (conversion, SI to traditional units, 1 pmol/l ≃ 0.012 μg/l).

14.6 ± 2.2 U/litre, afternoon 14.3 ± 1.9 U/litre, evening 13.9 ± 1.4 U/litre).

Relationship between plasma hormone concentrations and performance in psychological tests

Table IV shows the correlations in ATD patients between the mean plasma hormone concentrations and psychological test scores. Plasma cortisol concentrations, particularly in the evening, were inversely correlated with psychological test scores; that is, the more demented patients had higher plasma cortisol concentrations. Orientation was the only subtest which showed a positive significant correlation with afternoon and evening plasma TSH concentrations. Evening plasma GH concentrations were inversely correlated with psychological test scores. There were no significant correlations between plasma ESN concentrations and psychological test scores.

Intercorrelations between plasma concentrations of TSH, GH, cortisol, prolactin and ESN in ATD and in control subjects

Plasma cortisol concentrations in the evening were inversely related to TSH concentrations in ATD ($r = -0.50$, $P<0.05$) but not in control subjects. Plasma TSH concentrations in the morning positively correlated with PRL concentrations in ATD ($r = 0.50$, $P<0.05$). Afternoon plasma concentrations of GH correlated inversely with ESN concentrations in ATD ($r = -0.52$, $P<0.05$). In the control subjects the only significant correlation was a positive correlation between evening plasma concentrations of GH and PRL ($r = 0.83$, $P<0.001$).

Diagnostic implications

Morning plasma TSH concentrations were useful in discriminating between ATD and age-related MDD patients, with 13 ATD patients (72%) but none of the MDD patients having TSH concentration greater than 4.5 mU/litre. When morning plasma TSH and ESN concentrations (TSH > 4.5 mU/litre; ESN < 170 pmol/litre) were used to discriminate between ATD, MDD, other dementias and control subjects, 75% of the ATD patients were correctly identified, together with one patient with multi-infarct dementia and one control subject, but no MDD patients; a specificity of 86% (Fig. 2).

Discussion

The present study has identified selective changes in plasma concentrations of TSH, GH and ESN that are specific to ATD and which can be related to the

TABLE IV

Correlations between mean morning, afternoon and evening plasma hormone concentrations and the score achieved in psychological tests in Alzheimer patients

	Morning	*Afternoon*	*Evening*
Growth hormone ($n = 17$)			
Total score	0.01	-0.06	-0.58*
Orientation	-0.06	-0.13	-0.40
Paired Associates Learning + Logical Memory	-0.12	-0.08	-0.56*
Apraxia	-0.03	0.03	-0.33
Agnosia	0.10	-0.03	-0.46*
TSH ($n = 18$)			
Total score	0.14	0.29	0.30
Orientation	0.37	0.43*	0.40*
Paired Associates Learning + Logical Memory	0.24	0.35	0.38
Apraxia	-0.19	0.17	0.14
Agnosia	0.08	0.17	0.24
Cortisol ($n = 18$)			
Total score	-0.31	-0.18	-0.42*
Orientation	-0.41*	-0.16	-0.59**
Paired Associates Learning + Logical Memory	-0.38	-0.10	-0.38
Apraxia	-0.41*	-0.15	-0.27
Agnosia	-0.25	-0.05	-0.54*
Oestrogen-stimulated neurophysin ($n = 16$)			
Total score	0.11	0.20	0.19
Orientation	0.26	0.12	0.23
Paired Associates Learning + Logical Memory	0.17	0.17	0.29
Apraxia	-0.06	0.23	0.16
Agnosia	-0.21	-0.14	-0.16

*$P<0.05$; **$P<0.02$.

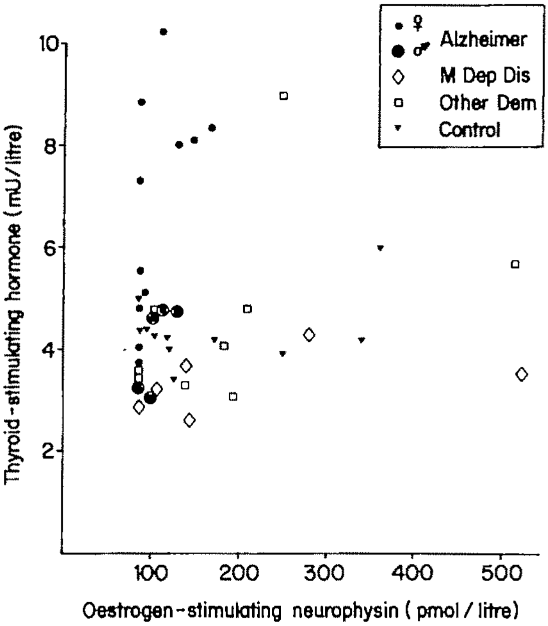

FIG. 2 Morning plasma thyroid-stimulating hormone concentrations plotted with morning oestrogen-stimulated neurophysin concentrations for patients with Alzheimer-type dementia, major depressive disorder, other dementias and control subjects.

known neurochemical deficits in ATD. Although raised plasma GH concentrations could be due to increased release of GH-releasing hormone (GHRH), it is more likely in ATD to be due to decreased release of somatostatin, which is known to be deficient in ATD. Since somatostatin also inhibits TSH, a similar mechanism may account for the increased plasma TSH concentrations in ATD. Hypothalamic TSH releasing hormone (TRH) concentrations are normal in post-mortem tissue in ATD (Yates *et al*, 1983*a*) and it is, therefore, unlikely that raised plasma TSH concentrations are due to increased release of TRH.

The increase in plasma GH and TSH concentrations is more marked in the female ATD patients. Raised morning plasma TSH concentrations are more common in women, particularly those over the age of 45 years. In the Whickham Community Survey (Tunbridge *et al*, 1977), the percentage of morning TSH concentrations greater than 6 mU/litre was 7.5% for females and 2.8% for males for the whole population, irrespective of age. The percentage of raised TSH concentrations in the age range 55–64 years was 10% for females and 1.9% for males, and in the age range 65–74 years was 8% for females and 6.9% for males. In the present study, seven out of the 13 female ATD patients (54%) had morning TSH concentrations greater than 6 mU/litre, far in excess of that expected in the general population. The increase in plasma TSH concentrations in female ATD patients appears to be central, because peripheral indices of thyroid function (T_3, T_4 and rT_3) were within the normal range and similar to those of control subjects. Plasma GH concentrations did not correlate with TSH in either ATD or control subjects, but the control of the secretion of these hormones is complex and somatostatin is only one of many factors involved (Mendelson, 1982; Scanlon *et al*, 1980). Evening plasma TSH concentrations in ATD were inversely correlated with plasma cortisol concentrations, and it is possible that raised cortisol concentrations in the more severely demented patients inhibit TSH secretion (Re *et al*, 1976).

The highly significant reduction of plasma ESN concentrations in ATD may be related to the marked deficiency in ATD brain of acetylcholine which is known to stimulate oxytoxin release (Poulain & Wakerley, 1982) and, therefore, presumably the oxytocin-associated ESN. The decreased ESN concentrations may, however, also be due to decreased synthesis of ESN itself, since in rats the staining intensity for the neurohypophysial hormones decreases progressively with age, especially in the paraventricular nucleus and in oxytocin-containing neurones (Watkins & Choy, 1980), but recent studies of human brain have failed to show any change in the area of oxytocin cells with increasing age (Fliers *et al*, 1985). Scrapie in sheep, which has some neuropathological features in common with those of ATD, causes degeneration of magnocellular neurones, with loss of neurophysin in the posterior pituitary gland (Parry & Livett, 1976).

The majority of studies report that concentrations of dopamine and homovanillic acid are normal in ATD (Yates *et al*, 1979; Mann *et al*, 1980). Consistent with this finding, plasma prolactin concentrations were similar in ATD patients and age-related controls in the present study, as well as in the study of Balldin *et al* (1983). The raised evening plasma cortisol concentrations in ATD compared with control subjects are not useful diagnostically, because the morning and evening plasma cortisol concentrations are increased in other dementias and increased throughout the day in MDD, as has also been shown to be the case in many previous studies of depression (Gibbons, 1964; Carpenter & Bunney, 1971; Sachar *et al*, 1973). Raised plasma cortisol concentrations are found in many psychotic patients, irrespective of the presence of depressed mood (Christie *et al*, 1986). The raised evening plasma cortisol concentrations in the ATD patients appears to be a feature of the more severely demented patients, with an inverse relationship between cortisol concentrations and psychological test scores. Only three of 15 ATD patients (20%) showed early escape from dexamethasone suppression, and the non-suppressors included the two most severely demented patients in the study. Previous studies in demented patients have suggested a 40–50% incidence of early escape from dexamethasone suppression (Raskind *et al*, 1982; Spar & Gerner, 1982; Balldin *et al*, 1983; Coppen *et al*, 1983), similar incidence to that reported in endogenous depression (Carroll *et al*, 1981), but the majority of patients, unlike those in the present study, were hospitalised, severely demented patients.

Plasma TSH and ESN concentrations are useful in discriminating between ATD, other causes of dementia and depression. Morning TSH concentrations greater than 4.5 mU/litre, identified 72% of ATD and no depressed patients, and the additional use of morning ESN concentrations improved the discrimination between ATD, other dementias, MDD and control subjects to a sensitivity of 75% and specificity of 86%.

These results show significant increases in the concentrations of GH, TSH and cortisol and a decrease in concentration of ESN in the plasma of patients with ATD. Increased plasma GH and TSH concentrations in ATD are consistent with reports of reduced somatostatin in post-mortem neurochemical studies, while decreased ESN may be caused by decreased cholinergic activity. The finding of predictable changes in pituitary output based on established neurochemical abnormalities supports the rationale for neuroendocrine studies in psychiatry. These characteristic hormonal changes in ATD provide the basis for psychopharmacological analysis of the central neurochemical abnormalities of ATD and offer important biological markers for discriminating between ATD, other dementias and major depression.

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References

Balldin, J., Gottfries, C.-G., Karlsson, I., Lindstedt, G., Langstrom, G. & Walinder, J. (1983) Dexamethasone suppression test and serum prolactin in dementia disorders. *British Journal of Psychiatry*, **143**, 277–281.

Carpenter, W. T. Jr & Bunney, W. E. (1971) Adrenal cortical activity in depressive illness. *American Journal of Psychiatry*, **128**, 31–40.

Carroll, B. J., Feinberg, M., Greden, J. F. Tarika, J., Albala, A. A., Hasket, R. F., James, N.McI., Krnofol, Z., Lohr, N., Steiner, M., de Vigne, J. P. & Young, E. (1981) A specific laboratory test for the diagnosis of melancholia. *Archives of General Psychiatry*, **38**, 15–22.

Christie, J. E., Whalley, L. J., Dick, H., Blackwood, D. H. R., Blackburn, I. M. & Fink, G. (1986) Raised plasma cortisol concentrations are a feature of drug-free psychotics and not specific for depression. *British Journal of Psychiatry*, **148**, 58–65.

Coppen, A., Abou-Saleh, M., Milln, P., Metcalfe, M., Harwood, J. & Bailey, J. (1983) Dexamethasone suppression test in depression and other psychiatric illness. *British Journal of Psychiatry*, **142**, 498–504.

Davies, P. (1979) Neurotransmitter related enzymes in senile dementia of the Alzheimer type. *Brain Research*, **171**, 319–327.

——, Katz, D. A. & Crystal, H. A. (1982) Choline acetyltransferase, somatostatin, and substance P in selected cases of Alzheimer's disease. In *Alzheimer's Disease: Report of Progress in Research* (eds S. Corkin, K. L. Davis, J. H. Growdon, E. Usdin, J. Wurtman). New York: Raven Press.

Davis, B. M., Levy, M. I., Rosenberg, G. S., Mathe, A. & Davis, K. L. (1982) Relationship between growth hormone and cortisol and acetylcholine: a possible neuroendocrine strategy for assessing a cholinergic deficit. In *Alzheimer's Disease: Report of Progress in Research* (eds S. Corkin, K. L. Davis, J. H. Growdon, E. Usdin, J. Wurtman). New York: Raven Press.

Duncan, D. B. (1961) Bayes rules for a common multiple comparisons problem and related student-*t* problems. *Annals of Mathematical Statistics*, **32**, 1013.

Eisenson, J. (1954) *Examination for Aphasia*. New York: The Psychological Corporation.

Fliers, E., Swaab, D. F., Pool, Chr., W. & Verwer, R. W. H. (1985) The vasopressin and orcytocin neurones in the human supraoptic and paraventricular nucleus; changes with aging and in senile dementia. *Brain Research*, **342**, 45–53.

Gibbons, J. (1964) Cortisol secretion rate in depressive illness. *Archives of General Psychiatry*, **10**, 572–575.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

Hyyppa, M. T., Tenkku, M., Liira, J. & Jaykka, H. (1980) Low cerebrospinal fluid prolactin levels in patients with central cerebral atrophy. *Journal of the Neurological Sciences*, **47**, 145–146.

Isaacs, B. & Walky, F. A. (1964) A simplified paired associate test for elderly hospital patients. *British Journal of Psychiatry*, **110**, 80–83.

Ishii, T. (1965) Distribution of Alzheimer's neurofibrillary changes in the brain stem and hypothalamus of senile dementia. *Acta Neuropathologica*, **6**, 181–187.

Kimura, D. & Archibald, Y. (1974) Motor functions of the left hemisphere. *Brain*, **97**, 337–350.

Mann, D. M. A., Lincoln, J., Yates, P. O., Stamp, J. E. & Toper, S. (1980) Changes in the monoamine containing neurones of the human CNS in senile dementia. *British Journal of Psychiatry*, **136**, 533–541.

Mendelson, W. B. (1982) Studies of human growth hormone secretion in sleep and waking. *International Review of Neurobiology*, **23**, 367–389.

Parry, H. B. & Livett, B. G. (1976) Neurophysin in the brain and pituitary gland of normal and scrapie-affected sheep. *Neuroscience*, **1**, 275–299.

Pierotti, A. R., Harmar, A. J., Simpson, J. & Yates, C. M. (1986) High molecular weight forms of somatostatin-like immunoreactivity are reduced in Alzheimer's disease and Down's syndrome. *Neuroscience Letters*, **63**, 141–146.

Poulain, D. A. & Wakerley, J. B. (1982) Electrophysiology of hypothalamic magnocellular neurones secreting oxytocin and vasopressin. *Neuroscience*, **7**, 773–808.

Raskind, M., Peskind, E., Rivard, M.-F., Veith, R. & Barnes, R. (1982) Dexamethasone suppression test and cortisol circadian rhythm in primary degenerative dementia. *American Journal of Psychiatry*, **139**, 1468–1471.

Re, R. N., Kourides, I. A., Ridgway, E. C., Weintraub, B. D. & Maloof, F. (1976) The effect of glucocorticoid administration on human pituitary secretion of thyrotropin and prolactin. *Journal of Clinical Endocrinology and Metabolism*, **43**, 338–346.

Reichlin, S. (1985) Neuroendocrinology. In *Williams Textbook of Endocrinology*, (eds J. D. Wilson & D. W. Foster). 7th edn. London: Saunders.

Rossor, M. N., Iversen, L. L., Reynolds, G. P., Mountjoy, C. Q. & Roth, M. (1984) Neurochemical characteristics of early and late onset types of Alzheimer's disease. *British Medical Journal*, **288**, 961–964.

Sachar, E. J., Hellman, L., Roffwarg, H. P., Halpern, F., Fukushima, S. & Gallagher, T. (1973) Disrupted 24-hour patterns of cortisol secretion in psychotic depression. *Archives of General Psychiatry*, **28**, 19–24.

Samorajski, J., Ho, B. T., Kralik, P. M. & Hartford, J. T. (1982) Serum prolactin changes with age, senile dementia, and dihydroergotoxine mesylate treatment. In *The Aging Brain: Cellular and Molecular Mechanisms of Aging in the Nervous System* (eds E. Giacobini, G. Giacobini, G. Filogamo, A. Vernadakis). New York: Raven Press.

Scanlon, M. F., Lewis, M., Weightman, D. R., Chan, V. & Hall, R. (1980) The neuroregulation of human thyrotropin secretion. In *Frontiers in Neuroendocrinology* (eds L. Martini, W. F. Ganong). New York: Raven Press.

Seth, J. & Brown, L. M. (1978) A simple radioimmunoassay for plasma cortisol. *Clinica Chimica Acta*, **86**, 109–120.

Spar, J. E. & Gerner, R. (1982) Does the dexamethasone suppression test distinguish dementia from depression? *American Journal of Psychiatry*, **139**, 238–240.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, **36**, 773–782.

Turnbridge, W. M. G., Evered, D. C., Hall, R., Appleton, M. B., Clark, F., Evans, J. G., Young, E., Bird, T. & Smith, P. A. (1977) The spectrum of thyroid disease in the community of the Whickham Survey. *Clinical Endocrinology*, **7**, 481–493.

Watkins, W. B. & Choy, V. J. (1980) The impact of aging on neuronal morphology in the rat hypothalamo-neurohypophysial system: an immunohistochemical study. *Peptides*, **1**, 239–245.

Wechsler, D. (1955) *Manual for Wechsler Memory Scale*. New York: Psychological Corporation.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

Wood, P. L., Etienne, P., Lal, S., Gauthier, S., Cajal, S. & Nair, N. P. V. (1982) Reduced lumbar CSF somatostatin levels in Alzheimer's disease. *Life Sciences*, **31**, 2073–2079.

Yates, C. M., Allison, Y., Simpson, J., Maloney, A. F. J. & Gordon, A. (1979) Dopamine in Alzheimer's disease and senile dementia. *Lancet*, **ii**, 851–852.

——, Ritchie, I. M. & Simpson, J. (1981) Noradrenaline in Alzheimer-type dementia and Down syndrome. *Lancet*, **ii**, 39–40.

——, Harmar, A. J., Rosie, R., Sheward, J., Sanchez De Levy, G., Simpson, J., Maloney, A. F. J., Gordon, A. & Fink, G. (1983*a*) Thyrotropin-releasing hormone and substance P immunoreactivity in post mortem brain from cases of Alzheimer-type dementia and Down's syndrome. *Brain Research*, **258**, 45–52.

——, Simpson, J., Gordon, A., Maloney, A. F. J., Allison, Y., Ritchie, I. M. & Urquhart, A. (1983*b*) Catecholamines and cholinergic enzymes in pre-senile and senile Alzheimer-type dementia and Down's syndrome. *Brain Research*, **280**, 119–126.

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Increased Plasma LH in Manic-Depressive Illness:

Evidence of a State-Independent Abnormality

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Basal plasma luteinising hormone concentrations and the LH responses to LH-releasing hormone injection were higher in 11 young men after recovery from mania than in 15 control subjects. Since plasma concentrations of testosterone and sex hormone binding globulin were similar in the recovered manic compared with the control subjects, the increased LH response to LHRH is likely to have been due to the priming effect of LHRH consequent on an increased release of LHRH into hypophysial portal vessel blood. Abnormal control of LHRH, and thereby LH release, is a state-independent feature in male patients with mania and may be a useful trait marker for this disorder.

Manic-depressive illness is a recurrent psychosis of unknown aetiology. Episodes of mania and depression are followed by periods of complete recovery and, in the natural history of this disorder, changes from normality to illness can take place with characteristic suddenness. Previously, we have reported that in acute mania in drug-free young men, plasma luteinising hormone (LH) concentrations were higher than in schizophrenia or in healthy control subjects (Whalley *et al*, 1985). The increase in plasma LH concentrations could not have been due to decreased negative feedback of testosterone because plasma testosterone and sex hormone binding globulin (SHBG) did not differ between groups.

The increased plasma LH concentrations in mania could be due to a central abnormality leading to increased release of LH-releasing hormone (LHRH), to increased pituitary responsiveness to LHRH, or to both, since LHRH increases the responsiveness of the pituitary gland to itself, the so-called priming effect of LHRH (Aiyer *et al*, 1974; Fink, 1979). In the present paper we have investigated whether plasma LH concentrations remain elevated after recovery from mania, which would indicate whether the abnormality is either state- or trait-dependent, and also whether pituitary responsiveness to LHRH is altered in men during the euthymic phase of manic-depressive illness.

Method

Eleven recovered manic-depressive and 15 control male subjects were studied. The recovered manic-depressive patients had sufficient case-note data to meet Research Diagnostic Criteria (RDC, Spitzer *et al*, 1975) for at least one episode of mania. All had been free of any psychotropic medication, other than lithium, for at least 2 weeks. The mean ages (±s.e.; range) for the patients were 36 years (±3.1; 25–53) and for the control group 33 years (±2.7; 19–59). All studies were started between 08:00 and 09:00 after an overnight fast. Blood samples were obtained from an in-dwelling cannula (with a heparin lock) which was placed in a forearm vein about 20 minutes before the first sample was taken. Samples were taken at the following times in relation to injection of 100 μg LHRH dissolved in 1 ml aqueous solution: (−60, −30, −5, +15, +30, +45, +60, +90, +120 and +180 minutes). Blood was collected into lithium-heparin coated tubes containing 100 Kallikrein inactivator units of aprotinin, and kept at 4°C until centrifugation at 1600*g* and 4°C. The plasma was stored at −40°C until hormone assay, as described previously (Whalley *et al*, 1985). The inter-assay (and intra-assay) coefficients of variation were as follows: LH 5.6% (3.8%); FSH 5.7% (3.6%); prolactin (2.6%); testosterone 6.8% (7.0%); SHBG 6.6% (5.8%). The maximal incremental change in plasma hormone concentration following LHRH injection was calculated as the difference between the maximum concentration during the first 90 minutes after LHRH and the concentration 5 minutes before LHRH injection. Data were log-transformed to approximate normality before comparisons between groups were made by analysis of variance for repeated measures (with the conservative Greenhouse–Geisser correction of probabilities (Winer, 1971). Student's *t*-test was used to locate the timing of significant differences.

Results

The results are shown in Fig. 1.

Basal plasma LH concentrations were significantly greater ($P<0.025$) in the recovered patients than in the control subjects. After LHRH injection, plasma LH concentrations were greater ($P<0.05$–$P<0.01$) in the recovered patients than in the control subjects at +15, +30, +45, +60, +90 and +180 min, and ANOVA with repeated measures showed a significant difference ($P<0.01$) between groups. The mean maximum increments in plasma LH concentrations were significantly greater ($P<0.01$) in the patients (mean ± s.e., 9.5 ± 1.8 *IU*/litre) than in the control subjects (5.6 ± 0.5 *IU*/litre).

Plasma FSH concentrations increased significantly (ANOVA with repeated measures, $P<0.002$) in both the patient and control groups after LHRH injection, but there were no significant differences between the two groups in either the basal plasma FSH concentrations or the maximal increments in plasma FSH concentrations.

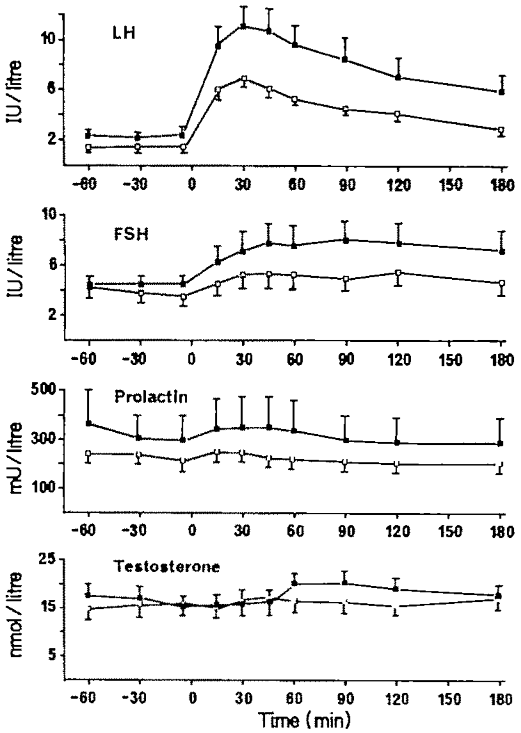

FIG. 1 Plasma concentrations of luteinising hormone (LH), follicle stimulating hormone (FSH), prolactin and testosterone in 11 recovered manic-depressive men (■——■) and 15 male control subjects (□——□) before and after 100 μg LHRH injection at 0 min.

Plasma prolactin concentrations increased slightly but significantly (ANOVA with repeated measures, $P<0.001$) after LHRH injection. There were no significant differences between the two groups.

Plasma testosterone concentrations did not change after LHRH injection and there were no significant differences between the two groups. Plasma SHBG concentrations in blood samples obtained immediately before LHRH injection (mean ± s.d.; controls, 40.1 ± 12.7 nmol/litre; patients, 40.7 ± 23.1 nmol/litre) and at the time of the maximum increment in plasma LH concentration, for each subject, did not differ between patients and control subjects.

Discussion

The fact that the plasma LH concentrations before LHRH injection were significantly greater in recovered male manic-depressive patients compared with healthy control subjects, together with our previous finding of increased plasma LH concentrations during acute mania (Whalley *et al*, 1985), suggests that increased plasma LH concentrations are a trait-dependent feature of manic-depressive illness which is independent of the state. As was the case in our previous study (Whalley *et al*, 1985), the increased plasma concentrations of LH in recovered manics could not be attributed to a failure of negative feedback, since the plasma concentrations of testosterone and SHBG were not significantly different from the values in normal subjects. The increased pituitary responsiveness to LHRH in recovered manics compared with the normal subjects could have been due to differences between the groups in plasma concentrations of sex steroids. However, since the plasma testosterone and SHBG concentrations were similar in the recovered manic and control subjects, a more likely explanation is that the responsiveness of the pituitary gland in patients with mania had been increased as a consequence of increased LHRH release and the priming effect of LHRH (Aiyer *et al*, 1974; Fink, 1979). The priming effect of LHRH has long been invoked to explain the positive correlation between basal plasma LH concentrations and the magnitude of the LH response to LHRH in man (Aiyer *et al*, 1974; Roth *et al*, 1972; Gual, 1973; Isurugi *et al*, 1973; Siler & Yen, 1973; Bell *et al*, 1973; Yen *et al*, 1973; Zarate *et al*, 1973). The cause of the increased release of LHRH in mania remains unknown but is likely to involve the stimulatory effect of either increased central α-noradrenergic activity or reduced central opioid activity (Kalra & Kalra, 1983). As was also the case in acute drug-free manic men (Whalley *et al*, 1985), there were no significant differences between the manic and control subjects in the plasma concentrations of FSH. Neither was there a between-group difference in the plasma FSH response to LHRH. Possible explanations for the dissociation between LH and FSH secretion have been discussed previously (Whalley *et al*, 1985).

Most recovered patients were on long-term lithium therapy and this could be a possible explanation of the differences between the two groups of subjects. Several factors, however, suggest that this is not the case. In our earlier study (Whalley *et al*, 1985), we detected increased plasma LH concentrations in young men with acute mania, none of whom had received lithium therapy, and there are no data available in the literature indicating that lithium increases plasma LH concentrations (Brown *et al*, 1981) or that lithium increases the LH response to LHRH (Joffe *et al*, 1986). The present study is, therefore, consistent with our earlier report, although the extent of the increase is much less in the recovered patients than in the subjects with acute mania. Our earlier findings also prompted several studies by members of our group on the effects of lithium on hypothalamic–pituitary–gonadal function. Follow-up studies on small numbers of patients with acute mania treated by lithium provided no suggestion that lithium increases plasma LH concentrations (some of these patients are reported in the earlier study by Whalley *et al*, 1985). Although lithium affects the turnover of phosphatidyl inositol (Gonzales & Crews, 1984), which in turn mediates some of the actions of LHRH (Naor *et al*, 1984), experimental studies in our laboratory show that lithium does not affect the releasing or priming actions of LHRH *in vitro* (M. Johnson, P. R. Mitchell & G. Fink, unpublished).

In sum, the present data show that increased basal plasma LH concentrations and an increased LH response to LHRH may be a state-independent feature of manic-depressive illness. The persistent abnormality may be a disorder of the central neural control of LHRH release, possibly involving monoaminergic or opioid neurotransmission. Since a catecholaminergic mechanism is also involved in the release of melatonin from the pineal gland, it is tempting to speculate that there may be an association between the two apparent trait markers of mania, the increased LH release which we reported and the supersensitivity to light in terms of melatonin release reported recently (Lewy *et al*, 1985).

References

Aiyer, M. S., Chiappa, S. A. & Fink, G. (1974) A priming effect of luteinising hormone releasing factor on the anterior pituitary gland in the female rat. *Journal of Endocrinology*, **62**, 573–588.

Bell, J., Spitz, I., Slonim, A., Perlman, A. Segal, S., Palti Z., Rabinowitz, D. (1973) Heterogeneity of gonadotropin response to LHRH in hypogonadotropic hypogonadism. *Journal of Clinical Endocrinology and Metabolism*, **36**, 791–794.

Brown, G. M., Grof, E. & Grof, P. (1981) Neuroendocrinology of depression, a discussion. *Psychopharmacology Bulletin*, **17**, 10–12.

Fink, G. (1979) Neuroendocrine control of gonadotrophin secretion. *British Medical Bulletin*, **35**, 155–160.

Gonzales, R. A. & Crews, F. J. (1984) Characterisation of the cholinergic stimulation of phosphoinositide hydrolysis in rat brain slices. *Journal of Neuroscience*, **4**, 3120–3127.

Gual, C. (1973) Clinical effects and uses of hypothalamic releasing and inhibiting factors. In *Frontiers in Neuroendocrinology* (eds W. F. Ganong, L. Martini). New York, London and Toronto: Oxford University Press.

Isurugi, K., Wakabayashi, K., Fukutani, K., Takayasu, H., Tamoki, B.-I. & Okada, M. (1973) Responses of serum luteinising hormone and follicle-stimulating hormone levels to synthetic luteinising hormone-releasing hormone (LH-RH) in various forms of testicular disorders. *Journal of Clinical Endocrinology and Metabolism*, **37**, 533–539.

Joffe, R. T., Post, R. M., Ballenger, J. C., Rebar, R., Gold, P. W. (1986) The effects of lithium on neuroendocrine function in affectively ill patients. *Acta Psychiatrica Scandinavica*, **73**, 524–528.

Kalra, S. P. & Kalra, P. S. (1983) Neural regulation of luteinising hormone in the rat. *Endocrine Reviews*, **4**, 311–351.

Lewy, A. J., Nurnberger, J. I., Wehr, T. A., Pack, D., Becker, L. E., Powell, R. L., Newsome, D. A. (1985) Supersensitivity to light: possible trait marker for manic-depressive illness. *American Journal of Psychiatry*, **142**, 725–727.

Naor, Z., Molcho, J., Zilberstein, M. & Zakut, H. (1984) Phospholipid in gonadotropin-releasing hormone target cells: comparative studies. In *Hormonal Control of the Hypothalamo-Pituitary–Gonadal Axis* (eds K. W. McKerns & Z. Naor). New York: Plenum Press.

Roth, J. C., Kelch, R. P., Kaplan, S. L., Grumbach, M. M. (1972) FSH and LH response to luteinising hormone releasing factor in prepubertal and pubertal children, adult males and patients with hypogonadotropic and hypergonadotropic hypogonadism. *Journal of Clinical Endocrinology and Metabolism*, **35**, 926–930.

Siler, T. M. & Yen, S. S. C. (1973) Augmented gonadotropin response to synthetic LRF in hypogonadal state. *Journal of Clinical Endocrinology and Metabolism*, **37**, 491–494.

Spitzer, R. L., Endicott, J. & Robins, E. (1975) *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders*. New York: New York State Department of Mental Hygiene, New York State Biometrics Institute, Biometrics Research.

Whalley, L. J., Christie, J. E., Bennie, J., Dick, H., Blackburn, I. M., Blackwood, D., Sanchez Watts, G. & Fink, G. (1985) Selective increase in plasma luteinising hormone concentrations in drug free young men with mania. *British Medical Journal*, **290**, 99–102.

Winer, B. J. (1971) *Statistical Principles in Experimental Design*. New York: McGraw Hill.

Yen, S. S. C., Rebar,R., Vandenberg, G. & Judd, H. (1973) Hypothalamic amenorrhea and hypogonadotropinism: response to synthetic LRF. *Journal of Clinical Endocrinology and Metabolism*, **36**, 811–816.

Zarate, A., Kastin, A. J., Soria, J., Canales, E. S. & Schally, A. V. (1973) Effect of synthetic luteinising hormone-releasing hormone (LH-RH) in two brothers with hypogonadotropic hypogonadism and anosmia. *Journal of Clinical Endocrinology and Metabolism*, **36**, 612–614.

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Lithium in the Treatment of Aggression in Mentally Handicapped Patients

A Double-Blind Trial

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In a double-blind trial lasting 4 months in 42 mentally handicapped patients, the effect of lithium on aggression was assessed in comparison with placebo. In the lithium-treated group, 73% of patients showed a reduction in aggression during treatment. There were significant differences in mean weekly aggression scores and in the frequency of aggressive episodes between the lithium and placebo groups. Side-effects were noted in 36% of the lithium group (and 20% of the placebo group), but were mainly transitory. There were no episodes of toxicity, and no patients had to be withdrawn from the trial. Lithium appears to be worth a 2-month trial in such patients, where repeated aggression has not been relieved by more appropriate placement, occupation or company.

Lithium has been reported to reduce aggressive behaviour in prisoners and delinquents (Sheard, 1971; Tupin *et al*, 1973; Sheard, 1975; Sheard *et al*, 1976) and in mentally retarded patients (Forssman & Walinder, 1969; Dostal & Zvolsky, 1970; Morrison *et al*, 1973; Cooper & Fowlie, 1973; Micev & Lynch, 1974; Worrall *et al*, 1975; Lion *et al*, 1975; Goetzl *et al*, 1977; Dale, 1980; Sovner & Hurley, 1981; Craft & Mathews, 1984; Tyrer *et al*, 1984). In 1981, Sovner & Hurley reviewed the publications to date on the use of lithium carbonate in mentally handicapped individuals and concluded that more controlled studies needed to be carried out before any definitive statements about lithium's effectiveness could be made. The object of this work was to assess the efficacy of lithium carbonate in the control of aggression in mentally handicapped patients, under the conditions of a double-blind trial against placebo.

Method

Five centres participated in the trial, contributing a total of 42 patients. All were in-patients, exhibiting levels of aggression or self-mutilation severe enough to require routine drug therapy. Patients were excluded from the trial if they were already receiving treatment with lithium. A general physical examination was carried out before trial entry and patients in whom lithium treatment was contra-indicated were excluded, as were any patients with a cyclical pattern to their disorder.

The trial protocol was approved by the ethical committee of each participating centre. The nature of the trial and the procedures involved were explained to each patient (at an appropriate level) and to a relative or responsible guardian. Their consent was required before the patient could be included in the trial.

The trial was preceded by a 4-week run-in period during which all patients received treatment with two placebo tablets once daily. Patients were then allocated to two parallel groups to receive either lithium carbonate or placebo for a period of 12 weeks. Allocation to either the active or placebo group was made by minimisation, a sophisticated form of randomisation which provides for optimum balancing of patient groups in respect of factors which might affect the response to treatment (Taves, 1974). The minimisation procedure was carried out using a microprocessor, into which details of each patient were entered.

During the run-in period and the 12 weeks of the trial, the level of aggression was assessed daily by the nursing staff on duty, who differed during the study, but who all used the five-point scale devised by Dale (1980): 1 = well behaved; 2 = mood uncertain; 3 = overt aggression or attempted aggression; 4 = additional medication required to control patient; 5 = seclusion required. Patients who did not exhibit at least two episodes of overt aggression in two separate weeks of the run-in period were not entered into the trial.

The medical officer assessed each patient at the start of the run-in period, 2 weeks later, at trial entry and after 1, 2, 3, 6, 9 and 12 weeks of the trial. Before trial entry a blood sample was taken for estimation of blood urea, serum creatinine and either protein-bound iodine or serum thyroxine. At each medical assessment blood was taken for the estimation of serum lithium concentration and serum creatinine. The occurrence of possible side-effects of lithium treatment and of enuresis or epileptic fits was recorded.

The starting dose of trial medication was a single daily dose of two 400 mg tablets of a controlled-release formulation of lithium carbonate ('Priadel') or matching placebo. Dosage was subsequently adjusted to maintain serum lithium levels in the recommended therapeutic range

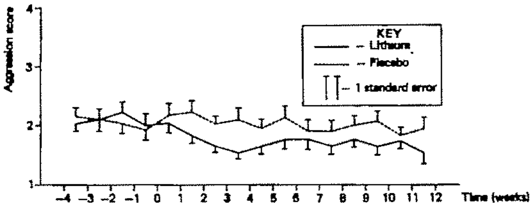

FIG. 1 Group means and standard errors of weekly mean aggression scores.

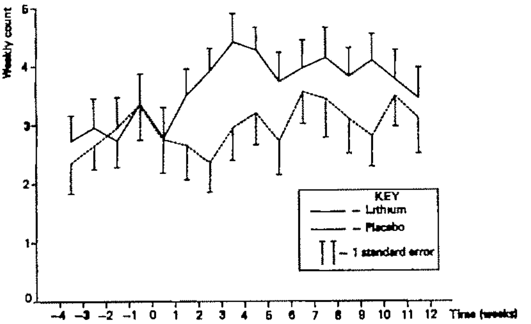

FIG. 2 Group means and standard errors of weekly counts of aggression score = 1.

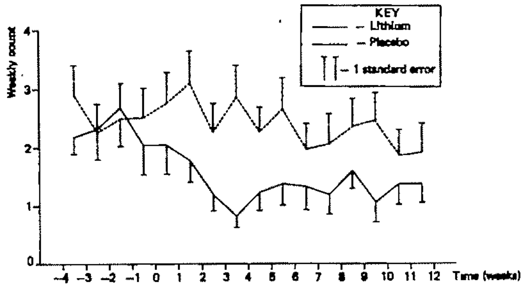

FIG. 3 Group means and standard errors of weekly counts of aggression score = 3 or more.

(0.7–1.0 mmol/litre 12 hours post-dose). The pathologist or another doctor at each centre provided dummy results for serum lithium concentrations for placebo patients in order to retain blindness. Other medication currently being given (e.g. tranquillisers, anti-convulsants) was continued as required but, as far as possible, no new treatment was started during the trial. Any additional medication needed was recorded. Patients could be withdrawn from the trial in the event of severe symptoms requiring other treatment or severe side effects causing concern.

In the statistical analysis of mean aggression scores and counts, the repeated measures nature of the data was taken into account, and repeated measures analysis was carried out using the BMDP 2V repeated measures program. The difference between the treatment groups in terms of response to treatment was assessed using the χ^2 test.

Results

Table I shows the background data for patients in the two treatment groups, which are seen to be comparable for all the factors listed, most of which were taken into account in the process of minimisation.

Since the level of aggression varied greatly from one day to the next, weekly mean aggression scores were calculated for each patient by adding his daily aggression scores for each week and dividing by seven. Figure 1 shows the means of these weekly mean aggression scores for the two groups. Whereas the weekly mean aggression scores are comparable during the run-in period, from the start of active treatment

TABLE I
Background data for the treatment groups

	Placebo (n = 20)	*Lithium (n = 22)*
Age: years		
Median	32.5	33
Range	22–55	19–65
Weight: kg		
Median	54.0	60.5
Range	42–72	39–87
Sex		
Male	14	15
Female	6	7
Severity of handicap		
Mild	1	3
Severe	19	19
Duration of stay in hospital: years		
Median	12	13
Range	0–26	2–49
Aggressive	11	11
Self-mutilating	0	1
Aggressive and self-mutilating	9	10
Family history of psychiatric illness		
Yes	3	3
No	7	6
Unknown	10	13
Centre		
Bryn-y-Neuadd	6	6
Llwyn View	1	2
Ely	2	1
Leavesden	6	7
Leicester Frith	5	6

the weekly mean aggression scores for the lithium group start to fall, and remain below the weekly mean aggression scores for the placebo group for the duration of the trial (i.e. the average level of aggression is lower in the lithium group than in the placebo group during the trial period). Repeated measures analysis, using the mean aggression score over the run-in period as a covariate for each patient, demonstrated a significance difference, almost at the 1% level ($P = 0.012$) between the global treatment means. The covariate correlated highly with the subsequent patient responses $P < 0.001$ and was therefore worth including in the analysis.

Figure 2 shows the mean of weekly counts of aggression scores of 1; that is, the mean number of days in each week that the patients in each group were well behaved, showing no aggression. The groups show comparable counts during the run-in period, but during the trial period the weekly counts of 1 are higher in the lithium group than in the placebo group (i.e. patients on lithium were well behaved on more days of each week of the trial period than patients on placebo). On repeated measures analysis the group means are not significantly different at the 5% level ($P = 0.082$), although the covariate correlates well with subsequent patient records ($P < 0.001$).

Figure 3 shows the mean of weekly counts of an aggression score of 3 or more for each treatment group (i.e. the mean number of days in each week that a score of 3 or more, indicating overt aggression, was recorded in each patient group). During the run-in period the two groups do not differ greatly, and indeed the counts cross over, but during the trial proper they separate and there are fewer counts of 3 or more occurring in the lithium group than in the placebo group. (Clinically, this means that in patients on lithium, aggressive outbursts were recorded less frequently than in patients on placebo.) Repeated measures analysis revealed a significant difference between the group means at the 1% level ($P = 0.002$), with the covariate correlating well with subsequent patient records ($P < 0.001$).

The tendency for patients in the placebo group to show a gradual improvement (in terms of lower mean weekly aggression counts of 3, and higher mean weekly aggression counts of 1) during the trial, may have been due to the inevitable lapses in daily recording as time went on, with progressive under-reporting of aggressive episodes. This would have tended to favour the placebo group, in which aggression was more prevalent during the treatment period, rather than the lithium group.

Individual evaluation was made of the daily aggression scores during the study. In the lithium group 16 patients showed reduced aggression during treatment (73%), two patients showed increased aggression (9%) and four patients showed no change (18%). In the placebo group six patients (30%) showed some reduction in the level of aggression during the trial period compared with the run-in period (50%) showed no change. This difference in response, four patients showed increased aggression (20%) and ten patients between the two treatment groups is shown to be significant on applying the χ^2 test to the resulting contingency table ($P = 0.03$).

Side-effects (thirst, polyuria, inco-ordination, tremor, drowsiness, and vomiting) were reported in eight patients on lithium (36%) but were transitory apart from one case of a patient who developed a persistent tremor, with serum lithium levels of 0.76–1.25 mmol/litre, for whom concurrent procyclidine was prescribed. In one patient lithium treatment was discontinued on account of vomiting, but was subsequently recommenced without problems and the vomiting was attributed to intercurrent infection. 'Lithium side-effects' were also reported in four patients in the placebo group (20%). There were no episodes of toxicity and no patients were withdrawn from the study.

Of the four patients with enuresis in the lithium group, two showed increased frequency of enuresis during lithium treatment, one showed a reduction in frequency and in one patient (whose aggression was dramatically controlled by lithium) enuresis ceased completely. In the placebo group one patient with enuresis was reported to be better during the trial and one was worse.

The only patient subject to fits was in the lithium group, and showed no change in the frequency of seizures during the trial period.

The initial daily dose of 800 mg lithium carbonate was not sufficient, in most cases, to bring the serum lithium concentration above 0.7 mmol/litre, and subsequent dosage adjustments were needed. A serum lithium concentration of 0.7–1.0 mmol/litre seemed necessary for clinical effect. Higher levels than this tended to be associated with tremors or inco-ordination. The highest serum lithium concentration recorded during the trial was 1.25 mmol/litre.

Where serum lithium levels were adequate, a response to treatment was sometimes seen within the first 2 weeks, but in other cases improvement was not apparent for 6–8 weeks. There was a tendency towards lower serum lithium levels at the end of the trial, reflecting either dosage reduction or possibly lack of compliance, which may explain the slight increase in aggression noted in some lithium patients towards the conclusion of the study.

The administration of additional medication to control aggression was recorded in three patients in the lithium group and six patients in the placebo group.

It is probably misleading in a trial of this size to draw conclusions regarding the response to lithium therapy of any particular patient subgroup. However, it was noted that all the patients in the lithium group with high scores for aggression during the run-in period responded particularly well to treatment.

Discussion

In this double-blind trial there was a reduction in aggressiveness in 73% of mentally handicapped patients treated with lithium. There was a statistically significant difference between the treatment groups in terms of the weekly mean aggression scores, and the weekly counts of aggressive episodes during the trial period. It is worth noting that these were difficult patients, many of whom had not responded to other therapies.

This confirms the observations and conclusions of previous investigators who have studied the effects of lithium in aggressive mentally handicapped patients (see the introduction to this paper) and have reported a beneficial effect in 50–88% of such patients.

Obviously the benefits of lithium therapy in the mentally handicapped have to be weighed against the risks, but as in-patients they can be carefully supervised so that the danger of lithium intoxication should be minimal. In this series, classical side-effects were noted in 36% of the patients in the lithium group, but these were mainly short-lived and in no case necessitated the discontinuation of lithium. The reporting of side-effects normally associated with lithium therapy in 20% of the patients in the placebo group was interesting as it showed that blindness was maintained, although it is sometimes claimed that this is not possible in trials with lithium. The side-effects occurring in placebo-treated patients were probably due to other drug therapy on which they were currently maintained.

The effect of lithium treatment on pre-existing enuresis was variable, two patients becoming worse during lithium treatment and two showing improvement. In this respect our series of patients did not reflect the problems of excessive thirst and bed-wetting described by Dostal and Zvolsky (1970), but confirmed the observation by Dale (1980) that lithium treatment may be associated with cessation of enuresis in a patient whose aggression is successfully controlled.

One patient with fits showed no exacerbation during lithium treatment.

From this study, and from previous work (Dostal & Zvolsky, 1970; Sheard, 1975; Goetzl *et al*, 1977; Sovner & Hurley, 1981) a 12-hour serum lithium concentration of 0.7–1.0 mmol/litre should be aimed at for clinical effect. Higher levels were associated with tremor and inco-ordination in our patients, and with neurotoxicity in an earlier trial by Worrall *et al* (1975), although these authors attributed the signs in their two patients to the combination of lithium and underlying brain damage. Most aggressive patients (perhaps because they tend to be hyperactive) need more than 800 mg/day lithium carbonate as a single daily dose to achieve therapeutic levels, and may require as much as 3000 mg/day (Dale, 1980). Once such levels are reached, a clinical response may become evident in 2–8 weeks. A controlled release formulation suitable for once-daily dosage (e.g. Priadel) is obviously easier to administer to difficult patients, and in long-term use is less likely to cause side-effects or renal changes (Plenge *et al*, 1982).

In a cross-over trial in 25 mentally handicapped patients, Tyrer *et al* (1984) used multiple correlation of placebo–lithium factor scores on background variables in an attempt to identify factors associated with a good response to lithium. They concluded that improvement was less likely to occur in socially withdrawn male patients with frequent aggressive outbursts. The identification of subgroups of responders or non-responders seems to have been a particular fascination of workers involved in trials of lithium. Manipulation of the data at the end of a trial invariably yields such subgroups, which may be meaningful but are frequently spurious findings, particularly in small trials. There is a danger that such findings become accepted dogma, and patients who might respond to lithium may therefore not be considered for such treatment. As if to illustrate this, our patients with high initial aggression scores responded particularly well to lithium, and both male and female patients were well represented among the responders. Lithium therapy is therefore worth a 2-month trial in any patient whose repeated aggression or self-mutilation is proving difficult to control.

This double-blind trial, together with the now considerable experience of earlier workers in this field, should serve to establish the place of lithium as a potentially useful agent in the management of those mentally handicapped patients whose uncontrollable aggressiveness makes long-term stays in hospital necessary.

References

COOPER, A. F. & FOWLIE, H. C. (1973) Control of gross self-mutilation with lithium carbonate (letter). *British Journal of Psychiatry*, **122**, 370–371.

CRAFT, M. & MATHEWS, J. (1984) Lithium in mental handicap: a review. *Mental Handicap*, **12**, 117–118.

DALE, P. G. (1980) Lithium therapy in aggressive mentally subnormal patients. *British Journal of Psychiatry*, **137**, 469–474.

DOSTAL, T. & ZVOLSKY, P. (1970) Antiaggressive effect of lithium salts in severe mentally retarded adolescents. *International Pharmacopsychiatry*, **5**, 203–207.

FORSSMAN, H. & WALINDER, J. (1969) Lithium treatment on atypical indication. *Acta Psychiatrica Scandinavia*, **20**, 34–40.

GOETZL, U., GRUNBERG, F. & BERKOWITZ, B. (1977) Lithium carbonate in the management of hyperactive aggressive behavior of the mentally retarded. *Comprehensive Psychiatry*, **18**, 599–606.

LION, J. R., HILL, J. & MADDEN, D. J. (1975) Lithium carbonate and aggression: a case report. *Diseases of the Nervous System*, 36, 97–98.

MICEV, V. & LYNCH, D. M. (1974) Effect of lithium on disturbed severely mentally retarded patients (letter). *British Journal of Psychiatry*, **125**, 110.

MORRISON, S. D., ERWIN, C. W., GIANTURCO, D. T. & GERBER, C. J. (1973) Effect of lithium on combative behavior in humans. *Diseases of the Nervous System*, **34**, 186–189.

PLENGE, P., MELLERUP, E. T., BOLWIG, T. G., BRUN, C., HETMAR, O., LADEFOGED, J., LARSEN, S. and RAFAELSEN; O. J. (1982) Lithium treatment: does the kidney prefer one daily dose instead of two? *Acta Psychiatrica Scandinavia*, **66**, 121–128.

Sheard, M. H. (1971) Effect of lithium on human aggression. *Nature*, **230**, 113–114.

Sheard, M. H. (1975) Lithium in the treatment of aggression. *Journal of Nervous and Mental Disease*, **160**, 108–118.

Sheard, M. H., Marini, J. L., Bridges, C. I. & Wagner, E. (1976) The effect of lithium on impulsive aggressive behavior in man. *American Journal of Psychiatry*, **133**, 1409–1413.

Sovner, R. & Hurley, A. (1981) The management of chronic behavior disorders in mentally retarded adults with lithium carbonate. *Journal of Nervous and Mental Disease*, **169**, 191–195.

Taves, D. R. (1974) Minimization: A new method of assigning patients to treatment and control groups. *Clinical Pharmacology and Therapeutics*, **15**, 443–453.

Tupin, J. P., Smith, D. B., Clanon, T. L., Kim, L. I., Nugent, A. & Groupe, A. (1973) The long-term use of lithium in aggressive prisoners. *Comprehensive Psychiatry*, **14**, 311–317.

Tyrer, S. P., Walsh, A., Edwards, D. E., Berney, T. P. & Stephens, P. A. (1984) Factors associated with a good response to lithium in aggressive mentally handicapped subjects. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, **8**, 751–755.

Worrall, E. P., Moody, J. P. & Naylor, G. J. (1975) Lithium in non-manic-depressives: antiaggressive effect and red blood cell lithium values. *British Journal of Psychiatry*, **126**, 464–468.

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Brief Reports

Mania Following Head Injury

A case of mania subsequent to head injury is presented. The literature reporting cases of mania after head injury is reviewed. It is suggested that the concept of Secondary Mania be broadened to include cases occurring after head injury. It is also suggested that a true bipolar illness can be secondary.

Manic-Depressive Disorder is usually regarded as a 'functional' disorder, of unknown aetiology. However, depression occurring secondary to medical illness has long been described and Feighner *et al* (1972) gave the concept nosological status in their classification of the affective disorders. Krauthammer & Klerman (1978) introduced the concept of Secondary Mania. They described mania resulting from drugs, infection, neoplasm, epilepsy and metabolic disturbances. They distinguished such a syndrome from mania which occurs as a phase of idiopathic manic-depressive disorder. They argue that 'mania is best considered a clinical syndrome of multiple aetiology'. A case of mania resulting from head injury which fits their criteria for secondary mania is described. A search of the literature provided 20 other cases of a manic psychosis following head injury.

Case report

A 48-year-old female entertainer fell from a trapeze and received multiple injuries in July 1985. In addition to fractured ribs, vertebra and iliac crest she also injured her head and lost consciousness for approximately 10 minutes. Skull X-rays showed no fracture. However, she had post-traumatic amnesia of approximately 3 days and developed bilateral fourth cranial nerve palsies with diplopia. On recovery from her physical injuries she was confused and disorientated but CT brain scan was normal. Over the next few days she became overactive and grandiose and was transferred to the psychiatric department. This was 3 weeks after her fall. On admission here she was fully orientated but displayed marked pressure of speech and flight of ideas. Although she was noted to laugh and cry alternatively she reported feeling 'very happy', 'super' and 'very strong', and told us that she was writing a book about her experiences which was sure to be a best-seller. After admission she did not sleep for long at night and insisted on doing 'exercises', requiring restraint on a few occasions. She had no previous psychiatric history and family history was likewise negative. She had suffered a fall 2 years previously and had fractured her pelvis but had no psychiatric problems.

She was treated with chlorpromazine and haloperidol and gradually settled over the following 8 weeks. However, she then developed a depressive episode with sadness, tearfullness and insomnia. This episode responded well to mianserin. However, her diplopia persisted. Prior to discharge an EEG was performed which was normal. Psychological testing showed her to have an IQ of 85 (Mill Hill Vocabulary Test/Ravens Progressive Matrices). Test results on the Wechsler Memory Scale suggested "possible residual deficits in the areas of visual memory and retention of new unfamiliar verbal material". Also, her result on the Benton Visual Retention Test showed some residual deficit in the visual spatial area.

She was quite well by October 1985 and was discharged to return to the UK. She was lost to follow up.

Literature review

A series of 44 post-head-injury patients described by Tennent (1937) included two with a definite diagnosis of mania. Hillbom (1951) and Achte *et al* (1969) reporting the long-term follow-up of a very large number of brain-injured Finnish war veterans identified seven cases of manic depressive disorder. Hoheisel & Walch (1952) described five cases of post-traumatic manic-depressive psychosis. Four of these patients had a positive family history. Parker (1957) described a patient who developed a manic depressive psychosis subsequent to head injury at the age of 41. He had no previous psychiatric history and no family history. However, Parker points out that there were 'emotional factors' in this case which could have been at least partly responsible for his psychiatric disorder. The autopsy of a 49-year-old man who developed a manic-depressive illness after head injury at the age of 41 was described by Deshimaru *et al* (1977). They speculated that his manic-depressive disorder was due to bruising of the bilateral orbital surfaces of the frontal lobes. Cohn *et al* (1977) described a 12-year-old boy who developed 'agitated hypomanic behaviour' after he sustained a closed head injury. He was treated successfully with lithium. A 39-year-old man who developed a bipolar disorder after injury to his right hemisphere was described by Gregersen & Hagen (1983). Lithium had a stabilising effect on his mood. A 23-year-old woman who developed a manic-depressive illness responsive to chemotherapy was described by Sinanan (1984). Khanna & Srinath (1985) described a 17-day episode of mania occurring in a 13-year-old girl with no previous psychiatric history and no family history of affective illness.

Discussion

Whilst the patient described here had no abnormality identified on CT brain scan the fact that she had residual bilateral fourth nerve palsies and some residual cognitive deficits indicates some degree of organic impairment. She was also rendered unconscious by the injury which implies some disruption of the deep structures of the brain (Cairns, 1952). If her affective illness was associated with this impairment it must be regarded as a secondary affective illness in the same way that affective illness occurring due to neoplastic structural damage is secondary. It is unlikely that a 'latent' affective illness was merely precipitated by the head injury as she was aged 48 at the time of the injury and had no previous psychiatric illness and no family history of affective illness. (The cases described above of Parker, Deshimaru, Cohn, Gregersen, Sinanan and Khanna were similar. In most of the others no information on past history or family history is available.) The fact that her psychiatric illness occurred directly after her head injury with no lucid normothymic period intervening also indicates that the head injury was directly causative. It is generally accepted that the shorter the interval between a head injury and the development of a psychosis the stronger is the likelihood of the two conditions being linked (Weller, 1985). This case fulfils the criteria for Secondary Mania as outlined by Krauthammer & Klerman (1978) and thus supports Khanna & Srinath (1985) when they argue that head injury should be included as an aetiological category for Secondary Mania. The patient described here suffered from a depressive episode following the manic phase. Most of the post-traumatic patients described in the literature also developed a bipolar disorder. This would indicate that not only unipolar depression and unipolar mania can sometimes be subsequent to physical illness but that this is also true of a bipolar state. This is supported by Jampala & Abrams (1983) who report the case of a man who developed a bipolar illness after a right-sided cerebral infarction. Oyewumi & Lapierre (1981) report a case of bipolar disorder secondary to brain stem injury and Forrest (1982) described bipolar illness after a right hemispherectomy. This is further evidence against what Krauthammer & Klerman (1978) call the 'one agent-one syndrome' model of causation and supports the idea that unipolar and bipolar affective disorder may be caused by differing pathophysiological pathways.

Further research is needed to clarify whether bipolar disorder subsequent to head injury follows the same course as idiopathic manic-depressive disorder. However, the impression from the literature is that such is the case and that secondary bipolar disorder can be chronic and episodic. Prophylaxis with lithium has generally been shown to be successful in cases of affective disorder secondary to structural lesions (Oyewumi & Lapierre, 1981); Rosenbaum & Barry, 1975; Cohen *et al*, 1980). However, Jampala & Abrams (1983) suggest that carbamazepine may be the 'rational choice' for the treatment of Secondary Mania.

The prognosis for a patient who develops mania secondary to head injury must remain guarded and possibly lithium or carbamazepine prophylaxis should be routinely considered.

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References

ACHTE, K. A., HILLBOM, E. & AALBERG, V. (1969) Psychoses following war brain injuries. *Acta Psychiatrica Scandinavica*, **45**, 1–18.

CAIRNS, H. (1952) Disturbance of consciousness in lesions of the mid-brain and diencephalon. *Brain*, **75**, 109.

COHEN, M. R., NISKA, R. W. (1980) Localized right cerebral hemisphere dysfunction and recurrent mania. *American Journal of Psychiatry*, **137**, 847–848.

COHN, C. K., WRIGHT, J. R. & DE VAUL, R. A. (1977) Post-head-trauma syndrome in an adolescent treated with lithium carbonate—case report. *Diseases of the Nervous System*, **38**, 630–631.

CUMMINGS, J. L. & MENDEZ, M. F. (1984) Secondary mania with focal cerebrovascular lesions. *American Journal of Psychiatry*, **141**, 1084–1087.

DESHIMARU, M., MIYAKAWA, T. & SUZUKI, T. (1977). An autopsy case of head injury with a manic depressive state. *Brain Nerve (Tokyo)*, **29**, 787–790.

FEIGHNER, J. P., ROBINS, E., GUZE, S. B. *et al* (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

FORREST, D. V. (1982). Bipolar illness after right hemisphrectomy. *Archives of General Psychiatry*, **39**, 817–819.

GREGERSEN, B. & HAGEN, S. (1983) Secondary Mania after cerebral injury. *Ugeskr Laeger*, **145**, 3661.

HILLBOM, E. (1951) Schizophrenia-like psychoses after brain trauma. *Acta Psychiatrica et Neurologica Scandinavica*, **60**, 36–47.

HOHEISEL, H. P. & WALCH, R. (1952) Uber manisch-depressive und verwanate Verstimmungszustande nach hirnverletzung. *Archiv fur Psychiatrie und Zeitschrift Neurologie*, **188**, 1–25.

JAMPALA, V. C. & ABRAMS, R. (1983) Mania secondary to left and right hemisphere damage. *American Journal of Psychiatry*, **140**, 1197–1199.

KHANNA, S. & SRINATH, S. (1985). Symptomatic Mania after minor head injury. *Canadian Journal of Psychiatry*, **30**, 236–237.

KRAUTHAMMER, C. & KLERMAN, G. (1978) Secondary Mania. *Archives of General Psychiatry*, **35**, 1333–1339.

OYEWUMI, L. K. & LAPIERRE, Y. D. (1981) Efficacy of lithium in treating mood disorder occurring after brain stem injury. *American Journal of Psychiatry*, **138**, 110–112.

PARKER, N. (1957) Manic depressive psychosis following head injury. *Medical Journal of Australia*, **2**, 20–22.

ROSENBAUM, A. M. & BARRY, M. J. (1975) Positive therapeutic response to lithium in hypomania secondary to organic brain syndrome. *American Journal of Psychiatry*, **132**, 1072–1073.

SINANAN, K. (1984) Mania as a sequel to a road traffic accident. *British Journal of Psychiatry*, **144**, 330–331.

TENNENT, T. (1937) Discussion on mental disorder following head injury. *Proceedings of the Royal Society of Medicine*, **30**, 1092–1093.

WELLER, M. P. I. (1985). The sequelae of head injury and the post concussion syndrome. In *Recent Advances in Clinical Psychiatry*, (ed. K. Granville-Grossman). London: Churchill Livingstone.

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Compensation Psychosis

Kennedy (1946) stated that "A compensation neurosis is a state of mind, born out of fear, kept alive by avarice, stimulated by lawyers and cured by a verdict". This theme, supported by Miller's influential paper in 1961, has ensured that generations of patients have been regarded with suspicion if they dared to present with psychological symptoms following an accident. The view remained unchallenged for over a decade and is still often presented to, and accepted by, our Courts, despite the fact that Miller reached his conclusions after examining patients who presented for legal reports in whose cases it was not surprising that he found a relationship between compensation and psychological sequelae. The psychological effects of proceeding litigation on the victim of an accident remain a matter of current debate. McKinley *et al* (1983), reported differences between those patients suffering a severe blunt head injury claiming compensation and those not claiming compensation. The reports given by relatives of changes in both patients were very similar. However, reports given by patients themselves differed with claimants reporting slightly more symptoms than non-claimants. In a study by White in 1981, the author followed up 163 victims of accidents admitted to the Birmingham Accident Hospital (76 burns and 87 general accidents). One year after their accident, psychological sequelae were found in approximately two-thirds of the group, one-third being moderately-to-severely psychologically affected. There was no statistical difference between those victims in whom the accident had given rise to litigation and in whom the case was still proceeding and those victims where compensation was not an issue.

Miller also reported that symptoms disappeared following compensation, but again recent studies disagree with this finding (Merksey & Woodforde, 1972; Kelly & Smith, 1981). Merksey & Woodforde (1972) report that neuronal loss and axonal damage has been documented even after mild head injury suggesting therefore, that, when a head injury results from the accident, some symptoms might be understood in organic terms allowing further room for misunderstanding and disagreement.

Case history

Mr H.T. was aged 51 at the time of his accident in 1982. Whilst working at a building site a 35 lb thermolite block was thrown from a first floor window down towards a skip on the ground below. On route it struck him a glancing blow on the back of his head, pushing his head forward towards the skip. It is uncertain whether or not he was rendered unconscious, he had a pre-traumatic amnesia of only a few minutes and the length of his post-traumatic amnesia is uncertain. His skull was not fractured and, although he was taken to hospital after the accident, he was not admitted.

The workman who threw the thermolite block accepted full responsibility, explaining that he had not seen Mr H.T. when he threw it. The blame was also accepted by the company. Our patient bore no resentment towards his fellow-worker, though he did feel that his employers had been perhaps less than sympathetic.

In the weeks that followed the accident, he experienced feelings of unreality, during which he would touch various objects to ensure that they really did exist. According to his wife he needed constant help and guidance, behaving rather like a child. On occasions, she would even have to do up his shoelaces. He was irritable, argumentative and easily upset. He complained that his memory was impaired, and also of being depressed, saying that he felt a sense of frustration and despair due to his memory impairment. He retained a good appetite and he did not describe sleep disturbance or diurnal variation. Whereas previously he had suffered no disturbance of sexual function, following the accident he was unable to sustain an erection.

Our patient had suffered no previous psychiatric illness and there was no history of any such illness in the family. Prior to his accident he was described as a hardworker, conscientious, and enjoying a good sense of humour.

He is the third of eight children and enjoyed a happy, normal childhood. He attended school until the age of 14 and became an apprentice carpenter and joiner, receiving further education at technical college (day release and evening classes). He later became a foreman carpenter and then a site manager, the latter being his employment for 25 years. At the time of the accident he had been working for that particular building contractor for only a few months.

He married at the age of 24 and has one adult son with whom he gets on well. Both he and his wife stated that they had enjoyed an excellent relationship prior to the accident. The change in his behaviour subsequently did, however, affect it adversely. He has never drunk to excess and was not taking medication at the time of the accident.

Assessment and progress

Mr H.T. was initially referred by his general practitioner for treatment and only later did his solicitor request a legal report. It was felt that many of his symptoms could be understood in terms of a traumatic neurosis but that it was appropriate to investigate further. Both EEG and CT scan proved normal and there was no evidence of intellectual deterioration. His memory quotient, however, was significantly impaired. These tests were carried out on four occasions between 1982 and 1985. His full scale IQ (WAIS) varied between 118 and 121 with the memory quotient varying between 90 and 106. There was no evidence of progressive deterioration. EEG and CT scan repeated in 1985 again proved normal.

Following his accident in February, 1982, he was off work for approximately 5 months. After returning to work for 2 weeks he was unable to continue, becoming markedly upset because he could not recall giving or receiving instructions or whether he had completed various tasks. Following a fortnight at home he again returned to work, then once more found himself unable to cope with his duties, remaining at home for a further 6 weeks. He returned to work again, but in August 1983, became markedly anxious and depressed and was admitted to a psychiatric hospital. He returned to work in November 1983, and remained there until January 1984, at which time he was again admitted to hospital and has not worked since that time. Whilst at home, he was able to exercise some of his skills in the form of carpentry but would require his wife to help him as otherwise he could not remember where he had placed his tools and would constantly mislay them.

In October 1985, he became increasingly agitated and refused to leave his home as he felt convinced that neighbours were spying upon him and that the police were about to raid his house in order to look for evidence to use against him. He was admitted to hospital as an emergency. He freely and spontaneously stated that he felt people would spy upon him in order to interfere with his claim for compensation. He described how, during a previous hospital admission, he came to realise that a fellow patient, a solicitor by profession, had been planted by his former company's solicitors in order to spy upon him and obtain evidence to use against him in court. He believed that a neighbour, a director of a security firm had tricked him into helping him mend a car in order to prove that he was not ill. He suspected that his neighbours were reporting his actions back to the insurance company and was convinced that hidden cameras and microphones were recording his every move and utterance.

He was commenced on Trifluoperazine and within 7 days his paranoid symptomatology was much less evident and a few weeks later he was able to admit that all his previous beliefs must have been erroneous and were "more like a nightmare than reality". During his admission he ate and slept well, mixed with other patients and at no time appeared depressed or tearful. His compensation psychosis has not recurred but his memory deficit and symptoms of anxiety and depression remain some months following receipt of a £50 000 settlement.

Discussion

Following a head injury, this patient initially presented with three problems; (1) a post-traumatic neurosis, (2) a post-traumatic personality change as described by Brooks & McKinlay (1983) and (3) a stable impairment of memory with preservation of global intellectual function. Nearly 4 years after the injury he developed features of a paranoid psychosis, describing delusions whose content reflected his preoccupation with the current compensation claim and legal proceedings. His memory impairment could be explained as follows:

(a) that his memory loss was in essence a deliberate disability produced either to gain compensation in monetary terms or to achieve psychological advantage;

(b) the memory impairment was due to an hysterical amnesia i.e. was produced by unconscious dynamic mechanisms which served to protect the victim of this accident from certain fears which had been caused or resurrected by the accident;

(c) memory impairment resulted from cerebral damage.

The first possibility would seem to the authors to be unlikely. Individuals who deliberately produce poor results from psychometric testing tend either to do badly on all the tests or to produce erratic results. In this case tests of intellectual function were similar on each occasion. Initially, verbal scales were slightly higher than performance scales (initial two tests) later becoming virtually identical. Memory quotient remained abnormal when tested on four occasions over 5 years. An organic explanation is most likely and it is supported by the direction of the discrepancy between verbal and performance scales at the initial assessments. It would be hard to accept that a lay person could fake his responses across a wide battery of subtests in order to produce an organic looking pattern. The above tests cannot unequivocally distinguish between organic and hysterical amensia, but again the pattern of results would make the former most likely.

Hysterical amnesia is usually of short duration. The hallmark of hysteria is said to be the absence of psychological distress i.e. 'belle indifference' with the hysterical symptoms protecting the patient against the cause of his distress. In this case the memory loss lasted for 4 years. It remains despite the settlement of his compensation case: the memory difficulties have themselves been the greatest cause of distress to him.

The aetiology of his compensation psychosis occurring as described some 4 years after the accident might also be open to debate.

As a result of differing medical opinions and the failure of the insurance company to agree to early settlement of the case, our patient eventually saw six consultants and two psychologists. He became increasingly preoccupied and anxious over the legal proceedings and particularly upset when he felt there was any implication (by those acting for the insurance company) that he was choosing not to work. It could therefore be argued that the development of his psychosis was due to a natural progression of dynamic processes.

The second possibility is that he coincidentally suffered a psychotic illness and, because of his preoccupation, his delusions naturally took the form described.

A third explanation, the most controversial but perhaps the most interesting, postulates that the organic process which causes memory impairment has eventually led to the development of his psychosis. This accepts the effects of dynamic stresses but suggests an underlying organic abnormality. In this case the process would have caused functional abnormality (both memory impairment and psychosis) without evidence of structural damage as demonstrable by an EEG or CT scan. Such a process is described in the model hypothesised by Wexler (1986) who suggests processes which are intermediary constructs between anatomy and cellular physiology on the one hand, and subjective experience and behaviour on the other.

Whatever the explanation, this patient, like many who have the misfortune to suffer an accident, has experienced great distress. Perhaps in 1986 Kennedy's aphorism could be amended so that psychiatric illness following accidents could be said "to be born out of trauma, kept alive by lack of understanding, complicated by litigation, but requiring appropriate treatment".

References

Brooks, D. N. & McKinlay, W. W. (1983) Personality and behaviour change after severe blunt head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **46**, 336–344.

Kelly, R. & Smith, B. N. (1981) Post traumatic syndromes; another myth discredited. *Journal of the Royal Society of Medicine*, **74**, 275–277.

Kennedy, I. (1946) The mind of the injured worker, its effect on disability periods. *Compensation Medicine*, **1**, 19–24.

McKinlay, W., Brooks, D. N. & Bond, M. R. (1983) Post-concussional symptoms, financial compensation and outcome of severe blunt head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **46**, 1084–1091.

Merksey, M. & Woodforde, J. M. (1972) Psychiatric sequelae of minor head injury. *Brain*, **95**, 521–528.

Miller, H. (1961) Accident neurosis. *British Medical Journal*, **1**, 919–925.

Wexler, B. E. (1986) A model of brain function and its implication for psychiatric research. *British Journal of Psychiatry*, **148**, 357–362.

White, A. (1981) Psychiatric study of patients with severe burn injuries. *British Medical Journal*, **284**, 465–467.

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Oxygen Saturation during Electroconvulsive Therapy

Oxygen saturation was measured during anaesthesia and electroconvulsive therapy (ECT) using ear oximetry. Significant hypoxia occurred during some treatments; desaturation related to the number of ventilations performed after muscle relaxation and prior to the shock. Desaturation did not correlate significantly with seizure duration. These findings highlight the need for adequate oxygenation during ECT.

The major goal of anaesthesia for ECT is prevention of complications such as discomfort, fractures, aspiration of gastric contents, bradycardia and hypoxia. Monitoring of oxygenation is important during anaesthesia and, as clinical signs are often unreliable, ear oximetry is recommended as a more precise method of detecting hypoxia. The present study measured haemoglobin saturation during routine anaesthesia and ECT to assess the incidence of hypoxia under normal circumstances.

Method

Local Ethical Committee approval was obtained for the study and only patients unable or unwilling to consent were excluded. Four separate ECT sessions were studied, involving 40 patients who were grouped according to which anaesthetist was responsible for them. Anaesthetists were asked not to deviate from their usual practice and did not know what was being recorded; they were not allowed to see the oxygen saturations during the study. The apparatus was unobtrusive and did not interfere with the routine of the list. All patients were anaesthetised using methohexitone and suxamethonium without atropine. A flow of 6–10 litres per minute of 100% oxygen was invariably administered, using a Mapleson C circuit, without a carbon dioxide absorber. Positive pressure ventilation was performed following the administration of the muscle relaxant, both before and after the seizure. The number of positive pressure ventilations given prior to the shock and the time at which oxygen was administered after the seizure were recorded. The seizure duration was measured using the isolated arm technique (Fink & Johnson, 1982).

Oxygen saturation was measured using a Hewlett-Packard ear oximeter, which measures saturations in the range of 65–100%. The patient's ear was 'arterialised' by brisk rubbing for 15–30 seconds, and the probe was applied to the pinna. A potentiometric recorder made a continuous trace of the oxygen saturation during anaesthesia and ECT. The change in oxygen saturation was calculated by subtracting the lowest saturation reached after seizure and before the administration of oxygen from the baseline value.

Data were analysed using Pearson's Product Moment Correlation Coefficient and analysis of variance, followed by the Tukey test, where appropriate.

Results

Four anaesthetists were responsible for 10 patients each; 33 patients were female and 7 were male; the median age was 49 and the age range 21–78 years. The doses of methohexitone and suxamethonium used by each anaesthetist did not differ significantly (Table I).

TABLE I
The doses of anaesthetic drugs used in the four groups of patients

	Anaesthetist 1	2	3	4
Methohexitone: mg				
Mean	66	64	51	74
SD	24	22	17	28
Suxamethonium: mg				
Mean	45	30	35	35
SD	10	8	6	6

The range of oxygen saturations recorded during ECT was 79–100%. 16 subjects demonstrated a fall in oxygen saturation of more than 5% from the baseline value, lasting from 15–75 seconds. Prior to anaesthesia there were no significant differences in oxygen saturation between the four groups, and the degree of hypoxia was unrelated to initial oxygen saturation (Table II). Patients in group 1 remained better oxygenated than those in groups 2 ($P<0.01$), 3 ($P<0.01$) and 4 ($P<0.05$) (Table II). The only difference in anaesthetic technique between the groups was the number of ventilations performed after the suxamethonium, but prior to the shock; anaesthetist 1 ventilated more than the others ($P<0.01$, Table II). The change in oxygen saturation correlated with the number of ventilations given prior to ECT ($r=0.65$, $P<0.001$). All anaesthetists commenced oxygenation within 60 seconds after the seizure had finished, and the oxygen saturation invariably returned to baseline levels or higher.

The seizure duration was significantly less in group 2 compared with group 1 ($P<0.05$, Table II). The duration of seizure did not correlate significantly with the change in oxygen saturation ($r=0.21$).

TABLE II

Baseline and change in oxygen saturation, number of ventilations given and seizure duration in the four groups of patients

	Anaesthetist			
	1	2	3	4
Baseline O_2 saturation: %				
Mean	94.3	92.8	92.2	95.1
SD	2.5	3.7	2.7	2.3
Range	87–98	84–96	87–95	92–98
Change in O_2 saturation: %				
Mean	+4.0*	−7.0	−5.2	−1.1
SD	2.0	5.8	4.0	5.2
Range	0–+6	−13–+1	−11–+2	−10–+4
No. of ventilations				
Mean	18.2**	4.8	7.2	8.8
SD	4.9	1.5	4.5	1.6
Range	11–25	4–9	3–15	7–12
Seizure duration: s				
Mean	34.9	15.6†	31.1	24.7
SD	17.4	4.9	19.5	14.4
Range	15–65	6–23	8–80	15–60

*Group 1 significantly less hypoxic than groups 2 ($P<0.01$), 3 ($P<0.01$) and 4 ($P<0.05$). **Group 1 significantly more ventilations given than groups 2, 3 and 4 ($P<0.01$). †Group 2 significantly shorter seizure duration than group 1 ($P<0.05$).

Discussion

Death during ECT has been ascribed to cardiac problems involving both vagal and ectopic mechanisms (Bankhead *et al*, 1950). The ECT seizure causes widespread physiological changes due to autonomic stimulation. Bradycardia followed by tachycardia usually occurs, with hypotension followed by hypertension, therefore cardiac output falls initially, exacerbating tissue hypoxia, before rising. Low oxygen saturation occurring as heart rate and blood pressure increase predisposes to myocardial ischaemia and arrhythmias. Atropine was recommended to reduce vagal problems, however, its routine use has been challenged as it does not contribute to cardiac stability during ECT (Wyant & Macdonald, 1980).

As early as 1950, Bankhead and colleagues suggested that cardiac ectopic phenomena occur during the 'deepest cyanosis following the convulsion', but the use of oxygen during ECT did not then become routine. In a description of ECT in 1968 the shock was delivered 50 seconds after the relaxant and, following the clonic phase, three hand ventilations were delivered using, 'room air . . . and never oxygen' (Pitts *et al*, 1968). As recently as 1979 it has been claimed that oxygenation during ECT is unnecessary (Joshi, 1979), although apnoea after the shock can last for a few minutes, and will cause significant hypoxia if untreated. The present study was designed to monitor oxygenation in the clinical situation during routine anaesthesia and ECT. Significant hypoxia was demonstrated and desaturation correlated with the number of ventilations performed prior to the shock, a simple variable in anaesthetic practice.

The present study supports the concept that hyperventilation before ECT may prolong the seizure, as the patients who received fewest ventilations (group 2) had the shortest seizure duration, perhaps reflecting hypercapnia. Hyperventilation with oxygen therefore prevents hypoxia, and may also enhance therapy. As hypoxia was not related to the duration of the seizure, it may be that carbon dioxide is a more important determinant of seizure activity.

The occurrence of hypoxia is demonstrated in the present study, but not shown to be detrimental. In normal circumstances transient hypoxia should be of little consequence, but if accompanied by other problems, for example cardiovascular changes or vomiting, may be more serious. A survey of anaesthetic practice in 1980 showed that most patients are now kept well oxygenated, but a few clinics still did not routinely administer oxygen (Pippard & Ellam, 1981). As the same study showed that over 50% of the anaesthesia for ECT is performed by anaesthetists in training, teaching should emphasise the need for adequate oxygenation.

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References

BANKHEAD, A. J., TORRENS, J. K. & HARRIS, T. H. (1950) The anticipation and prevention of cardiac complications in electroconvulsive therapy. *American Journal of Psychiatry*, **106**, 911–917.

FINK, M. & JOHNSON, L. (1982) Monitoring the duration of electroconvulsive therapy seizures. *Archives of General Psychiatry*, **39**, 1189–1191.

JOSHI, V. G. (1979) Modifying ECT with a safe, simple and economical technique. *British Journal of Psychiatry*, **134**, 128.

PIPPARD, J. & ELLAM, L. (1981) Electroconvulsive treatment in Great Britain. *British Journal of Psychiatry*, **139**, 563–568.

PITTS, F. N., WOODRUFF, R. A., CRAIG, A. G., RICH, C. L. & ST LOUIS, A. B. (1968) The drug modification of ECT II. Succinylcholine dosage. *Archives of General Psychiatry*, **19**, 595–599.

WYANT, G. M. & MACDONALD, W. B. (1980) The role of atropine in electroconvulsive therapy. *Anaesthesia and Intensive Care*, **8**, 445–450.

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Anorexia Nervosa and Gastric Rugal Hyperplasia

A case of anorexia nervosa associated with gastric rugal hyperplasia in a 36-year-old male is reported and some speculations are made about the relationship between these two conditions.

Although anorexia nervosa in the male is an uncommon condition, recent studies have shown that males account for 5–10% of all cases (Hogan *et al* 1974; Anderson & Mickalide, 1983). Anorexia nervosa has been associated with a range of medical conditions, including hypothalamic tumour (Heron & Johnson, 1976), pancreatitis (Schoettle, 1979), Gaucher's Disease (Erman & Murray, 1980), Superior-mesenteric-artery syndrome (Froese *et al*, 1978) and pneumoperitoneum (Al-Mufty & Bevan, 1977). Several authors have pointed out that in cases where anorexia-like symptoms co-exist with a physical condition, it is not always easy to decide whether the weight loss is a primary or secondary symptom (Schoettle, 1979). Others have indicated the importance of excluding organic conditions in anorexia nervosa (Heron & Johnson, 1976). The latter authors also comment that even when anorexia nervosa-like symptoms are of organic origin there may be some response to psychological treatment alone, thus highlighting the importance of correct diagnosis before treatment.

Case history

A 29-year-old male wholesale butcher was admitted in an emaciated state measuring 1.765 m but weighing only 55.10 kg. Although overweight as a child, he had weighed 73.00 kg for many years. He gave a 2-year history of gradual weight loss and had lost 25% of his original body weight by the time he was first seen. A previously fit man, during the month prior to admission he had complained of satiety following small meals together with feeling light-headed. He had a 2-week history of being short of energy and, just prior to admission, had collapsed at work whilst carrying a meat carcass on his back. The patient's wife reported that he had always been a loving husband and father who had been quite involved in bringing up their three children. He had often become anxious about his health when developing minor physical illnesses but rapidly returned to his cheerful self on recovery. She had first noticed a problem 2 years previously following the birth of their last child. The patient had again become anxious about his health and, being concerned that his hair might be falling out, had arranged to have a hair transplant. Shortly after that he became concerned about his weight and refused food that she had cooked insisting on providing only high protein meals for himself. He also began to count the calorie content of his meals with the aid of slimming magazines which he hid from the family. By the time of referral he had excluded nearly all carbohydrates from his diet and had begun to abuse laxatives. Although not previously an athletic man, he had recently taken up weight-lifting to the exclusion of all other leisuretime activities. Over the same period he had lost all sexual interest in his wife in spite of their having had a previously satisfactory sexual relationship.

Examination showed marked weight loss, muscle wasting, dry skin and hair and cold cyanosed extremities. His pulse was 60/minute, blood pressure 115/80 mmHg and oral temperature 36.5 °C. The abdomen was scaphoid with no abnormal palpable masses. Even when shown his scaphoid abdomen on video tape he insisted that it protruded. He could not accept that his admission weight was below that expected for a man of his height and build but agreed that he should gain weight though felt that this could only be achieved through muscle development by weight training with reduced food intake.

Investigations revealed a lowered total protein of 53 g/litre with serum albumin of 37 g/litre, raised blood urea of 100 mmol/litre, and raised serum free cortisol of 745 mmol/litre. A barium meal, undertaken because of his complaints of early satiety, demonstrated prominent gastric folds in the body and fundus of the stomach. Although the radiology suggested malignancy, endoscopy and biopsy showed hypertrophic gastropathy with gastric foveolar hyperplasia. There was no evidence of increased gastric secretion even following pentogastric stimulation.

Pending investigation the patient was placed on a regime of bed rest and gradual refeeding which initially resulted in some weight gain. He continued to request laxatives and slimming magazines and, after his wife had refused to co-operate in this, purchased large quantities of a particular brand of mints from the mobile hospital shop which were eventually shown to carry a warning about their laxative qualities. Following this episode his weight fell to 54.00 kg. The patient was treated as a case of Ménètrièr's disease and was prescribed Ranitidine. This resulted in weight gain and eventual disappearance of his enlarged gastric rugae. He was discharged home having regained his admission weight of 55.10 kg and when followed up 6 weeks later had gained a further 5.00 kg. Three months later he was seen again weighing 49.60 kg, having failed to return to work, instead spending most of his time running or weight training. He admitted to excessive exercise and laxative abuse expressing a wish to look like 'Mr Universe'. He was readmitted when a repeat endoscopy showed no evidence of recurrence of his gastric condition. He responded slowly to a refeeding programme and was eventually discharged to day care. He continued to be preoccupied by his weight and physique. He lost his job, was divorced by his wife, and lost most of his friends since they were unable to tolerate his somatic preoccupations. His weight-lifting continued and resulted in the development of tiny muscles contrasting with the emaciation of the rest of his body. More recently he has benefitted from out-patient group psychotherapy and, at the time of writing, has gained part-time employment as a butcher. His weight fluctuates at around 65.10 kg.

Discussion

This patient's intense fear of becoming fat which did not abate as weight loss progressed, persistent disturbance of body image, weight loss of over 25% of his original body weight, and refusal to maintain his body weight over a minimal norm for his age and height, would fulfil diagnostic criteria for anorexia nervosa as defined by DSM-III (American Psychiatric Association, 1980), apart from the requirement for absence of physical illness which would account for the weight loss. With reference to Feighner's more stringent criteria for the diagnosis of this condition (Feighner *et al*, 1972), the requirement for weight loss to be at least 25% of original body weight is clearly met as is that for a distorted implacable attitude towards the eating of food, this being a persistent feature. There was no evidence of any other major psychiatric disorder, although the patient had clearly suffered from anxiety prior to the onset of the eating disorder. With regard to the presence of anorexia, this patient would admit that he was hungry if asked, but stated it was inappropriate for him to eat because this would mean becoming fat. The Feighner criteria includes anorexia as a diagnostic feature but it has recently been suggested that this term should be modified to that of self starvation (Vanderyckan & Meerman, 1984). The same authors point out that an age of onset of under 25 years is illogical, and others have reported cases in the male who meet all other criteria but this one (Andersen & Mickalide, 1983). With these reservations, therefore, the patient would also fit Feighner's criteria apart from the necessity for the absence of any medical disorder which might explain the weight loss.

Since enlargement of the gastric folds associated with low blood protein levels was first described by Ménètrièr in 1888 there have been several reports in the literature describing benign enlargement of the gastric rugae, together with cystic dialation of the glandular mucosa (Butz, 1960). These features were all present in this case, although Searcy & Malagelada (1984) have recently suggested that such patients should be considered as two groups: those with enlarged gastric folds, hypoproteinaemia and oedema constituting Ménètrièr's disease and a separate group with enlarged gastric folds but lacking the other features. They suggest that this idiopathic hypertrophic gastropathy group has a different aetiology, prognosis and response to treatment. This patient had a total protein level within the range suggested by Searcy & Malagelada for patients with Ménètrièr's disease but there was no oedema, his serum albumin being within the normal range. It would, therefore, be more appropriate to place him into Searcy's idiopathic hypertrophic gastropathy group. Although spontaneous remission has been reported (Frank & Kern, 1967), benign gastric rugal hyperplasia is usually a chronic condition which has previously been treated by gastrectomy in those cases complicated by significant protein loss. Recently Vendelboe (1981) has advocated the use of beta intestinal H_2 antagonists since these reduce protein loss and promote regression of the hypertrophic rugae thus avoiding the necessity for surgery. Such an effect was demonstrated by this patient who, when gastroscoped following 4 months treatment with Ranitidine, exhibited no further evidence of gastric pathology.

It is interesting to speculate on the relationship of the enlarged gastric folds to anorexia nervosa. Benign gastric rugal hyperplasia is considerably more common in men (Fieber & Rickert, 1981), and occasionally presents in the 20s and early 30s as does anorexia nervosa in the male (Butz, 1960). In this patient, discomfort on eating, with early satiety and marked weight loss were prominent features which occur commonly in gastric rugal hyperplasia (Searcy & Malagelada, 1984). The patient clearly meets diagnostic criteria for both anorexia nervosa and benign gastric rugal hyperplasia, but the former condition persisted after endoscopic examination confirmed successful treatment of the latter. The patient continued to complain of similar symptoms, in particular early satiety, long after all signs of enlarged gastric rugae had disappeared. Although initially gaining weight in hospital and for a period following discharge he eventually deteriorated and was readmitted at a lower weight than that at which he originally presented.

The causes of benign gastric rugal hyperplasia are unknown but there is no evidence to suggest that anorexia nervosa might be one of them. Although isolated reports of gastro-intestinal complications of anorexia nervosa have been reported (Froese *et al*, 1978), an association with gastric rugal hyperplasia has not. If the relationship between these two conditions were causal one would expect to see them together more often. There is, however, some suggestion that the presentation by this patient of these two conditions together is not entirely coincidental. He had long been anxious about coping with his expanding family exemplified by his concern about hair loss following the birth of his last child. To him losing his hair meant that he was getting too old to care for the family properly and he felt that a hair transplant would enable him to cope again. It had always been important to him to be fit and well so that when he first felt epigastric discomfort he became anxious that there might be something seriously physically wrong with him and he had not wanted to talk to anybody about it. At this point he reached a crisis when, unable to face the prospect of supporting his family whilst sick, he sought a way of becoming fit again without first seeking a diagnosis of his condition. He decided to diet because he thought that this would make him look and thus feel more fit. As he lost weight his anxiety was reduced as the weight loss due to physical illness became masked by that induced intentionally. Anorexia nervosa arising during adolescence often follows a similar crisis in coping with adult responsibilities with weight loss alleviating the associated anxiety and thus becoming self-perpetrating (Crisp, 1980). Vanderyckan & Meerman (1984) suggest a multifactorial aetiology of eating disorders and point out factors which can precipitate anorexia nervosa in predisposed individuals in both the pre-pubertal and post-adolescent age groups. Predisposing factors in this patient would include his failure to successfully complete the tasks of adolescence, leaving him with doubts about his ability to be a husband and father, and his longstanding somatic preoccupations. The physical symptoms of Ménètrièr's disease leading to a fear of serious illness could be regarded as the main precipitating factors.

This sort of relationship between two conditions is one of precipitation of the psychiatric illness in a predisposed individual at the onset of the physical one rather than of a causal link. This case, therefore, demonstrates the importance of considering a co-existent physical condition whenever a diagnosis of anorexia nervosa is made after adolescence, especially in the male with no previous symptoms of an eating disorder.

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References

AL-MUFTY, N. S. & BEVAN, D. H. (1977) A case of subcutaneous emphysema, pneumomediastinum and pneumoperitoneum associated with functional anorexia. *British Journal of Clinical Practice*, **31**, 160–161.

AMERICAN PSYCHIATRIC ASSOCIATION, COMMITTEE ON NOMENCLATURE AND STATISTICS (1980) *Diagnostic and Statistical Manual of Mental Disorders*, III. Washington, DC: American Psychiatric Association.

ANDERSEN, A. E. & MICKALIDE, A. D. (1983) Anorexia nervosa in the male: an underdiagnosed disorder. *Psychosomatics*, **24**, 1066–1075.

BUTZ, W. C. (1960) Giant Hypertrophic Gastritis: a report of fourteen cases. *Gastroenterology*, **39**, 183–190.

CRISP, A. H. (1980) *Anorexia Nervosa: Let Me Be*. London: Academic Press.

ERMAN, M. K. & MURRAY, G. B. (1980) A case report of anorexia nervosa and Gaucher's Disease. *American Journal of Psychiatry*, **137**, 858–859.

FEIGHNER, J. P., ROBINS, E., GUZE, S. B., WOODRUFF, R. A., WINOKER, G. E. & MUNOZ, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, **26**, 57–63.

FIEBER, S. S. & RICKERT, R. R. (1981) Hyperplastic gastropathy. *American Journal of Gastroenterology*, **76**, 321–329.

FRANK, B. W. & KERN, F. J. (1967) Ménètrièr's disease: spontaneous metamorphosis of giant cell hypertrophy of the gastric mucosa to atrophic gastritis. *Gastroenterology*, **53**, 953–960.

FROESE, A. P., SZMUILOWICZ, J. & BAILEY, J. D. (1978) The Superior-mesenteric-artery syndrome. Cause or complication of anorexia nervosa. *Canadian Psychiatric Association Journal*, **23**, 325–327.

HERON, G. B. & JOHNSON, D. A. (1976) Hypothalamic tumor presenting as anorexia nervosa. *American Journal of Psychiatry*, **133**, 580–582.

HOGAN, W. M., HUERTE, E. & LUCAS, A. R. (1974) Diagnosing anorexia nervosa in males. *Psychosomatics*, **15**, 122–126.

SEARCY, R. M. & MALAGELADA, J. R. (1984) Ménètrièr's disease and idiopathic gastropathy. *Annals of Internal Medicine*, **100**, 565–570.

SCHOETTLE, V. C. (1979) Pancreatitis—A complication, a concomitant or a cause of an anorexia nervosa like syndrome. *Journal of the American Academy of Child Psychiatry*, **18**, 384–390.

VANDERYCKAN, W. & MEERMAN, R. (1984) *Anorexia Nervosa—a clinician's guide to treatment*. Berlin: Walter de Gruyter.

VENDELBOE, M. & JESPERSEN, J. (1981) Hypertrophic protein-losing gastritis (Ménètrièr's disease) treated with cimetidine. *Acta Medica Scandinavica*, **209**, 125–127.

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A Case of Mosaicism with Fragile-X and XXY Components

The case of a 63-year-old severely mentally handicapped man is reported with chromosomal mosaicism. His karyotype was established as mosaic 46XY/47XXY with the fragile site present in a proportion of cells of both cell-lines. He showed phenotypic features which could be related both to the fragile-X and Klinefelter's syndromes.

A 63-year-old man with severe mental handicap was discovered to have an abnormality both in number and morphology of the X chromosomes. His chromosomes were examined when his sister's granddaughter's child died and the diagnosis of Reye's syndrome and spastic cerebral palsy was made at the Hospital for Sick Children, Great Ormond Street. He was found to be a chromosomal mosaic. Two per cent of cells showed the XXY karyotype. A fragile-X chromosome was found in 34% of the cells examined, including XXY cells. In the affected XXY cells, only one of the X chromosomes showed the fragile site.

Case history

He is the youngest of four sibs. His mother's first two pregnancies resulted in the birth of healthy normal daughters. Her third pregnancy ended in miscarriage. At the end of her fourth pregnancy she gave birth to a normal daughter who, however, died at 6 months. Her fifth pregnancy led to the birth of a blind male child with a hare lip who lived only 3 days. Her sixth, seventh and ninth pregnancies ended in miscarriage. In her eighth pregnancy she gave birth to a daughter who was feeble-minded and epileptic. Her tenth and last pregnancy led to the birth of the propositus.

He was admitted to a hospital for the mentally handicapped at the age of 18 years under Section 15 of the Mental Deficiency Act of 1913 having been found to be neglecting himself. His early milestones are reported to have been delayed. He attended elementary school for 4½ years but failed to progress. When he was 9 years of age he was thought to be mentally defective and was sent for a trial period at a special school. He appeared not to benefit from this and was deemed to be an idiot and attended occupation and craft centres. He is reported to have suffered a road traffic accident as a child, but no details of this are available. Apart from a prostatectomy, his medical history is uneventful and he is not currently on any regular medication.

He has a pleasant personality and is friendly but is shy in a group or with strangers. His speech is limited; mostly it is monosyllabic. Occasionally he utters a full sentence. His voice is of normal male pitch. He can dress and undress himself, feed himself and take himself to the toilet. He helps the ward staff with small domestic tasks and can even cook simple items. He enjoys taking the responsibility of looking after other residents in the ward. Occasionally he is aggressive when disturbed or annoyed by others.

On physical examination he is obviously mentally retarded, 170 cm in height, moderately obese and weighs 70 kilos. He is of eunuchoid habitus with wide hips and relatively narrow shoulders. His pubis-sole measurement is 83 cm and pubis-vertex measurement is 87 cm. He has a pronounced dorsal kyphoscoliosis. He has a long face with a high forehead and relatively large ears. He is edentulous with a slightly protruding lower jaw, high arched palate and a slight strabismus of the left eye. There is moderate gynaecomastia but distribution of body hair is of normal male type. The penis is of normal size with both testes descended and the right testis is slightly smaller in size. The hands and feet are of normal size with moderate pes planus. He walks with short shuffling gait but there is no other obvious abnormality in the central nervous system. X-ray of skull and elbows are normal and X-ray of spine showed slight scoliosis. His serum testosterone level is within normal limits, being 15 mmol/litre (normal range of male 10 mmol/litre–38 mmol/litre).

Discussion

Considering the relatively high frequency of Klinefelter's syndrome and the fragile X syndrome, patients with occasional coincidence of both syndromes is to be expected. To our knowledge there have been four case reports in the literature with the concurrence of both conditions in the same patient. These could show variability with respect to phenotypic manifestation which is not altogether surprising as the relative strength of expression of the two syndromes is likely to vary in individuals in which they are concurrent.

Wilmot *et al* (1980) observed a case with less severe mental retardation and less characteristic facial

TABLE I

Comparison of clinical features in different case reports with both XXY and fragile-X

	Case of Wilmot et al (1980)	*Case of Froster–Iskenius et al* (1982)	*Case of* Fryns *et al* (1983)	*Case of* Fryns *et al* (1984)	Present case
Age		16 years	42 years	44 years	63 years
Mental retardation					
moderate	+	–	+	+	–
severe	–	+	–	–	+
Height		176 cm	175 cm	165 cm	170 cm
Long face with relatively big ears	–	+	–	+	+
Prognathism	–	–	–	+	+
Axillary and pubic hair		Present	Present	Scanty	Present
Gynecomastia		+	–	–	+
Macro-orchidism	–	–	–	–	–
Small soft testes	+	+	+	+	Smaller right testis
Serum testosterone		Low	Normal		Normal
Karyotype and % cells with fragile-X	47 XXY 2% Fragile-X	47 XXY 27.9% Fragile-X (3.6% in inactivated X)	XY/XXY (50% XXY) 2% of 47 XXY Showed Fragile-X	47 XXY 51% Fragile-X (20% in both active and non-active X)	XY/XXY (2% XXY) 34% Fragile-X in both XY and XXY

expression than are usually found in patients with fragile X syndrome. His patients had small testes and these findings were thought to be due to the effect of the second X chromosome. On the other hand Froster-Iskenius *et al* (1982) reported a case with mental retardation which was more severe than usually observed in Klinefelter's syndrome while testicular hypotrophy, gynaecomastia and hormonal levels were more characteristic of the 47 XXY condition. In the same case facial appearance with large ears was probably the expression of the fragile X chromosome (see Table I). In the case reported by Fryns *et al* (1983) the face was less typical of the fragile X syndrome, being rounded, and without obvious prognathism. The genital development was compatible with mosaic Klinefelter's syndrome: both testes were small and soft, but testosterone levels and penile size were normal. In their study only 2% of the cells with 47, XXY showed the fragility at Xq27–28 but no fragile site could be found in 46, XY cells. In another report Fryns *et al* (1984) reported a moderately mentally retarded male with a long face and relative prognathism, gynecoid pelvis and small soft testes. His secondary sexual characteristics were poorly developed with scanty axillary and pubic hair. In some cells, fragile sites could be found on both X chromosomes.

In the present case, the facial appearance and severe mental retardation is consistent with the fragile X syndrome. The presence of XXY cells could well be responsible for the gynaecomastia and the reduced size of the right testis. Unfortunately the occurrence of fragile X chromosome in other members of the family could not be determined as permission for further investigations was refused. The recurrent foetal loss suffered by the mother of our case may have been associated with chromosomal imbalance. The possibility of maternal syphilis was considered as a potential cause of recurrent foetal loss—obviously the mother was not available for investigation, but serological tests for syphilis were carried out in the case of the propositus and were found to be negative. In the small number of cases described,

be negative. In the small number of cases described,

there appears to be a tendency of the fragile X to show characteristic facies and a degree of mental retardation, while the remaining features tend to be those of Klinefelter phenotype. It may be of particular significance that the testicular pathology of Klinefelter's syndrome prevents the expression of the fragile X macro-orchidism.

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References

Froster-Iskenius, U. Schwinger, E., Weigert, M. & Fonatsch, C. (1982) Replication pattern in XXY cells with fra(X). *Human Genetics*, 60, 278–280.

Fryns, J. P., Kleczkowska, A., Kubien, E., Petit, P., Haspeslagh, M., Lindemans, I. & Van Den Berghe, H. (1983) XY/XXY mosaicism and fragile X syndrome. *Annales Genetiques*, 26, 251–253.

Fryns, J. P., Kleczkowska, A., Wolfs, I. & Van Den Berghe, H. (1984) Klinefelter syndrome and two fragile X chromosomes. *Clinical Genetics*, 26, 445–447.

Wilmot, P. L., Shapiro, L. & Duncan, P. A. (1980) The Xq27 fragile site and 47,XXY. *American Journal of Human Genetics*. (Suppl.) 94A: 285.

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Epilepsy and Psychosis – Ask The Family

Psychotic episodes and epilepsy can occur without mental retardation in tuberous sclerosis. The diagnosis may depend on elucidating the family history since physical signs may not be obvious. Establishing the diagnosis makes genetic counselling possible.

Tuberous sclerosis is a cause both of epilepsy (Rakoff, 1981) and of psychotic episodes resembling schizophrenia (Herkert *et al*, 1972). It is inherited by autosomal dominant transmission although a spontaneous mutation rate as high as 80% has been suggested (Bundey & Evans, 1969). About two-thirds of patients are mentally retarded (Nevin & Pearce, 1968). Even within a family, the clinical features of tuberous sclerosis may show great variability and a family history of epilepsy or of psychiatric illness may be lacking. Members of the family may not be aware that their differing abnormalities are due to the same underlying inherited disorder.

Case report

A 19-year-old epileptic man was referred by the police surgeon. He had been arrested as he had attempted to draw public attention to his stated mission from God "to send the earth back into the sun" by damaging a car. For the previous 2 years he had a history of increasing social isolation and apathy, staying at home and making no attempts to gain friends or employment. For 3 months before admission he had become increasingly aggressive and had begun to express bizarre ideas, particularly in the postictal state.

On admission the patient was withdrawn and reluctant to communicate. He answered questions briefly, though frequently not to the point, and offered no spontaneous conversation. His affect was flat and he was preoccupied with the megalomanic delusional system that he was "Father of all the people of the world", having a mission "to turn the world back into the sun and thus make people believe in Jesus Christ". He also expressed a delusion that the world would end in 1 week. He made no further attempt to act on his delusions in the ward setting. He suffered from thought broadcasting and passivity feelings and had auditory hallucinations of God talking to him. He showed marked concrete thinking in interpreting proverbs. His concentration was poor, mainly because of his preoccupation with the delusions and hallucinations. Though perplexed, his memory and orientation were normal.

Physical examination revealed three small depigmented areas on his back, which were dismissed at the time as being of no consequence. Routine biochemical and haematological investigations were within normal limits. A series of EEGs all showed continuous irregular delta activity with occasional spike discharges localised to the left anterior temporal region.

The patient's father, with whom he had lived following his parents' divorce some years previously, reported that his son had been epileptic from the age of 10 with generalised tonic-clonic seizures. Some 4 years later, his previously good seizure control deteriorated and post-ictal episodes of wandering and confusion began. He had been treated with carbamazepine. In the years leading up to his admission, the frequency of his generalised seizures increased to as many as ten per day and he had once been admitted to hospital in status epilepticus.

A diagnosis of an organic psychosis was made and, in addition to carbamazepine, treatment was started with haloperidol and later flupenthixol decanoate with a good response. Psychometric testing performed when the acute psychotic symptoms had abated showed a Wechsler Adult Intelligence Scale IQ of 99 (verbal scale 97, performance scale 102).

His father had no knowledge of any family history of psychiatric illness, epilepsy or serious physical illness. However, when the patient's mother was interviewed 2 weeks after his admission, subungual fibromata were noted on her fingernails. These were also found on her toenails and were apparently also possessed by her own mother. In view of this, tuberous sclerosis was suspected. Re-examination of the patient showed that the previously discounted areas of depigmented skin were recognisable as ash-leaf patches. Computed tomography of brain confirmed the diagnosis, demonstrating several calcific opacities, two lying in a subependymal position lateral to the bodies of the lateral ventricles. Bilateral temporal lobe atrophy was also noted.

Having established the diagnosis it became possible to initiate a suitable rehabilitation programme for the patient, to offer genetic counselling to his mother and her family and to give a more accurate prognosis.

Discussion

Tuberous sclerosis may present in a variety of ways and with a plethora of clinical features other than the classical triad of adenoma sebaceum, epilepsy and mental retardation (Person & Perry, 1979). Bundey & Evans (1969) investigated patients in whom adenoma sebaceum appeared after the age of 20. Of patients with an affected parent only 20% were epileptic. This finding is the more striking because of the preponderance of severe childhood cases in their study.

Psychosis presenting in affected individuals with no history of epilepsy has also been described (Herkert *et al*, 1972). Schizophrenic-like psychosis and epilepsy, particularly temporal lobe epilepsy, are well known to co-exist (Beard & Slater, 1962). Psychotic phenomena akin to those found in schizophrenia may occur during a temporal lobe seizure and Perez *et al* (1985), from their study of 24 patients with epilepsy and psychosis, concur with the hypothesis that discharges in the dominant temporal lobe are associated with a diagnosis of schizophrenia. The anatomical lesions of the temporal lobes in our patient are of interest in this respect.

Our case demonstrates that tuberous sclerosis may present as a psychotic illness and that physical signs may be overlooked or dismissed as within normal limits. The diagnosis should be considered particularly in patients presenting with psychosis combined with epilepsy. It illustrates the importance of obtaining a detailed family history to aid the differential diagnosis of possible inherited organic disorders. Family members may not be aware of, or may not reveal, significant information. It is necessary to interview more than one relative, particularly if members of the patient's immediate family are divorced or separated.

References

Beard, A. W. & Slater, E. (1962) The schizophrenic-like psychosis of epilepsy. *Proceedings of the Royal Society of Medicine*, **55**, 311–316.

Bundey, S. & Evans, K. (1969) Tuberous sclerosis: A genetic study. *Journal of Neurology, Neurosurgery and Psychiatry*, **32**, 591–603.

Herkert, E. H., Wald, A. & Romero, O. (1972) Tuberous sclerosis and schizophrenia. *Diseases of the Nervous System*, **33**, 439–445.

Perez, M. M., Trimble, M. R., Muray, N. M. F. & Reider, I. (1985) Epileptic psychosis: an evaluation of PSE profiles. *British Journal of Psychiatry*, **146**, 155–163.

Person, J. R. & Perry, H. O. (1979) Recent advances in the phakomatoses. *International Journal of Dermatology*, **17**, 1–13.

Rakoff, J. (1981) Tuberous sclerosis in the aetiology of the so-called idiopathic epilepsies and psychoses. *South African Medical Journal*, **60**, 574–578.

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An Epidemic of Suicide Attempts by Burning in a Psychiatric Hospital

Three patients attempted suicide by burning in one ward of a psychiatric hospital in a period of less than 1 month. No such attempts had occurred there in the previous 9 years. The three patients were schizophrenics with depressive features who occupied the same room in the ward. It seems reasonable to suggest that at least the choice of method and the timing of these suicide attempts were influenced by imitation. Hospital staff should be especially aware of the influence of imitation in suicide in order to avert such undesired epidemics.

Different views have been expressed on the role of imitation in suicide. William Farr (1841) wrote: "No fact is better established in science than that suicide is often committed from imitation". Suicide rates increase briefly following publicity given to the suicide of famous people (Hewitt & Milner, 1974; Phillips, 1974). A statistical association has been shown between reports of suicide inquests in a local paper and the subsequent suicide of men under 45 years (Barraclough *et al*, 1977). These authors are convinced that suicide is contagious.

Durkheim (1897) adopted a different point of view and wrote that suicide "exposes a state which is the true generating cause of the act and which probably would have produced its effect even had imitation not intervened".

An epidemic of suicide by burning was reported in England and Wales in 1978–1979 (Ashton & Donnan, 1979). Between October 1978 and March 1979, there were 42 deaths caused by burning with suicidal intent, while the average in the previous 15 years was only 19 cases/year. The epidemic started following wide publicity in the press given to the suicides of two women who burned themselves in public on 2 and 4 October 1978. It seems reasonable to suggest that at least the choice of the method of suicide was influenced by the publicity surrounding those two cases.

In our closed psychiatric ward, there were no suicide attempts by burning in the last 9 years. The occurrence of three such attempts in a period of less than 1 month drew our attention to the phenomenon of imitation in a psychiatric hospital and to the possible influence of the first suicide attempt on the timing and choice of method in the other two patients.

Case reports

Case A

This single woman, aged 23, born in Israel to parents of European origin, had a schizophrenic illness starting at the age of 16 years when she was first admitted to hospital. Since then she had been admitted several times in acute psychotic states. There were partial remissions but her social and occupational functioning were markedly affected. She never took her medications regularly. Her present admission was because of an exacerbation of her schizophrenic illness expressed by disturbance of thinking, blunted affect, auditory hallucinations and negativistic behaviour. The patient had a striking appearance—tall, blonde with blue eyes and with a ready smile. She played the piano well and, in spite of the severe course of her illness, had a special status among the patients in the department. Treatment with trifluoperazine (Stelazine) 60 mg/day brought about an improvement in her condition.

Three months after her admission, she learnt that her parents were about to separate and that she would have to reside with her father, and she attempted suicide by pressing a burning towel against her body. As a result she suffered split and full thickness burns over about 9% of her body surface. In the course of the attempt, she smiled and expressed a blunted affect, claiming that she had reacted to voices she had heard. These voices had hinted that in this way her suffering would cease. Subsequently, she continued to express suicidal ideation and 2 weeks after the suicide attempt, she was moved to another room.

Case B

This woman, aged 22, who shared the new room of Case A, was born in Israel to a father of oriental origin (Lebanon) and an Israeli-born mother. The patient, married, without children, had suffered from schizophrenia that started at the age of 15 in the form of affective outbursts. During a previous depressive state, she had attempted suicide by cutting her wrists. This time she was admitted to hospital with depressed mood, formal thought disorder, delusions, auditory and visual hallucinations. Four days after her admission, she attempted suicide by tying a nylon cord around her neck. Because there was a possibility that the patient was pregnant, she started receiving ECT which brought about a slight improvement in her condition. However, she continued to lie in bed, with marked evidence of delusions and hallucinations. When it became clear that the patient was not pregnant, the treatment was changed to trifluoperazine (Stelazine) 40 mg/day.

When her condition did not improve significantly after 3 months, she became worried that she might be transferred to another institution for chronic patients. As a protest she attempted suicide by setting fire to a shirt and pressing it against her chest, thus suffering burns over about 2% of the body surface. She later reported that she got the idea and the method from conversations with patient A, who had committed a similar attempt 3 weeks earlier, and who now was in the bed next to hers.

Case C

A female patient, aged 47, born in Iraq, married and the mother of three children, shared the same room with patients A and B. She was admitted to hospital in a state that seemed, at first, as agitated depression following a suicide attempt by swallowing insecticide, in an open ward of another hospital. In our ward, she attempted suicide by trying to strangle herself with a sheet.

Her clinical condition was characterised by detachment, agitation, insomnia, thinking disorders, auditory hallucinations (that gave her orders) and suicidal thoughts. Treatment with neuroleptics, tricyclic antidepressants and ECT did not change her condition. In the course of her extended stay in hospital, the deterioration in her affect and function became prominent and, based on the symptoms, she was diagnosed as schizophrenic. It is noteworthy that the patient's daughter suffered from depression and had also attempted suicide. A number of second degree relatives suffer from schizophrenia or depression.

On the same morning that patient B, who shared the room with her, tried to burn herself, patient C had come to her rescue and summoned help. Half an hour later she shut herself in the restroom, wrapped toilet paper around her body and set fire to it. This caused burns over 3% of the body surface. This attempt was not the last one. After her discharge from hospital she jumped from the third floor of her apartment and was severely hurt.

Discussion

Suicide by burning is unusual in Western countries. In England and Wales it accounted for less than 1% of suicides in 1963–1977. Burning as a method of suicide or attempted suicide is rare also in the United States (Weissman, 1974). In contrast, this method is considered more common in Israel. The last survey that was conducted on this subject in Israel (Modan *et al*, 1970) found that 27.7% of female suicides and 2.9% of male suicides were by burning. This method was found especially prominent among women of Asian and African origin among whom 77.1% of completed suicides were by burning.

The population of completed suicides usually differs in its characteristics from that of attempted suicides (Weissman, 1974), but when suicide by burning is considered, these differences are not clear. In a series from an Australian burns unit (Persley & Pegg, 1981), 47% of suicide attempts by burning died in the hospital. Thus, in about half of the cases the attempted suicide becomes completed suicide. For this reason, it seems to us correct to compare our three cases of attempted suicide with other reports of epidemics of completed suicides.

Although suicide or attempted suicide by burning is supposed to be common in Israel, we feel that data from the 1960s (Modan *et al*, 1970) are not adequate in the 1980s and that methods of suicide in this country have changed and are now closer to those of other Western countries.

Three suicide attempts by burning in a closed psychiatric department is an exception that has not been recorded before. The Geha Psychiatric Hospital is a main district hospital that serves half a million population. Sixty per cent of the inpatients are diagnosed as schizophrenics and most of them return to their community. A small number of patients are transferred to other hospitals on a chronic basis.

Our three patients were considered as high-risk patients for suicide. All of them suffered from schizophrenia accompanied by depressive features. Patients B and C had already attempted suicide more than once, using different techniques. After patient B's suicide attempt, she admitted that she had been influenced by patient A, her new room mate. Also in patient C, who attempted suicide 30 min after her room mate, it seems obvious that imitation played an important role. The tendency to suicide attempts existed in all three patients, but it seems reasonable to suggest that the choice of method and timing were influenced by imitation.

It is worthwhile mentioning that a fourth schizophrenic patient with paranoid delusions toward her neighbours, who shared a room with patient A at the time of the burning, tried to set fire to the neighbours' apartment after discharge from the hospital and as a consequence was severely scalded in her lower extremities. This patient had not until then, made any attempts to harm her neighbours and so we assume that there was some influence related to the suicide attempt of patient A.

In three other reported epidemics of suicide in psychiatric hospitals (Kobler & Stotland, 1964; Kaline, 1968; Anonymous, 1977), the authors blame the epidemics on weakness of professional leadership, faulty supervision and staff demoralisation. Although the contribution of these factors cannot be denied completely, it seems that no attention has been paid to the role of imitation which existed in relation to timing and choice of method. The Crest Hospital epidemic reported by Kobler & Stotland in 1964, started with a patient who killed himself by cutting his wrists, which happened 1 week after another patient was found in his room, bleeding profusely from deep cuts in his throat and wrists. This patient had been rescued and therefore was not included in the suicide epidemic. However, his influence on starting the epidemic seems very significant. In another epidemic (1977), the first two out of three patients who committed suicide were found hanging in their hospital room.

In the current epidemic the staff can be criticised and blamed for not being sufficiently alert to the phenomenon of imitation. It was a mistake to transfer patient A, who had a special status among the patients, to a room together with two other patients known as high-risk cases for suicide. Had these two patients not been exposed to the influence of patient A, their present suicide attempts might have been averted.

It may be concluded that more attention should be paid to the role of imitation in suicide, especially in psychiatric hospitals where patients are more exposed to the influence of other patients. Every act of suicide or attempted suicide necessitates a response on the part of the staff so that this act does not become a subject for imitation. One possibility is to gather the patients for a group session after a suicidal case in order to neutralise the identification with the act by suggestible patients. It also seems appropriate to bring up this subject in individual sessions, especially with high-risk patients. By taking these precautions, such epidemics may perhaps be prevented and thus the distress they cause to the staff and the patients alike may be spared.

References

Anonymous (1977) A suicide epidemic in a psychiatric hospital. *Diseases of the Nervous System*, **38**, 327–331.

Ashton, J. R. & Donnan, S. P. B. (1979) Suicide by burning—a current epidemic. *British Medical Journal*, **2**, 769–770.

Barraclough, B., Shepherd, D. & Jennings, C. (1977) Do newspaper reports of coroners' inquests incite people to commit suicide? *British Journal of Psychiatry*, **131**, 528–532.

Durkheim, E. (1897) *Le Suicide*. Paris. Translated (1952) as *Suicide: A Study in Sociology*, by J. A. Spaulding & C. Simpson. London: Routledge & Kegan Paul.

Farr, W. (1841) *Third Annual Report of the Registrar General Births, Deaths and Marriages in England (for 1839–1840)*. London: HMSO.

Hewitt, D. & Milner, J. (1974) Drug related deaths in the United States—first decade of an epidemic. *Health Services Reports*, **89**, 211–217.

Kaline, M. J. (1968) Suicide among patients in mental hospitals. *Psychiatry*, **31**, 32–43.

Kobler, A. L. & Stotland, E. (1964) *The End of Hope*. London: The Free Press of Glencoe, Collier-Macmillan.

Modan, B., Nissenkorn, I. & Lewkowski, S. R. (1970) Comparative epidemiologic aspects of suicide and attempted suicide in Israel. *American Journal of Epidemiology*, **91**, 393–399.

Persley, G. V. & Pegg, S. P. (1981) Burn injuries related to suicide. *Medical Journal of Australia*, **1**, 134.

Phillips, D. P. (1974) The influence of suggestion on suicide: substantive and theoretical implications of the Werther effect. *American Sociological Review*, **39**, 340–354.

Weissman, M. M. (1974) The epidemiology of suicide attempts, 1960 to 1971. *Archives of General Psychiatry*, **30**, 737–746.

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Point of View

Depression in the General Population: Comparability of Survey Results

T. K. J. CRAIG, G. W. BROWN and T. O. HARRIS

Bebbington (1986) draws attention to a possible cause for concern regarding the comparability of recent epidemiological research utilising the PSE-ID-CATEGO system of psychiatric caseness determination. In taking issue with our observation that cases in our recent community survey (Brown *et al*, 1985) were broadly similar in terms of severity to those reported in a recent series of out-patients (Sashidharan, 1985), he makes the point that such conclusions are only justifiable when it can be confidently assumed that thresholds for symptom inclusion are applied uniformly between research centres. He argues that this assumption may well be invalid and marshalls two arguments to suggest that the Bedford team may have adopted less stringent rating thresholds. Firstly, he draws attention to the very different population prevalences of disorder reported in recent surveys, contrasting one-year prevalence figures for the Bedford College survey in Islington (Brown *et al*, 1985) with the lower rates reported for other investigations in Edinburgh (Dean *et al*, 1983) and Camberwell (Bebbington *et al*, 1981). Secondly, he reminds us of the body of evidence which suggests that psychiatrists operate stricter criteria for PSE symptoms than their lay colleagues (Wing *et al*, 1977*a*; Sturt *et al*, 1981).

Before taking these arguments and their implicit conclusion too seriously, however, a number of qualifications are in order. Regarding the first argument, we would wish to add that before attempting a comparison of *overall* prevalence rates, attention ought to be directed at ensuring that like is compared with like, in terms of both the general characteristics of the populations concerned and the period over which symptoms have been collected in order to determine caseness. The composition of the Islington survey was deliberately weighted in favour of a specific subgroup of the general population (working-class women with children living at home) who were expected to manifest a particularly high prevalence of psychiatric disorder, and consequently any attempt to compare rates of disorder between this and other surveys must begin by identifying and extracting data for similar groups of women (in terms of class position and parenthood). Each centre, having utilised the PSE, has available a measure of symptoms present in the month prior to interview (i.e. that are generated by the use of the PSE in its most simple form), and we would argue that it is this data which should be used as the starting point when comparing rates between centres, as it is less open to the effects of problems of recall, or those which might result from local modifications. The Bedford College caseness rating based on symptoms measured for the peak of the disorder in the whole preceding year is clearly different from either the episode-based approach used by the Edinburgh researchers, or the 'back-weighted' prevalence estimate calculated for the Camberwell study for approximately a two-month period. Finally, as we are concerned with determining whether differences in prevalence rates between centres might be attributable to differences in thresholds for rating individual symptoms, it would seem logical to use a method of determining caseness which is guaranteed to be applied uniformly by all centres. The ID-CATEGO computer programme, being entirely free from the vagaries of potentially biased researchers, provides the ideal such technique. Dr P. Surtees and Dr P. Bebbington have kindly supplied the necessary material to do this. If all these steps are followed, and ID level 5 is taken to discriminate between cases and non-cases for working-class women with a child at home, the disparity in reported prevalence rates between centres is largely eliminated: the rate for Edinburgh is 18.2% (Surtees, P. G., personal communication); that for Islington is 23.1% and that for Camberwell is 25.0% (Bebbington, P., personal communication). The fact that the two London surveys are so similar suggests that in so far as there are other differences in rates reported by the three centres, they cannot be greatly explained by differences in the rating of constituent symptoms and are likely to reflect real population differences.

Regarding Bebbington's second point, we would concur that there is a risk that lay interviewers overrate on the PSE relative to psychiatrists. However, this is not uniformly borne out in available evidence. Firstly, the studies quoted by Bebbington refer to the proclivities of naïve raters. The study of Wing *et al* (1977*a*), for example, compared PSE ratings made by a psychologist, a social worker, and a psychiatrist who had received *no* formal PSE training, and the results are hardly generalisable to a team which has been trained in the use of the instrument. (One undertakes training precisely because naïve raters are likely to make inaccurate judgements about thresholds). The other study quoted (Sturt *et al*, 1981) seems to suggest that such biases persist even after training, but closer examination of the details of this report show that the raters involved were very different from those employed in the Islington study. For a start, although they were professional interviewers, none had had prior experience with the PSE and all received "a shortened (3-day) version of the usual training course" (Sturt *et al*, 1981). The interviewers were employed as a first-level screen to identify possible cases whose disorder was then formally ascertained by a second interview. Leaving aside the difficulties of interpretation which arise because of recovery or fluctuation of symptoms between interviews, it is our contention that the performance of these semi-trained interviewers cannot be fairly compared with that of a clinical team who received full training and ongoing corrective supervision. Perhaps Bebbington might have referred to the considerable evidence that *after adequate training*, lay interviewers are capable of accurately and reliably administering the PSE, some learning to modify their threshold criteria upwards and others to acquire a tougher perspective (e.g. Cooper *et al*, 1977; Wing *et al*, 1977*b*).

In conclusion, we believe that the issues raised by Bebbington must be considered seriously. It behoves all researchers in this field to co-operate throughout the collection of data. Perhaps, in future, joint rating meetings and mutual monitoring would serve to strengthen confidence in technique.

References

BEBBINGTON, P. (1986) Depression: distress or disease? *British Journal of Psychiatry*, **149**, 479.

——, HURRY, J., TENNANT, C., STURT, E. & WING, J. K. (1981) Epidemiology of mental disorders in Camberwell. *Psychological Medicine*, **11**, 561–579.

BROWN, G. W., CRAIG, T. K. J. & HARRIS, T. O. (1985) Depression: distress or disease? Some epidemiological considerations. *British Journal of Psychiatry*, **147**, 612–622.

COOPER, J. E., COPELAND, J. R. M., BROWN, G. W., HARRIS, T. & GOURLAY, A. J. (1977) Further studies on interviewer training and inter-rater reliability of the Present State Examination. *Psychological Medicine*, **7**, 517–523.

DEAN, C., SURTEES, P. G. & SASHIDHARAN, S. P. (1983) Comparison of research diagnostic systems in an Edinburgh community sample. *British Journal of Psychiatry*, **142**, 247–256.

SASHIDHARAN, S. P. (1985) Definitions of psychiatric syndromes – comparisons in hospital patients and general population. *British Journal of Psychiatry*, **147**, 547–552.

STURT, E., BEBBINGTON, P., HURRY, J. & TENNANT, C. (1981) The Present State Examination used by interviewers from a survey agency: report from the MRC Camberwell community survey. *Psychological Medicine*, **11**, 184–192.

WING, J. K., HENDERSON, A. S. & WINCKLE, M. (1977*a*) The rating of symptoms by a psychiatrist and a non-psychiatrist: a study of patients referred from general practice. *Psychological Medicine*, **7**, 713–715.

——, NIXON, J. M., MANN, S. A. & LEFF, J. P. (1977*b*) Reliability of the PSE (ninth edition) used in a population survey. *Psychological Medicine*, **7**, 505–516.

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Book Reconsidered

Sociobiology, the New Synthesis: Edward O. Wilson*

Sociobiology was published in 1975. Four years earlier its author had published "an enormous and definitive book" on social insects, a work which established him as the world authority on that subject. In *Sociobiology,* Wilson set out to apply evolutionary theory to social behaviour not only in insects but throughout the animal kingdom. As a biologist he naturally included man among the animals, but it was this which caused the uproar. Had he omitted his last 33 pages – the chapter on "Man: from sociobiology to sociology" – his book would probably have led a quiet life as a well-written semi-popular textbook on social animals.

Sociobiology was praised as opening a new field in social study, a biological basis for a science of sociology. It was dismissed as the popularisation of a subject outside its author's field of competence, the term sociobiology being "merely a grand word for one branch of evolutionary ethology". And it was attacked with a surprising degree of virulence, not only by creationists and left-wingers but by a host of respectable academics as well (e.g. Sahlins, 1976; Rose *et al,* 1984). To a psychiatrist concerned with the vagaries of human nature, it is the virulence which is interesting; for as Oliver Wendell Holmes said of the indignation aroused by his remark about the pharmacopoeia and the fishes, "a loud outcry at a slight touch reveals the weak spot in a profession as well as in a patient".

The most acrimonious criticism of *Sociobiology* was that it used spurious biological analogy to give pseudo-scientific respectability to sexism and the capitalist ethic of the middle classes. Wilson had argued: that there was strong evidence of genetic factors in the social behaviour of animals (the social behaviour of chimpanzees, for example, is everywhere very similar, even among widely separated groups); that the social behaviour of apes and of present-day hunter-gatherer societies had much in common – including aggressive dominance-seeking males and docile females; and that, as these features most probably had a genetic basis, it was reasonable to presume similar genetic traits in civilised mankind, even if these were now largely overlaid by culture. But, said the critics, animals are not human, and to suppose their behaviour had anything in common with human behaviour was to be guilty of naive anthropomorphism. So *Sociobiology* was derided as mere myth-making, as a just-so story, as a piece of bourgeois thought; and its author's suggestion that his new synthesis might provide "an enduring set of first principles of sociology" (page 579) was considered absurd or offensive, not only by orthodox sociologists but also by philosophers who held ethics to be outside the scope of science.

What these criticisms imply, I think, is that Wilson stirred up the never-ending conflict between science and religion (or the secular counterparts of religion). The stability of most societies depends on the general acceptance of a moral code, whose authority derives from myths enshrined in holy writ. Ideas or discoveries which might undermine the credibility of these myths will threaten social stability; and the rulers of society, who derive their power from its stability, will naturally wish to suppress such dangerous knowledge – and have done so down history with a violence exemplified by the fate of Prometheus, Galileo, the Christian heretics, and the Russian dissidents. In 1690 the physician and philosopher John Locke argued that since civilised behaviour depended on a belief in God, the protection of the State should be withdrawn from atheists; and in the early 19th century the progress of geology was retarded by the fear that if God's special intervention in the course of nature were denied, it would lead to free-thinking and so to revolution (Gillespie, 1951). Wilson's contribution to dangerous knowledge was to support the idea that human social behaviour is partly under genetic control. The idea is dangerous (to those in power) because it implies a person cannot be held entirely responsible for his behaviour. If that were so, then society would be without a moral sanction against those who broke its rules. This explains why the Church disapproved of astrology, why Stalin disapproved of genetics (as shown by his support for Lysenko), and why the predestination of Calvin was of a kind "not to be enquired into".

The dominance of nurture over nature has been upheld not only by those wielding religious power

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but also by philosophers and sociologists fiercely opposed to religion. Hume, who defined a clergyman as "a person appropriated to teach hypocricy and inculcate vice" (Brady, 1984), and who, on his deathbed, told Boswell that "when he heard a man was religious he concluded he was a rascal", nevertheless believed the mind is a *tabula rasa* at birth and that a person's character and behaviour are formed entirely from what happens to him thereafter. John Stuart Mill (1873), whose father taught him that religion was not merely a mental delusion but a moral evil opposed to the progress of the human mind, deplored "the prevailing tendency to regard all marked distinctions of human nature as innate". He held this idea to be "one of the chief hinderances to the rational treatment of great social questions", and believed there were "irresistable proofs" that "by far the greater part" of natural differences between men were produced by "differences in circumstances". Darwin on the other hand, although also a freethinker, concluded that the greater part of human attributes were inborn (Barlow, 1958); and Galton (1883), who had found that both the physical and mental attributes of identical twins were strikingly similar even when they had been reared apart, considered this clear evidence of "the vastly preponderating effects of nature over nurture". Maudsley (1919), always biologically minded, took the same view: "civilised nature doesn't mean the vital (brutish) nature is any less".

There was still another criticism of *Sociobiology*. This was directed against its reductionist view that human attributes are all understandable in biological terms and therefore, ultimately, in terms of physics and chemistry. Maudsley was a reductionist, defending himself on the general ground that matter is more wonderful than you think (Maudsley, 1913). In recent years reductionism has been made respectable by the support of eminent physicists and biologists (Jaynes, 1976; Atkins, 1981; Smith, 1985; Dawkins, 1986). But this opinion, which would have been considered blasphemous in a religious age, is still emotionally or intellectually unacceptable to many. Even Alfred Wallace, Darwin's co-discoverer of the origin of species, never believed that evolution applied to Man. Evolutionary reductionism, first attacked by Samuel Butler in his *Luck or Cunning?*, has been criticised on the general ground that evolution is not a passive process but implies at every stage the presence of individual learning and purposive exploration (Popper, 1974; Granit, 1977), and on the particular ground that human mental powers are so much greater than other creatures' as no longer to be comparable, the influence of culture now quite outweighing that of genetics (Crook, 1980).

These multifarious criticisms of *Sociobiology*, and particularly the virulence of some of them, surely indicate the importance of the subject raised in its final chapter. Wilson did not deny the large part played by culture in shaping human society: he simply pointed out that there were probably some genetic factors too, and that it would be useful to enquire further into the nature and strength of such factors since, in so far as they were present, they would exert a constraint on the range of cultural possibilities: "the genes hold culture on a lease" (Wilson, 1978). The gain to sociology from accepting a more biological basis might be that this would involve taking a long-term evolutionary view of human nature in addition to the short-term cultural view. Darwin noted that there are animal species (e.g. the emu and the cassowary) where the usual sexual patterns of courtship and care of offspring are reversed – a phenomenon that might remind one, for instance, of that German tribe noted (and deplored) by Tacitus – and an interesting parallel has been drawn between the genetic variations of behaviour in animal species and the cultural variations of behaviour in human societies. But Darwin also believed in capitalism, in the innate superiority of men's minds over women's (his opinion was similar to that of Mill) (Darwin, 1871) and of the white races over the black (Dawkins, 1986); and Maudsley (1919) held that "democracy is in no wise lovely". The fact that we now tend to think differently illustrates the rapidity with which cultural values may change, and this suggests there is a risk in basing sociological theory on what may prove to be transient aspects of human thought and behaviour. "All sensible biologists", said Bonner (1975, see also Andreski, 1972), "are apprehensive of human sociology" because it seems such a "murky mixture of the obvious and the obscure". Wilson's book reinforced what is perhaps a growing opinion that if sociology is to become a science it must be based on biology (Pugh, 1978; Symons, 1979; Berghe, 1979). My own hope is that the furore over *Sociobiology* will represent an instance of today's outrageous idea becoming the commonplace of tomorrow.

References

ANDRESKI, S. (1972) *Social Sciences as Sorcery*. Harmondsworth: Deutsch.

ATKINS, P. (1981) *The Creation*. London: Freeman.

BARLOW, N. (1958) *The Autobiography of Charles Darwin 1809–1882*. London: Collins. p. 43.

BONNER, T. J. (1975) Review of E. O. Wilson's *Sociobiology*. *Scientific American*, **244**, 129–132.

BRADY, F. (1984) *James Boswell: The Later Years 1769–1795*. London: Heinemann. p. 89.

CROOK, J. H. (1980) *The Evolution of Human Consciousness*. Oxford: Clarendon.

DARWIN, C. (1871) *The Descent of Man* (2nd ed., 1874). London: Murray. p 859.

DAWKINS, R. (1986) *The Blind Watchmaker*. Harlow: Longmans.

GALTON, F. (1883) *Inquiries into Human Faculty*. London: Everyman (2nd ed. Dent). p 217.

GILLESPIE, C. C. (1951) *Genesis and Geology*. New York: Harper and Row.

GRANIT, R. (1977) *The Purposive Brain*. London: MIT.

JAYNES, J. (1976) *The Origin of Consciousness in the Breakdown of the Bicameral Mind*. New York: Houghton Mifflin.

MAUDSLEY, H. (1913) Mental organisation: an introductory chapter. *Journal of Mental Science*, **59**, 1–14.

—— (1919) War psychology: English and German. *Journal of Mental Science*, **65**, 65–87.

MILL, J. S. (1873) *Autobiography*. World Classics edition, 1958: Oxford University Press. pp. 34, 232.

POPPER, K. (1974) *Unended Quest: An Intellectual Autobiography*. London: Fontana-Collins.

PUGH, G. E. (1978) *The Biological Origin of Human Values*. London: Routledge and Kegan Paul.

ROSE, S., LEWONTIN, R. C. & KAMIN, L. J. (1984) *Not in Our Genes*. London: Penguin.

SAHLINS, M. (1976) *The Use and Abuse of Biology*. Ann Arbor: University of Michigan Press.

SMITH, C. G. (1985) *Ancestral Voices: Language and the Evolution of Human Conciousness*. New York: Prentice Hall.

SYMONS, D. (1979) *The Evolution of Human Sexuality*. New York: Oxford University Press.

VAN DEN BERGHE, P. L. (1979) *Human Family Systems: An Evolutionary View*. Oxford: Elsevier.

WILSON, E. O. (1978) *On Human Nature*. London: Harvard University Press. p 167.

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Dementia in 2001

SIR: In his review of the various studies of the prevalence of dementia in the elderly (*Journal,* February 1987, **150,** 193–200) Ineichen mentioned the effect of sample size on the results, but did not sufficiently emphasise its importance. The prevalence of a disorder in a sample is only an estimate of the prevalence in the target population, and the reliability of that estimate depends on the size of the sample. The most extreme findings (large and small) tend to be found in those surveys with the smallest samples, as is shown by the studies of severe dementia in those aged 65 and over quoted by Ineichen, where the sample size is inversely correlated with the deviation of the sample prevalences from the mean ($r = -0.78$; $P < 0.01$). The effect of sample size can be quantified by calculating the standard error or confidence interval of the finding, and where the sample is small these will be large in proportion to the estimated prevalence (Clarke *et al,* 1986).

This is particularly true for the very old, where influential prevalence figures for dementia have been derived from small sub-groups of the elderly within samples of those aged 65 and over. In view of the predicted expansion of this particular age group and the implications of this for medical and social services, it is surprising that our working estimates of the prevalence of dementia are still so vague. Ineichen's rule of thumb (1% of 65–74s and 10% of over 75s) may be correct, but in the present state of epidemiological knowledge it remains something of a guess.

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Reference

CLARKE, M., LOWRY, R. & CLARKE, S. (1986) Cognitive impairment in the elderly: a community survey. *Age & Ageing,* **15,** 278–284.

Khat Psychosis

SIR: I echo the call by Critchlow & Seifert (*Journal,* February 1987, **150,** 247–249) for greater awareness of khat consumption in the UK and its psychotropic effects.

Case Report: A 22-year-old Ethiopian male was brought to Accident and Emergency by his brother, with a one-year history of progressively odd behaviour. He had left college and had stopped claiming DHSS benefit. He had drawn strange patterns on the walls of his flat. On direct questioning he denied illicit drug use. On admission he was unkempt, restless, emotionally labile, and experiencing third person auditory hallucinations. He was normotensive with a heart rate of 85 per minute and small pupils. Urine drug screen was negative. He was treated with chlorpromazine (200 mg t.d.s.), and his positive symptoms disappeared after 72 hours. He remained lethargic and withdrawn. Four days later he again became restless, hallucinated, and was unable to sleep. He settled after 48 hours of continued medication. He later stated that prior to his exacerbation he had chewed khat leaves. He had been a habitual chewer from 10 years of age, and prior to admission he had chewed twice weekly.

The level of sympathetic arousal may be helpful in distinguishing the rare cases of khat intoxication producing manic-like psychosis (as described by Giannini & Costellani (1982)), from khat-induced exacerbation of psychosis. Halbach (1972) comments on the rarity of khat-induced psychosis in areas where chewing is endemic and suggests that most reports of associations are of exacerbation of psychosis in predisposed individuals. This may be explained by the bulky nature of the preparation only permitting low plasma levels of the amphetamine-like agent to be attained by chewing.

Khat leaves are chewed fresh for the best effect. The patient informed us that leaves were flown in twice weekly from Ethiopia and sold at a retail outlet in East London. A packet wrapped in banana leaves costs £5 and provides enough for an evening. There are no legal restrictions on the sale or use of this substance in the UK. The regular importation suggests there must be considerable demand for this substance in London. As yet there is no evidence that use of this substance has spread beyond the East African immigrant population. Khat use should be enquired after in patients from this region presenting with psychotic illness.

I would like to thank Dr H. F. Oakeley for his advice and permission to discuss his patient.

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References

GIANNINI, A. & COSTELLANI, S. (1982) A manic-like psychosis due to khat. *Journal of Toxicology – Clinical Toxicology*, **19**, 455–459.

HALBACH, H. (1972) Medical aspects of the chewing of khat leaves. *Bulletin of the World Health Organization*, **47**, 21–29.

Electroconvulsive Therapy in the Elderly

SIR: O'Shea *et al* (*Journal*, February 1987, **150**, 255–257) present the case of a 91-year-old patient who responded well to ECT. The oldest documented patient to receive ECT was a 94-year-old woman (Bernstein, 1972). We describe the successful use of ECT in a woman aged 103 years.

Case Report: The patient, who was born on 6 December 1883, was referred to us by her GP with a history of worsening depression over the preceding weeks. On admission she appeared very depressed and expressed the idea that God no longer wanted her. She felt that she was evil and that she belonged to the devil. She also said that she felt "dirty and rotten" inside. She was preoccupied with religious ideas and spoke of little else. Her family reported that her interest in outside events had diminished prior to admission and her appetite had deteriorated. She was diagnosed as suffering from a psychotic depression.

She told us that her first episode of depression had been 40 years previously, when she lived in England. This had recurred from time to time over the years and her first admission to this hospital was in 1977. That episode was similar in its presentation to the current one. At that time she was treated with ECT but relapsed soon after. Altogether, in 1977 she received three courses of ECT, each of approximately four treatments. There was no definite family history of affective illness. Physical examination during this recent admission revealed that she was suffering from mild congestive cardiac failure and atrial fibrillation. She was receiving digoxin and frusemide for this. She was treated initially with a course of mianserin and later of doxepin. These produced no change, and after eight weeks of in-patient care she was still depressed and miserable. We discussed ECT with her and her family and she agreed to receive a course. The anaesthetist had some reservations, but in view of her persistent depression and previous response to ECT he agreed that it was an acceptable risk to give her a general anaesthetic. She received two treatments in all and was remarkably improved after the second. She was somewhat confused after the treatment, but this disappeared within 24 hours. As she was by now well, it was decided not to proceed to a full course of ECT. It is now two months since the patient's discharge and she remains well.

We think that this report demonstrates the effectiveness of ECT as a treatment for psychotic depression even in the very elderly age-group. We agree with Weiner (1982) that ECT is a relatively safe treatment in the elderly if performed with due precautions.

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References

BERNSTEIN, I. C. (1972) Anorexia nervosa: 94-year-old woman treated with electroshock. *Minnesota Medicine*, **55**, 552–553.

WEINER, R. D. (1982) The role of electroconvulsive therapy in the treatment of depression in the elderly. *Journal of the American Geriatrics Society*, **30**, 710–712.

Delusional Parasitosis

SIR: Macaskill's report (*Journal*, February 1987, **150**, 261–263) of a patient's delusional infestation which responded to non-pharmacological treatment was optimistic, but probably describes a mild form of disorder – as might be expected when clear precipitants are present. However, the potential dangerousness of these patients must be emphasised.

Case report: After the death of his wife and 22 years of police service in Antigua this 70-year-old man emigrated to England, where he has lived alone and worked as a security officer for 18 years (until his retirement in 1982). His complaints about insects started in 1980 when he was rehoused in council property following compulsory purchase of his flat: since then his life has been dominated by delusions of infestation. He has persuaded the council to rehouse him three times, despite having set one flat on fire and been convicted of criminal damage for flooding another. He has also persuaded Environmental Health officers to fumigate his property eight times, although they have never seen any insects in his flats. He was detained under Section 3 of the Mental Health Act in 1983, following a fire, but he was discharged by a tribunal.

In November 1986 he was readmitted under Section 136 following two fires. Mental state on admission revealed a dishevelled, smoke-stained, angry man who shouted loudly and aggressively about his detention and complained that "insects swarm all over me". He was cognitively intact and had a normal computerised tomography scan. During the month of observation while detained under Section 2 this mental state persisted. A Section 3 order was made, and treatment with haloperidol syrup (Andrews *et al*, 1986), up to 30 mg/day, was started. There was a general improvement: he was able to conduct a normal conversation and he became calmer and even friendly. However, his delusions of infestation have persisted and he continues to buy large quantities of fly sprays. He has recently been treated with fluphenazine decanoate as a long-term treatment and because compliance was doubtful at times.

Suicide has previously been reported (Bebbington, 1976), but we are not aware of a report of fire setting associated with this condition, thus indicating that this may be a rare occurrence. However, fumigation is often used by these patients and it seems essential to explore any attempts or intentions by them to use fire or smoke to combat the infestation, and to consider admission in order to protect both the patient and neighbours. Many such patients relapse when treatment is stopped, which raises legal and ethical difficulties when long-term treatment is attempted.

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References

Andrews, E., Bellard, J. & Walter-Ryan, W. G. (1986) Monosymptomatic hypochondriacal psychosis; case studies of treatment with haloperidol. *Journal of Clinical Psychiatry*, **47**, 188–190.

Bebbington, P. (1976) Monosymptomatic hypochondriasis, abnormal illness behaviour and suicide. *British Journal of Psychiatry*, **128**, 475–478.

Is there a Lithium Withdrawal Syndrome?

Sir: The letter from Hawkins & Shepherd (*Journal*, February 1987, **150**, 273) contained a number of inaccuracies and misconceptions.

Firstly, they state that the words 'withdrawal' and 'relapse' are not interchangeable. I make it clear in my opening sentence that the paper is concerned with "the possibility of relapse being caused by drug withdrawal". Their criticism would seem to be an unnecessary exercise in semantics.

Secondly, they state that to show a withdrawal effect it is necessary for there to be a "fall in the relapse rate lower than expected for a period after the withdrawal syndrome has ended". This is clearly wrong. The question is whether there is an *increased* risk of relapse in the withdrawal period in addition to that which would be expected taking into consideration the natural history of the disease process. This is not the same thing.

Thirdly, they state that the theoretical relapse rate "can never be known", and ask "How can one distinguish a withdrawal state causing relapse and relapse alone?" Perhaps they are not aware that this is why control groups are used in order to estimate the theoretical relapse rate. In the control group only 8% of patients relapsed in the first three months, compared with 28% in the experimental group. This difference cannot be explained by the experimental group being at higher risk, because after three months the relapse rates are identical.

Finally, no firm conclusions can be drawn from a retrospective study, hence the title poses a question. Correspondence in the same issue (*Journal*, February 1987, **150**, 264–265) has highlighted the fact that despite increasing use of lithium there has been an increasing readmission rate for mania at a number of different centres. There has been no satisfactory explanation for this. One possibility is that repeated lithium withdrawal increases the number of relapses, and I think this topic deserves further consideration.

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BITE: Self-rating Scale for Bulimia

Sir: The paper by Henderson & Freeman (*Journal*, January 1987, **150**, 18–24) is a useful and timely development in the scientific study of eating disorders. However, four points require further consideration.

Firstly, the authors need to clarify whether they regard the BITE as a screening test or as a diagnostic instrument. The statement that "subjects achieving a high score have a high probability of meeting... criteria for bulimia" indicates clearly that it is a screening test. However, the observations that the BITE "can be used to *identify* binge-eaters in a given population" (our emphasis), and that it provides the information necessary to make a DSM–III diagnosis of bulimia, suggest that the authors also consider it to be a diagnostic instrument.

Secondly, the criterion for caseness is unclear. In study 1, the only criterion given for the patient group is that they were binge-eaters at "various stages of treatment". This is too imprecise – an operational definition is a central requirement for work of this nature. The absence of such a definition from study 1 is all the more mysterious since one was used in study 2. Where diagnostic criteria are considered, it is not always clear which are meant. DSM–III criteria were used in studies 2, 3, and 4, while the authors conclude, in their instructions for administration, that high scorers have a high probability of meeting "DSM–III criteria for bulimia *and* Russell's (1979) criteria for bulimia nervosa" (our emphasis). There is a crucial difference between the two: DSM–III criteria are relatively broad, in contrast to Russell's criteria which require evidence of a morbid fear of fatness. By which criteria does the BITE identify cases?

Thirdly, the authors adopted a case-control strategy. This method gives rise to over-optimistic estimates of the validity coefficients. As Williams *et al* (1980) noted:

> "a group of symptoms selected on the basis of the ability to discriminate between two distinct populations, i.e. 'known ill' and 'known well', may be effective in classifying respondents who happen to come from one of those groups. However, in epidemiology we are not presented with individuals who clearly belong to one of these two groups: we are presented with individuals whose probabilities of illness are distributed along a continuum. Instruments which can distinguish clearly between distinct caseness groups, i.e. well-separated locations on the continuum, need not necessarily perform well in classifying individuals from various and intermediate probabilities of illness."

Another problem with the case-control approach is that since the prevalence of caseness in the study population is set at 50%, the resulting positive predictive value will be considerably higher than that appropriate to the use of the same test in a population where the prevalence is much lower than 50% (Williams *et al*, 1982), as is invariably the case with eating disorders.

Fourthly, there are several methodological points which require clarification. For example, why did the control group in study 1 contain both men and women, whereas the patient group consisted only of women? How were the sub-scales derived? How were the cut-off points decided upon? Where does the proposed lower cut-off (10), which appears in the discussion but not the results, come from? This cut-off is claimed to be relevant in the identification of sub-clinical groups: how can this be so, when no such patients were studied?

The authors are premature in their claim that the BITE is "a tested, valid questionnaire". For example, they say that "the modified BITE produces neither false positives nor false negatives". This is much too sweeping a claim, based as it is on one relatively small validation study. While this questionnaire may fulfil an important need, more development work is required.

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References

RUSSELL, G. (1979) Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychological Medicine*, **9**, 429–448.

WILLIAMS, P., TARNOPOLSKY, A. & HAND, D. (1980) Case definition and case identification in psychiatric epidemiology: review and assessment. *Psychological Medicine*, **10**, 101–114.

——, HAND, D. & TARNOPOLSKY, A. (1980) The problem of screening for uncommon disorders – a comment on the Eating Attitudes Test. *Psychological Medicine*, **12**, 431–434.

Predictions of Outcome in Depressive Illness

SIR: Carney *et al* (*Journal*, January 1987, **150**, 43–48) claim that their study supports the dualist theory of classification of depressive illness. They reach this conclusion on the basis of the finding that their sample of depressive in-patients was not normally distributed on the Newcastle scale and the finding that outcome after two weeks differed between the endogenous and the neurotic groups. Their conclusions about both of these findings are open to different interpretations.

Firstly, depressed patients who are admitted to hospital are extremely unlikely to be a representative sample of all depressed patients: they have generally failed to respond to general practice or out-patient treatment with antidepressant medication. These non-responders will contain disproportionate numbers of patients with severe neurotic and severe endogenous features, the first group being relatively immune to physical treatments, the second group requiring more vigorous physical treatments. Thus, it is hardly surprising that depressed patients admitted "on clinical grounds" do not show a normal distribution of scores on the Newcastle scale.

Secondly, their conclusions about differing outcome between the two groups derives from a comparison of measures before and after fourteen days of a trial of antidepressant medication. Outcome is thus confused with treatment response. As the authors state, albeit in a different context: "the wisdom of attempting to base conclusions about diagnosis and classification on the response to a particular treatment is basically unsound".

Thus, these findings provide no convincing evidence for the dualist theory of the classification of depressive illness.

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The Impact of a Liaison Psychiatric Service on Patterns of Referral in a General Hospital

SIR: It is interesting to read of a change in referral rate associated with the organisation of a liaison psychiatric service (Brown & Cooper, *Journal*, January 1987, **150**, 83–87). However, it would be misleading if the discovery of such a link were to remain as a conclusion in itself.

The real question to be addressed is whether such a change in referral rate represents an increase in work done with patients and their families. If general hospitals were a virgin soil for such work then there would be grounds for thinking this to be so, but this is not the case. Social work departments have been developing liaison work for the past 90 years, and many of the problems encountered by liaison psychiatrists are familiar to us.

Despite the reluctance of some medical and surgical specialists to refer patients, it has been possible for the social worker to offer not only a practical service where appropriate but a skilled therapeutic service to patients suffering from mild affective and neurotic disorders, and preventive work with patients at high risk of such disorders.

For example, at St Charles Hospital, London, the Social Work Department offers counselling to surgical patients and patients with life-threatening medical conditions such as cancer, to all women seeking terminations of pregnancy, and to elderly patients and their relatives. They had also developed a system of assessment of patients admitted following deliberate self-harm in advance of the 1984 DHSS recommendations and in consultation with the psychiatrists at Springfield Hospital.

Because of the social worker's perspective it is often possible to offer a systematic approach which includes and takes account of family and network processes. It is frequently a failure to attend to these that leads to slow discharge or high re-admission rates. It would be unfortunate if an increase in the level of liaison psychiatry with a medical psychiatric model were to undermine this work.

Although much of this is written up in the social work journals it is likely that these are read even less frequently by doctors than the medical journals are read by social workers. Perhaps more collaboration and research is needed in this area.

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A Patient with Resistant Schizophrenia

SIR: We read with interest, but some surprise, the recent report by Roberts *et al* (*Journal*, December 1986, **149**, 789–793). The case highlights many of the difficulties of managing schizophrenia resistant to treatment. However, we were puzzled because the authors presented this as "clearly an unusual case". The tenor of the paper suggested that one is unlikely to find other such patients.

Watt *et al* (1983), in a prospective study, followed 121 patients presenting to the mental health service of a discrete geographical area for five years: 50% failed to become symptom-free in that time, and for over 40% each acute exacerbation of the disorder resulted in progressively less recovery. Among patients with a first episode of schizophrenia referred from nine medical centres in London and its surrounds 17 (nearly 7%) never left hospital throughout the first 20 months of follow-up. (Macmillan *et al*, 1986). On the Denis Hill (Secure) Unit, another ward at the Bethlem & Maudsley Joint Hospital, there are currently 16 out of a total of 27 patients with psychotic illnesses, mostly schizophrenic in type. Five of these have been floridly ill with positive symptoms, probably unremittingly so, for a time approaching that described by Roberts *et al* and have been continuously in hospital with well documented, vigorous treatment with the full range of medication appropriate to psychosis. Some have had ECT too. Another patient who first presented with psychosis and was first admitted only 18 months ago has similarly failed to show any significant reduction of his psychotic features. The other secure units in the South East Thames Region could report a similar situation and the Special Hospitals, in particular Broadmoor and Park Lane, could report many such patients. Even the open forensic unit at the Maudsley Hospital usually has one or two such patients.

How can it be that psychiatrists working together in the same institution can have such different experiences? We would speculate along the following lines. First, psychiatry is becoming increasingly compartmentalised, and with the very considerable clinical demands made on all of us there is insufficient time to explore the interesting byways of other people's wards and practice. Secondly, forensic psychiatric units have been encouraged by general psychiatrists to take aggressive patients off their hands and do so. In our experience treatment resistant schizophrenic patients are frequently aggressive. Thirdly, the policy of dismantling NHS asylums has forced a number of treatment resistant schizophrenic patients into non-medical settings, such as prisons, doss houses, and the streets.

One comment on the treatment resistance itself may be pertinent. Most schizophrenic patients who do not respond to medication (in the sense that their hallucinations and delusions do not go away) do, in fact, derive some benefits from medication, either in terms of sedation, in mood improvement, or in improvement of other neurotic symptoms. Furthermore, our current work shows that although our treatment-resistant psychotic patients almost invariably have unusual cognitive patterns on neuropsychological testing, most can be helped a great deal by the provision of a stress-free milieu (advocated by Murray), together with the careful provision of suitable occupations and recreations. Above all, they need appropriate supportive psychotherapy.

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MACMILLAN, J. F., CROW, T. J., JOHNSON, A. L. & JOHNSTONE, E. C. (1986) The Northwick Park study of first episodes of schizophrenia: III. Short-term outcome in trial entrants and trial eligible patients. *British Journal of Psychiatry*, **148**, 128–133.

WATT, D. C., KATZ, K. & SHEPHERD, M. (1983) The natural history of schizophrenia: a 5 year prospective follow-up of a representative sample of schizophrenics by means of a standardised clinical and social assessment. *Psychological Medicine*, **13**, 663–670.

SIR: I was astonished to read the Maudsley Grand Rounds case report (Roberts *et al*, *Journal*, December 1986, **149**, 789–793). Where is the data to support the view that all "acute symptoms of schizophrenia can be controlled pharmacologically"? Existing data suggests that a substantial proportion of cases show unremitting psychotic symptoms. The landmark Camberwell study of Brown *et al* (1972) included 29 patients who left hospital with persistent symptoms in the 101 consecutive admissions who were discharged to family households. The five patients who remained in hospital probably suffered persistent symptoms. Thus, it seems reasonable to conclude that at that time excellent treatment at the Maudsley Hospital was unable to induce a remission in one-third of cases.

Despite considerable recent advances in our understanding of the pharmacology of schizophrenia there is little evidence to suggest major advances in the efficacy of drug treatment. However, there have been developments in psychological interventions that appear to add to the efficacy achieved by drugs alone. These include a broad range of behavioural psychotherapy interventions (Hagen, 1975; Paul & Lentz, 1977; Falloon, 1985). Before advocating the low stress social environments offered by the long-stay asylum I would recommend pharmacologists to seek the assistance of a skilled behavioural psychotherapist in the comprehensive management of schizophrenia.

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References

BROWN, G. W., BIRLEY, J. L. T. & WING, J. K. (1972) Influence of family life on the course of schizophrenic disorders: a replication. *British Journal of Psychiatry*, **121**, 241–258.

FALLOON, I. R. H. (1985) *Family Management of Schizophrenia: A Study of Clinical, Social, Family and Economic Benefits*. Baltimore: Johns Hopkins University Press.

HAGEN, R. L. Behavioral therapies and the treatment of schizophrenics. *Schizophrenia Bulletin*, **13**, 70–96.

PAUL, G. L. & LENTZ, R. J. (1977) *Psychosocial Treatment of the Chronic Mental Patient*. Cambridge, MA: Harvard University Press.

Psychological Disorders in Obstetrics and Gynaecology

SIR: In her review of *Psychological Disorders in Obstetrics and Gynaecology*, Jequier (*Journal*, December 1986, **149**, 807) applauds the comprehensive coverage assembled by the editor of the book, R. G. Priest.

About one-fifth of all pregnancies end in spontaneous abortion or miscarriage, although estimates range from 10 to 43% (Miller *et al*, 1980; Llewellyn-Jones, 1982). This is comparable in frequency of occurrence to that of induced abortion, which Olley (1985) puts at about three in ten. Yet a complete chapter is given to the latter and the bare mention of the former is in a brief coverage of psychogenic factors in repeated abortion.

It is now being recognised in some of the literature (Raphael, 1984) that spontaneous abortion frequently has profound psychological effects and that these reactions are often missed or mishandled, partly because the event is dismissed as 'routine'. What a pity, then, that this "splendid contribution to the literature" has failed, like the reviewer, to give recognition to such a common and distressing, but often ignored, problem in obstetrics and gynaecology.

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References

MILLER, J. F., WILLIAMSON, E., GLUE, J., GORDON, Y. F., GRUDZINSKAS, J. G. & SYKES, A. (1980) Foetal loss after implantation: a prospective study. *The Lancet*, *ii*, 554–556.

LLEWELLYN-JONES, D. (1982) *Fundamentals of Obstetrics and Gynaecology Vol. I*. London: Faber and Faber.

Olley, P. C. (1985) Termination of pregnancy. In *Psychological Disorders in Obstetrics and Gynaecology* (ed. R. G. Priest). London: Butterworths.

Raphael, B. (1984) *The Anatomy of Bereavement*. London: Hutchinson.

Comprehensive Psychiatric Community Services for the Severely Disabled

Sir: I was interested in Wing & Furlong's plans for a "haven community" to serve the future needs of West Haringey's chronically mentally ill (*Journal*, October 1986, **149**, 449–457), as their proposals have much in common with the developments which have taken place within Oxford's Department of Rehabilitation and Community Care. They rightly stress that disability in one aspect of life does not necessarily imply an equal disability in all areas; any one patient should be able to obtain a unique combination of residential, occupational, and recreational services (Gan & Pullen, 1984).

In 1980 a service was started at Littlemore Hospital to meet the needs of Oxfordshire's 'new long-stay', although patients are accepted on the basis of clinical criteria rather than length of previous admission. This has evolved into the Young Adult Unit, based on a 24-bedded ward, The Eric Burden Community (EBC), on the edge of the Littlemore site (Pullen, 1986). The same building also houses the Unit's out-patient department and is the administrative centre of a developing network of hostels and group homes specifically for the young adult chronically mentally ill. Day care for this group includes a centre, based in an Oxford church, run by the Oxford branch of MIND (Hope & Pullen, 1985).

Our experiences confirm the necessity of providing continuity of care for the severely 'socially disabled' by a single team. A sense of 'haven' can also be created by the knowledge that wherever one happens to be living at the time one still 'belongs' (once accepted, patients remain on the case register of the department indefinitely).

One anxiety I have after reading about the Friern plans concerns the capacity of the proposed 12-bedded mother house to re-admit patients during their inevitable relapses. We have retained the EBC as a hospital ward (albeit one run as a therapeutic community in which, for example, the residents are responsible for the provision of most meals) because we feel that it is at times of crisis that long-term patients most need the care of those who know them well. The psychiatric hospital bed is an essential component of community care.

The Friern Haven appears to be an exciting and imaginative response to the present needs of some of the hospital's existing long-stay patients and to the future needs of Haringey. Elsewhere, other patterns of service will be more appropriate, but in every case there will need to be an integrated network of varied services. I join Wing & Furlong, however, in arguing "that responsibility for the most severely disabled and disturbed group... should be given high priority rather than left as a residual group to be provided for only when all other services are in place".

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References

Gan, S. & Pullen, G. P. (1984) The unicentre: an activity centre for the mentally ill. *British Journal of Occupational Therapy*, **47**, 216–218.

Hope, J. & Pullen, G. P. (1985) 'The Mill': a community centre for the young chronically mentally ill – an experiment in partnership. *British Journal of Occupational Therapy*, **48**, 142–144.

Pullen, G. P. (1986) Community study: The Eric Burden Community. *International Journal of Therapeutic Communities*, **7**, 191–200.

Delineating Social Phobia

Sir: Solyom *et al* (*Journal*, October 1986, **149**, 464–470) state that "the problem of overlap may also arise because both agoraphobia and social phobia may be based on fear of having a panic attack, i.e. both may suffer from panic disorder."

DSM–III and DSM–III–R (8/1/86 draft) attempt to distinguish social phobia from panic disorder and agoraphobia. Social phobics in DSM–III and DSM–III–R are defined by the occurrence of intense anxiety specifically in response to feeling scrutinised or evaluated by other people (or in anticipation of this). This differs from the panic attack associated with Panic Disorder or Agoraphobia. To meet the DSM–III–R definition of Panic Disorder, an individual has to experience intense anxiety episodes occurring at times when the patient is not the focus of others' attention.

Soylom *et al* appear to be questioning this diagnostic convention. The reasonable question to ask is: does the convention make sense? Specifically, is there a meaningful basis to distinguish between the anxiety experience of Social Phobia and the panic attacks associated with Panic Disorder or Agoraphobia with Panic Attacks?

Several lines of evidence suggest qualitative differences between the anxiety experience of social phobic and panic disorder or agoraphobic patients. Aimes *et al* (1983) noted differences in the symptom pattern.

Social phobics reported more blushing and muscle twitching, and less limb weakness, breathing difficulty, dizziness or faintness, actual fainting, and buzzing or ringing in the ears than did agoraphobics.

Analogue studies and open clinical trial suggest greater beta-blocker efficacy in social phobia than agoraphobia (Gorman *et al*, 1985). Tricyclics are quite helpful for blocking panic attacks. Clinically they seem less useful in social phobia. Controlled trials to confirm these impressions, however, are needed. MAO inhibitors appear useful in both conditions.

Plasma epinephrine does not appear to be elevated during *in vivo* or lactate induced panic attacks (Liebowitz *et al*, 1985). A study in progress is demonstrating that at least a subset of social phobics (perhaps 50%) demonstrate plasma epinephrine elevations of 50% or more during performance challenge. Studies in medical house officers suggest two to three-fold epinephrine rise during more stressful real life public speaking (Dimsdale & Moss, 1980). Social phobics are significantly less likely to panic with sodium lactate infusion than are agoraphobics (Liebowitz *et al*, 1985).

Onset and offset seem to differ as well. Social or performance anxiety is triggered only when the individual is faced with interpersonal evaluation or scrutiny, and is probably induced by cognitions that one will perform badly, look foolish, etc. Panic attacks related to panic disorder often occur unexpectedly, especially early on, without obvious cognitive precursors. When feeling anxious, social phobics prefer to be alone to recover their equilibrium; agoraphobics are less likely to panic, and more readily recover, in the presence of a trusted person.

Both agoraphobics and social phobics do fear the onset of severe anxiety experience. Available evidence, however, suggests important differences in pathophysiology, which Solyom's statement obscures.

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References

AIMES, P. L., GELDER, M. G. & SHAW, P. M. (1983) Social phobia: a comparative clinical study. *British Journal of Psychiatry*, **142**, 174–179.

DIMSDALE, J. E. & MOSS, J. (1980) Short-term catecholamine response to psychological stress. *Psychosomatic Medicine*, **42**, 493–497.

GORMAN, J. M., LIEBOWITZ, M. R., FYER, A. J., CAMPEAS, R. & KLEIN, D. F. (1985) Treatment of social phobia with atenonol. *Journal of Clinical Psychopharmacology*, **5**, 298–301.

LIEBOWITZ, M. R., FYER, A. J., GORMAN, J. M., DILLON, D., DAVIES, S. O., STEIN, J. M., COHEN, B. S. & KLEIN, D. F. (1985) Specificity of lactate infusions in social phobia *vs* panic disorders. *American Journal of Psychiatry*, **142**, 947–949.

——, GORMAN, J. M., FYER, A. J., LEVITT, M., DILLON, D., LEVY, G., APPLEBY, I. L., ANDERSON, S., PALIJ, M., DAVIES, S. O. & KLEIN, D. F. (1985) Lactate provocation of panic attacks: II. Biochemical and physiological findings. *Archives of General Psychiatry*, **42**, 709–719.

Clinical Tests of Memory as Sensitive and Specific Signs of Dementia

SIR: Psychogeriatricians could make greater use of Kopelman's evaluation of clinical tests of memory (*Journal*, October 1986, **148**, 517–525) if his organic and non-organic groups are sub-divided to allow the analysis of Alzheimer and depressed groups. This would extend the validity of several tests of memory, comprehension, and orientation as contributors to the common and problematic differential diagnosis of depressed versus Alzheimer patients. Using the number of subjects failing to score at cut-off point for each test (his Table III), the relative discrimination between these groups was assessed with the two-tail Fisher exact probability test (Armsen, 1955). The Gresham memory and orientation questionnaire, Wechsler logical memory, paired associates, name and address, and anomalous sentences tests all show significant discrimination beyond the 0.05 level of probability. Although test cut-off points were chosen to maximise the discrimination between organic (Korsakoff and Alzheimer) and non-organic (healthy controls and depressed), some show sensitivity exceeding 80% to Alzheimer's disease and specificity exceeding 80% relative to depression and healthy controls: logical memory with 45 minute retention below 50% of immediate retention; paired associates total score less than 14: name and address learnt in more than two trials. This is sufficiently promising to be worth cross-validating. A table of discrimination, sensitivity, and specificity is available from the author.

The publication of revised cut-off points chosen to maximise the discrimination of Alzheimer's disease from depression and normal controls, taking into account the base-rates for these pathologies, would greatly assist the selection and monitoring of cases in clinical trials. The availability of sensitivity, specificity, and misclassification rates as a function of test score (Anthony *et al*, 1982) would permit the selection of a cut-off point with high sensitivity for a screening study and a cut-off point with high specificity in a study of unambiguous groups of normal, depressed, and Alzheimer patients.

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References

ANTHONY, J. C., LERESCHE, L., NIAZ, U., VON KORFF, M. R. & FOLSTEIN, M. F. (1982) Limits of the 'Mini-Mental State' as a screening test for dementia and delirium among hospital patients. *Psychological Medicine*, **12**, 397–408.

ARMSEN, P. (1955) Tables for significance tests of 2 × 2 contingency tables. *Biometrika*, **42**, 494–505.

Caffeine and Panic Attacks

SIR: It has not been clearly shown that caffeine alone can cause panic attacks in normal subjects. I report an adverse reaction which occurred during a normal volunteer study in which caffeine was compared against placebo and rolipram (a CNS phosphodiesterase inhibitor).

Case report: A 21-year-old healthy male volunteer with neither a history of panic attacks nor severe anxiety was given 500 mg of caffeine – equivalent to 5 – 8 cups of coffee – in the form of 1 gm of caffeine citrate in an orange drink. After approximately 20 minutes the subject started to feel an intense dread, which was quickly followed by the somatic symptoms of severe anxiety, including sweating, palpitations, and physical restlessness. He was unable to tolerate staying in the experimental room, and he had fears of impending death. Diazepam (15 mg) was given intravenously, which quelled both the physical and the cognitive changes, but did not remove them entirely. After around 1½ hours the anxiety symptoms returned and further diazepam was required.

This adverse reaction has implications both in theory and in clinical practice. Firstly, the interaction between the benzodiazepines and caffeine is not yet fully understood (File *et al*, 1982); Ghoneim *et al*, 1986). It is known that caffeine is an adenosine receptor antagonist at concentrations found in plasma (Daly *et al*, 1981) and not, as had previously been thought, a phosphodiesterase inhibitor. Diazepam interacts at the benzodiazepine receptor, yet this experiment shows that a benzodiazepine will attenuate the symptoms of anxiety induced by caffeine. This is shown by the return of the anxiety after around 1½ hours, when the diazepam is unbound from the receptor; the plasma half-life of caffeine is of the order of 3–6 hours (or longer) in healthy non-smoking men (Axelrod & Reichenthal, 1953). This would imply that the causation of anxiety might in some way be related to adenosine receptor antagonism. Secondly, it shows that large doses of caffeine can cause severe anxiety in normal people, and therefore an estimation of caffeine intake needs to be part of the assessment of panic attacks. Although this may seem a large dose of caffeine it is not uncommon, as Graham (1978) showed, for people in the general population to drink this quantity in a 24-hour period. Finally, this finding also emphasises the importance of caffeine in the generation of anxiety, which does not amount to panic, in the coffee-consuming general population.

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References

AXELROD, J. & REICHENTHAL, J. (1953) The fate of caffeine in man and a method for its estimation in biological material. *Journal of Pharmacology and Experimental Therapeutics*, **107**, 519–523.

DALY, J. W., BRUNS, R. F. & SNYDER, S. H. (1981) Adenosine receptors in the central nervous system: relationship to the central actions of methylxanthines. *Life Sciences*, **28**, 2083–2097.

FILE, S. E., BOND, A. J. & LISTER, R. G. (1982) Interaction between effects of caffeine and lorazepam in performance tests and self ratings. *Journal of Clinical Psychopharmacology*, **2**, 102–106.

GHONEIM, M. M., HINRICHS, J. V., CHIANG, C-K. & LOKE, W. H. (1986) Pharmokinetic and pharmacodynamic interactions between caffeine and diazepam. *Journal of Clinical Psychopharmacology*, **6**, 75–80.

GRAHAM, D. M. (1978) Caffeine – its identity, dietary sources, intake, and biological effects. *Nutrition Reviews*, **36**, 97–102.

Hyponatraemia and Lofepramine

SIR: Hyponatraemia in psychiatric patients has been variously attributed to compulsive water drinking (Ferrier, 1985), to the primary psychiatric disorder (Singh *et al*, 1985), and to the syndrome of inappropriate secretion of anti-diuretic hormone (SIADH) induced by psychotropic drugs (Sandifer, 1983; Streeten *et al*, 1981). SIADH is a recognised complication of tricyclic antidepressants, and has been reported in association with amitriptyline and desipramine (Sandifer, 1983). We wish to report a case of hyponatraemia which was probably due to SIADH in association with lofepramine.

Case report: A 52-year-old married woman with a history of schizoaffective illness was admitted as an emergency into a medical ward with an acute onset of lethargy, anorexia, vomiting, severe weight loss, and confusion. There were no significant findings on physical examination, but she had repeated abnormal biochemical results. The initial report was: Na^+ 110 mmol/litre, K^+ 3.3 mmol/litre, urea 1.9 mmol/litre, and creatinine 64 mmol/litre. Her urine sodium and osmolarity were not determined.

She had been off all neuroleptics for six months, but had been prescribed lofepramine (140 mg daily) four weeks before her presentation. The hyponatraemia persisted until her lofepramine was stopped, and within 36 hours it resolved. It is of interest that she had been on fluphenazine, haloperidol, trifluoperazine, iprindole, and amitriptyline in the past without any undue effect.

It is likely that her condition was precipitated by lofepramine. However, we were unable to confirm this conclusively, since it would have needed a challenge test to do so. However, we hope that this case will draw attention to the possibility of a drug-induced hyponatraemia in psychiatric patients.

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References

FERRIER, I. N. (1985) Water intoxication in patients with psychiatric illness. *British Medical Journal, ii,* 1594–1596.

SANDIFER, M. G. (1983) Hyponatraemia due to psychotropic drugs. *Journal of Clinical Psychiatry,* **44,** 301–303.

SINGH, S., PADI, M. H., BULLARD, H. & FREEMAN, H. (1985) Water intoxication in psychiatric patients. *British Journal of Psychiatry,* **146,** 127–131.

STREETEN, D. H. P., MOSES, A. M. & MILLER, M. (1981) Disorders of the neurohypophysis. In *Harrison's Principles of Internal Medicine* (eds K. J. Isselbacher, R. D. Adams, E. Braunwald, R. G. Petersdorf & J. D. Wilson). Tokyo: McGraw-Hill.

Clomipramine and Flupenthixol – Additive Antidepressants?

SIR: The possibility that flupenthixol and tricyclics would have an additive antidepressant effect was first suggested by Reiter (1969), although the combination has never been specifically evaluated. This report shows the effect of manipulating flupenthixol dose when a stable tricyclic regime is being given concurrently.

Case report: A 31-year-old female with a severe, recurrent, unipolar, depressive psychosis met RDC criteria (Spitzer *et al,* 1977) for major depressive disorder, endogenous, psychotic, and retarded sub-types. She was non-delusional, but had suicidal and homicidal hallucinations. She was a DST non-suppressor, and had a Hamilton score of 38. In the first 25 months of her third episode, she was refractory to treatment with tricyclic antidepressants, lithium/tricyclic, tricyclic/antipsychotic, and lithium/tricyclic/antipsychotic combinations, carbamazepine and ECT. Marital therapy, cognitive-behaviour therapy, light-exposure and an exclusion diet were similarly ineffective.

At that time, her medication was changed to clomipramine (300 mg/day), L-tryptophan (1 g b.d.) and thioridazine (100 mg t.i.d.) but, despite four weeks on this regime, her mental state deteriorated and remained refractory to six further ECTs. Her hallucinations were prominent, she was sleeping only two or three hours a night, showed virtually no diurnal variation, and had become increasingly agitated. We decided to add flupenthixol to her regime, the depot form being chosen because of previous non-compliance: 20 mg was given initially and 40 mg three days later. Five days later she began to improve rapidly, with marked attenuation of hallucinations, sleep disturbance, and social withdrawal. Twenty-four hours later she was expressing optimism regarding the future, the first such emotion in over two years. Fortnightly injections were planned. Three days after her next injection she suddenly relapsed, but eight days later she improved. Two further cycles of eight days well and eight ill followed. We attempted to rationalise her medication by replacing thioridazine with flupenthixol, but, despite increasing flupenthixol, we were unable to reduce her thioridazine. Indeed, a pattern of decreased well-being on increased doses of flupenthixol developed, no symptomatic relief being obtained on 80 mg flupenthixol fortnightly.

At this stage further injections were withheld, in the expectation of clinical improvement. Nine days later (23 days after 80 mg flupenthixol had been administered) improvement began. The following day 20 mg was administered. Improvement continued until seven days later, when she claimed to be "as well as I have ever been". A regime of 20 mg fortnightly was adopted. On this regime, withdrawal of thioridazine and L-tryptophan and halving the dosage of clomipramine had no adverse effect on her mental state, and she remained free from affective symptomatology until her discharge six weeks later. Three weeks after discharge she began to deteriorate, hallucinations returning after a further three weeks and re-admission occurring one week later. She admitted not taking her clomipramine after discharge, although she had received flupenthixol injections as planned. Six days after reintroduction of clomipramine (150 mg/day) she began to improve.

Since the patient's medication regime was uncontrolled, it is not possible to draw firm conclusions about her response. However, improvement occurred only when flupenthixol and clomipramine were given in combination, and was maintained only with a dose of flupenthixol not normally considered antipsychotic. Indeed, beneficial effects were lost at higher doses. It is therefore unlikely that improvement was due to either agent alone. The close relationship between her mental state and manipulation of her medication make spontaneous fluctuation unlikely. Johnson & Malik (1975) describe mood elevation in the seven days following flupenthixol injection – our patient did not show this pattern.

It is likely that her response is unrelated to the antipsychotic effects of flupenthixol. It has been assumed that the antidepressant effect of flupenthixol is manifested at lower doses than the anti-psychotic effect – although this has never been proven by case studies or clinical trials. The most likely explanation for her response is an interaction between clomipramine and flupenthixol, not involving the anti-psychotic effects of the latter, the rapidity of the response to the drugs in combination suggesting synergism between the two compounds.

Illness episodes of this chronicity and severity are uncommon and we think that the fact that she responded to the combination is encouraging. More case reports of such efficacy in resistant depression would be necessary to assess the feasibility of this regime being used in a clinical trial in this condition.

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JOHNSON, D. A. W. & MALIK, N. A. (1975) A double-blind comparison of fluphenazine decanoate and flupenthixol decanoate in the treatment of acute schizophrenia. *Acta Psychiatrica Scandanavica*, **51**, 257–267.

REITER, P. (1969) On flupenthixol, an antidepressant of a new chemical group. *British Journal of Psychiatry*, **115**, 1399–1402.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1977) *Research Diagnostic Criteria (R.D.C.) For a Selected Group of Functional Disorders – 3rd Edition*. Biometrics Research: New York State Psychiatric Institute.

Water Intoxication in Congenital Neurosyphilis

SIR: Water intoxication may occur in almost any psychiatric disorder (Ferrier, 1985), but this is the first report of it in congenital neurosyphilis. In about 80% of cases of water intoxication the patients are psychotic (usually schizophrenic). Although this patient was psychotic, mentally retarded, and had some abnormal neurological signs, the precise diagnosis of congenital neurosyphilis was delayed, as by the time she was seen VDRL and TPHA were negative and Hutchinson's teeth had been extracted.

Case report: A 48-year-old mentally retarded woman was admitted to a psychiatric ward after she violently attacked her 75-year-old mother, with whom she still shared a bed at night. Prior to admission she was doing little apart from smoking heavily (80 cigarettes a day) and drinking fluids excessively. Each day she would consume 3 pots of tea, 6 pints of milk, 2.5 litres of orange juice, and an unknown quantity of water directly from the taps. The patient had three previous admissions to the psychiatric unit. The first time, at the age of 45, diagnoses of mental retardation, alcoholism, and depression with obsessional and hypochondriacal features were made. The second admission (aged 46) followed her attempt to set fire to her home. She was still abusing alcohol (8–10 pints of beer per day) and was depressed. While in hospital she drank water to such an extent that she developed hyponatraemia, hypokalaemia, and had *grand mal* seizures. During the third admission (at the age of 47) she was still drinking alcohol and water excessively, and was thought to be depressed. Following discharge she did not abuse alcohol, but continued to drink water as before. On this fourth occasion she presented as a slim, dishevelled, perplexed woman who was incontinent of urine. She was agitated, incoherent at times, mumbled, and drew a cross on my forehead with her finger and prayed, but was unable to explain why. She was disorientated in time and place, described herself as being frightened and everything around her as being larger (macropsia). There were no auditory hallucinations or systematised delusions. Physical examination of cardiovascular and respiratory systems was normal. There was an abdominal scar from a duodenal ulcer operation. Central nervous system examination showed decreased visual acuity and two small aneurysms in the right eye. The right knee jerk was absent and the right plantar response was up; the left plantar response was normal. The ankle jerks were absent. Proprioception was impaired, but more on the right side. Pin prick showed glove and stocking peripheral neuropathy. She had a tremor of both hands. Her gait was slightly ataxic. Musculoskeletal system was normal apart from swelling over right knee and left ankle.

Investigations: FBC, thyroid, liver function tests, serum glucose, midstream urine culture, ECG, and EEG were normal. VDRL, TPHA, FTA were negative. CT scan showed minimal degree of general cerebral atrophy. Urea, calcium, sodium, and potassium were below the lower end of the reference range, in keeping with excessive water intake. Urinary osmolality was 116 mosmol/kg (reference range 40–1400) and plasma osmolality was 277 mosmol/kg (reference range 275–295). Old case notes were obtained from the Rheumatology Department; those stated that at the age of 19 (when she presented with backache) her Hutchinson's teeth were noticed. WR was found to be negative, TPI positive, and CSF gave a typical Lange curve. She was treated with a full course of penicillin and Hutchinson's teeth were extracted.

Progress: following administration of trifluoperazine (15 mg nocte, orally) her fluid intake decreased, she became continent, and her mental state improved, but the serum electrolytes were still abnormal, so demeclocycline (250 mg q.d.s.) was added. Further restrictions on water intake and cigarette smoking were made. On discharge her mental state was normal apart from mental retardation (IQ 63) and slight memory defect. Her fluid intake was 1.9 litres daily, and all electrolytes were normal. Two weeks later she stopped taking her medication and soon had a relapse. The same treatment was attempted on an out-patient basis, and she again improved.

Diabetes insipidus has been reported as a rare manifestation of congenital neurosyphylis, but no previous reports of water intoxication were found in the literature. While in both conditions polydipsia and polyuria occur, in diabetes insipidus urinary osmolality is well below plasma osmolality, and serum urea and sodium tend to be raised. In water intoxication, both plasma and urinary osmolality are low and serum urea and sodium are below reference values. The syndrome of inappropriate antidiuretic hormone secretion in which hyponatraemia occurs can be excluded by the absence of raised urinary osmolality. This patient has been treated with trifluoperazine, and although psychotic symptoms were effectively treated the water intake remained excessive. Dopamine has been shown to inhibit vasoprotein release in man (Lightman & Forsling, 1980) and it is suggested that in this case its antagonism by trifluoperazine could have led to increased ADH. Demeclocycline, an antidiuretic hormone antagonist, was therefore added to the treatment, and this was followed by the return of serum electrolytes to reference values. However, the effects of neuroleptics on ADH secretion are inconsistent and I suggest that the personal habits of the patient such as smoking and tea and alcohol intake were not considered. This patient drank large quantities of tea and smoked heavily prior to this admission. Theophylline increases cAMP and diuresis. The effect of nicotine is to increase ADH and decrease diuresis, while alcohol has the opposite effect (Laurence, 1977). Therefore, smoking and tea intake should be controlled as part of treatment for water intoxication.

I am grateful to Dr D. Hailstone for his permission to report this patient.

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References

FERRIER, I. N. (1985) Water intoxication in psychiatric illness. *British Medical Journal*, **291**, 1594–1596.

LAURENCE, D. R. (1977) *Clinical Pharmacology*. London: Churchill Livingstone.

LIGHTMAN, S. L. & FORSLING, M. (1980) Evidence for dopamine as an inhibitor of vasoprotein release in man. *Clinical Endocrinology*, **12**, 39–46.

Neuroleptic Malignant Syndrome and Compulsive Water Drinking

SIR: The neuroleptic malignant syndrome (NMS) is thought to represent an idiosyncratic reaction to dopamine blockade, probably mediated via basal ganglia and hypothalamic pathways (Szabadi, 1984). Compulsive water drinking has been associated with a variety of organic and psychiatric disorders, particularly psychosis (Ferrier, 1985). Although the biochemical control of thirst remains uncertain, it seems likely to involve dopaminergic pathways in the hypothalamus. Both these conditions carry a significant morbidity and mortality and are probably underdiagnosed. I report a patient who exhibited compulsive drinking while suffering NMS.

Case report: A 27-year-old woman was admitted, mute, with generalised muscle rigidity, coarse Parkinsonian tremor, sialorrhoea, difficulty swallowing, and sweating. She was indiscriminately incontinent of urine, and had episodic attacks of tachycardia and profuse sweating. She was never pyrexial. This condition had developed immediately after starting haloperidol (one week previously), and failed to respond to oral or intravenous anti-cholinergic drugs. A diagnosis of NMS was made, and all neuroleptic drugs were stopped. One week later she was observed to be drinking copious amounts of fluid; drinking from taps, water jugs, undiluted bottles of squash, and continually requesting drinks on top of those provided with her diet. She was still rigid and incontinent. The excessive drinking lasted five days, and proved impossible to quantify. Urea and electrolytes had been normal prior to excessive drinking (blood urea slightly reduced at 2.7 mmol/litre; reference range 3.0–7.0 mmol/litre). Two weeks after discontinuation of neuroleptic drugs her condition had completely recovered and there was no evidence of psychosis.

Although I am not aware of any reported association between excessive drinking and NMS, Wedzicha & Hoffbrand (1984) described a case of recurrent NMS associated with hyponatraemia. The authors suggested that the hyponatraemia may have precipitated NMS, and they noted that the patient was observed to be drinking excessive water on the ward, which raises the possibility that the excessive drinking and hyponatraemia could have been secondary to NMS. The association between neuroleptic drugs and drinking behaviour is unclear. Smith & Clarke (1980) concluded that increased thirst is likely to be related to the hyperdopaminergic state, but speculate that neuroleptic drugs may induce thirst by a mechanism similar to that proposed for tardive dyskinesia, i.e. denervation supersensitivity, occurring at hypothalamic dopamine receptors.

Once recovered, our patient was unable to recall drinking excessively or to explain it. Unfortunately, we do not have the data to comment on whether water intoxication occurred, or if there was any disturbance of antidiuretic hormone production, although this would be of great interest. It is not possible to say whether the two conditions were causally related, but they do have in common a disturbance of hypothalamic dopamine function and at least one reported patient with NMS (Wedzicha & Hoffbrand, 1984) has been observed to drink excessively and to develop hyponatraemia. Given the difficulties in recognising compulsive drinking before serious consequences occur, in awareness of the possibility is essential.

References

Ferrier, I. N. (1985) Water intoxication in patients with psychiatric illness. *British Medical Journal*, **291**, 1594.

Smith, W. O. & Clarke, M. L. (1980) Self-induced water intoxication in schizophrenic patients. *American Journal of Psychiatry*, **137**, 1055–1060.

Szabadi, E. (1984) Neuroleptic malignant syndrome. *British Medical Journal*, **288**, 1399.

Wedzicha, J. A. & Hoffbrand, B. I. (1984) Neuroleptic malignant syndrome and hyponatraemia. *The Lancet*, *i*, 963.

Switch Mechanism in Affective Illness and Oral S-Adenosylmethionine (SAM)

Sir: A mechanism for switching manic-depressives out of depression has been postulated (Bunney *et al*, 1970). The spontaneous switch rate from depression to hypomania or mania in untreated unipolar and bipolar depressives was reported to be 10–14% over a six-month period (Bunney, 1978; Lewis & Winokur, 1982). Drugs acting on brain biogenic-amines, like tricyclic anti-depressants, are known to give rapid switches of mood from depression to mania; increases in these amines are thought to underlie these changes. The possibility of switching retarded, potentially suicidal depressives into a less hazardous euphoric state has long excited speculation (Bunney, 1978; Lewis & Winokur, 1982). There would be advantages if this could be effected by means of a naturally occurring physiological brain metabolite given by a non-intimidating procedure rather than by the use of drugs or ECT.

We have recently reported (Carney *et al*, 1983) switching in three of 12 depressives receiving i.v. SAM. We calculated that the probability of such an event occurring by chance within six days of starting SAM was remote ($P<0.001$) and it was probably associated with SAM. However, there was no certainty that oral SAM, unavailable during the previous study, would have the same effect as i.v. SAM.

At Northwick Park Hospital we gave SAM in daily doses of 500–1600 mg orally for 14–42 days in an open study to six endogenous depressive in-patients (Newcastle score 6 or more (Carney *et al*, 1965)). Three became hypomanic during the trial. Thus, a 26-year-old female teacher whose brother had developed mania after receiving i.v. SAM in the previous study became hypomanic. A 50-year-old female clerk who had become hypomanic when given i.v. SAM in the former study showed a similar reaction. A 70-year-old man with no previous or family history of affective illness, unresponsive to other anti-depressant treatments, including ECT (the other two patients were considered for ECT), developed hypomania. All had been retarded, non-verbal, seclusive, and exhibited nihilistic or intrapunitive delusions. Each went through a brief euthymic period of 1–3 days before suddenly becoming elated, with marked increases in the production of speech and activity, and all developed grandiose ideas. One had heightened libido. Over the trial period the mean plasma SAM level in four of these patients increased from 30.5 to 144.0 ng/ml, i.e. from 2 to 10 times the basal mean value.

We have already demonstrated that SAM can cross the blood-brain barrier and influence monoamine metabolism (Bottiglieri *et al*, 1984). These results with oral SAM support the earlier report of switching (Carney *et al*, 1983) associated with the use of i.v. SAM and again suggest that some apparently endogenous depression is SAM-responsive (Reynolds *et al*, 1984). SAM is the major source of methyl groups in the brain, and intimately connected with brain and amine metabolism and membrane and neuro-transmitter function. Further studies of oral SAM in endogenous depression would be worthwhile. We are currently undertaking double-blind placebo-controlled trials of i.v. and oral SAM in patients with endogenous depression.

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References

Bottiglieri, T., Laundy, M., Martin, R., Carney, M. W. P., Nissenbaum, H., Toone, B. K., Johnson, A. L. & Reynolds, E. H. (1984) S-adenosylmethionine influences monoamine metabolism. *The Lancet*, *ii*, 224.

Bunney, W. F. (1978) Psychopharmacology of the switch process in affective disorder. In *Psychopharmacology; A Generation of Progress* (eds M. A. Lipton, A. Damascio & K. F. Kellanil). New York: Raven Press.

——, MURPHY, D. C., GOODWIN, F. Y. & BORGE, G. F. (1970) The switch process from depression to mania. Relationship to drugs. *The Lancet*, *i*, 1022–1027.

CARNEY, M. W. P., ROTH, M. & GARSIDE, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

——, MARTIN, R., BOTTIGLIERI, T., REYNOLDS, E. H., NISSENBAUM, H., TOONE, B. K. & SHEFFIELD, B. F. (1983) Switch mechanism in affective illness and S-adnosylmethionine. *The Lancet*, *i*, 820–821.

LEWIS, J. L. & WINOKUR, G. (1982) The induction of mania; a natural history study with controls. *Archives of General Psychiatry*, **39**, 303–306.

REYNOLDS, E. H., CARNEY, M. W. P. & TOONE, B. K. (1984) Methylation and mood. *The Lancet*, *ii*, 196–197.

A HUNDRED YEARS AGO

Causes of Idiocy

Although Dr Down's patients came from every variety of social rank, he could not draw any safe deductions as to the influence of various occupations of the parents on the causation of idiocy. He had collected 400 cases with fair social antecedents, and had found that 75 per cent were the children of merchants, country gentlemen, officers in the army and navy, gentlemen of independent means and liberal education, and members of the titled aristocracy. No less a proportion than 25 per cent were the children of members of one or other of the three *learned professions*. Of these, 18 per cent were children of members of the clerical profession. Mr Galton finds that the legal profession (which procreates fewest idiots) gives birth to 11 per cent of the foremost men of eminence; that the medical profession (which procreates 1 per cent more of idiots than the legal) gives origin to 9 per cent of scientific men; while the clerical profession, which procreates the maximum amount of idiocy, gives birth to only 6 per cent of men of science. These statistics illustrated the process of natural selection.

Reference

DOWN, J. L. (1887) Mental affections of childhood and youth. *The Lancet*, 22 January 1887, 163.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

British Journal of Psychiatry (1987), **150**, 726–735

Book Reviews

Editor: Sidney Crown

Examination Notes on the Scientific Basis of Psychiatry. By TIMOTHY G. DINAN. Bristol: Wright. 1985. Pp 148. £7.25.

The aim of this book is to provide a revision text for the disparate array of basic sciences knowledge which currently comprises the syllabus for the Part 1 MRCPsych examination. It succeeds in twelve brief chapters. In his disarming preface Dinan, a lecturer at St Bartholomew's Hospital, warns that some readers might find the book "intellectually emaciated". He need not worry: the short notes format renders the text spare, certainly, but there is no lack of intellectual reward.

The opening chapters on neurosciences reflect Dinan's academic background and are excellent. Post-membership clinicians looking for an up-to-date distillation of this fast-moving area will find it here. Chapters on psychopathology, diagnosis and classification, psychopharmacology, epidemiology and statistics, and social psychiatry all achieve a uniformly high standard which should equip any candidate with more than adequate ammunition. The essentials of psychology and neuropsychiatry are all there, although future editions might usefully include a little more about neuropsychology and drop the old epilepsy classification. The single weak section is that on genetics: three pages is hardly enough to encompass the genetics of adult mental illness and, in my experience, the preliminary exam requires more than is here.

This is a good, cheap, compact book which capably fills a long-felt gap in the psychiatric textbook market.

S. W. LEWIS, *Lecturer and Honorary Senior Registrar, Institute of Psychiatry, London*

Psychopathology in Epilepsy: Social Dimensions. Edited by STEVEN WHITMAN and BRUCE P. HERMANN. Oxford: Oxford University Press. 1986. Pp 309. £35.00.

As recently as 1956, 18 American states still had "eugenic sterilisation laws applicable to people with epilepsy". This is just one of the more recent events in a story of prohibition and prejudice. It is hardly surprising that there should be a perceived sense of stigma pertaining to epilepsy and that this social process should have an effect on emotional reactions in people with the disorder.

It may seem self-evident that people with epilepsy should develop emotional problems not only as a direct consequence of the illness but also because of psychosocial issues such as threat of unemployment or perceived discrimination. However, there are less obvious factors; one contributor presents evidence that about two-thirds of his sample reported a fear that they may die with their next seizure.

The declared aim of this multi-author book is to redress the imbalance between biological and social approaches to the study of the psychopathology seen with epilepsy. Each chapter is designed to stand alone and, despite editorial introductions to each section, there is little opportunity to develop an argument through the whole work. The contributions vary in interest. It often seems that too much time is being devoted to a detailed discussion of the contributors' own research, especially when this has already been published. This is a pity, because there are some important messages in the book. One of the two (British) longitudinal studies demonstrates little evidence either of poorer abilities or of discrimination among children with epilepsy. Nonetheless, the subjects seemed to feel stigma; as children they were more prone to develop emotional disturbance, and as adults they were more discontented with their lives. This discrepancy between the subjective and the objective is both an academic puzzle and a therapeutic challenge.

This is a book priced for the library rather than the individual. It is unlikely to have a general appeal, but selective reading would be of interest to those who work in the field and have not read the original reports.

STUART TURNER, *Lecturer in Psychological Medicine, King's College School of Medicine and Dentistry, London*

Controversies in Alcoholism and Substance Abuse. Edited by BARRY STIMMEL. New York: The Haworth Press. 1986. Pp 236. $34.95.

This book contains a collection of papers dealing with various aspects of alcohol and substance abuse, from American workers in the discipline. Whilst the editor is at pains to disclose the 'politicalisation' of the organisations involved in this field, the only contribution I found contentious was a short paper on marijuana. The articles might therefore not be particularly controversial, but in the main they are thorough, well informed, and up-to-date. On the grounds that most of the bad habits, cults, and crazes of the USA, like 'Hurricane Charlie' eventually arrive on our shores, we should be forewarned and forearmed.

I found the contributions covering the abuse of cocaine helpful. Clinical, behavioural, and biochemical aspects are discussed in some depth. The description of, and the information derived from, the notional telephone helpline 800-Cocaine is particularly relevant to what we might be faced with in the future, whether it be the folklore and jargon or the statistics.

There are two articles on AIDS – one dealing with the epidemiology as applicable to the USA, and the association of AIDS with intravenous drug misuse, and the other concerned with diagnosis and early recognition. There are two papers on medicinal approaches to opiate abuse, including narcotic antagonists (naltrexone) and psychotropic drugs, followed by a contribution by the editor advocating caution in the use of psychotropic drugs for individuals with a past history of substance abuse, specifying some special hazards.

The use of narcotics for the relief of pain and the attendant underusage ('Americanised' into 'opiophobia') are dealt with sensibly if one reads beyond the jargon of the abstract which introduces each chapter.

Finally, I would single out (there are some other contributions on social drinking in pregnancy) a paper on the cost-effectiveness of in-patient treatment of alcoholics. This is a timely review and is not confined to transatlantic findings. The development of day units in the UK since the mid-seventies would appear to be vindicated on the grounds of both cost and effectiveness, when reviewed alongside experience in the USA and Canada.

G. SEDMAN, *Consultant Psychiatrist, St Ann's Hospital, Poole*

Research in Community and Mental Health, Vol 5. Edited by JAMES R. GRANTLY. London: Jai Press. 1985. Pp 319. £45.70.

This is the fifth volume in an annual series of collections of research in the epidemiology of mental health. It consists of eleven papers loosely grouped in four sections, although there is little of a common theme within, let alone between, the sections.

The concept of a volume which is in essence a hardback annual journal (albeit with papers which are somewhat extended compared with those in the average journal) seems a rather clumsy one, and I am not sure what it achieves. As one would imagine, the papers are variable in quality; some, e.g. Aneshensel's exposition of a model of the natural history of depressive symptoms, being of real interest. She postulates that the majority of the community consists of 'stayers' – defined in respect of depression either as constantly symptom-free (the majority) or constantly symptomatic (the minority) – and a significant minority consists of 'movers'. 'Movers' are sometimes symptomatic and sometimes asymptomatic, depending on other variables which she goes on to explain. This paper has a real relevance to daily clinical practice, but I found many of the other papers too remote from either the clinical practice or theoretical understanding. They concentrate on the relationship between psychiatric disorders and the social and cultural framework within which the disorders arise, and since the culture is a foreign one and is of little day-to-day relevance to a psychiatrist practising in the UK these are less easy to evaluate.

I do not think the book is worth the price for most private purchases.

GILLIAN WALDRON, *Consultant Psychiatrist, Tower Hamlets Health Authority, London*

Transsexualism and Sex Reassignment. Edited by WILLIAM A. W. WALTERS and MICHAEL W. ROSS. Oxford: Oxford University Press. 1986. Pp 191. £17.50.

This is a much better book than the last one on this topic I reviewed in this Journal (*Female-to-Male Transsexualism. Historical, Clinical and Theoretical Issues*, by Leslie Martin Lothsteing), and is a very interesting and useful book which should be read by people interested in the field. It is designed to be, and succeeds in being, appropriate for patients as well as clinicians. The book is from Australia, and includes contributions from physicians, surgeons, psychiatrists, and others concerned with the treatment of transsexual individuals in Melbourne. There is also a chapter written by a patient.

In a multi-author book on this topic there is inevitably some repetition in the chapters on gender identity, causes, and diagnosis, but I found this tolerable. I thought the more practical parts of the book, including details of gender reassignment surgery, welfare services, endocrine aspects, and voice, speech, and language considerations, to be best. The discussion of clinical aspects seemed a little incomplete and the consideration of the causes of transsexualism bedevilled by our profound ignorance in this field. On the whole these authors appear to favour a postnatal origin of transsexualism, but I found their presentation unconvincing. The problem is not with people who have multiple personality difficulties and perhaps other sexual problems as well as transsexual inclinations, for whom the sexual anomaly can be construed as part of a more general personality disturbance; the real difficulty is with transsexuals who I think in this book are said to have primary gender dysphoria who are frequently brought up in what, in ordinary terms, are normal families, have siblings without major personality problems, and are entirely without substantial psychosocial difficulties. It is difficult to see how an early family constellation so subtle as to be unnoticed on ordinary examination should serve to produce such a fundamental disturbance as gender dysphoria, unassociated with any other problem and not affecting other siblings exposed to the same parents.

This book seemed a bit muddled about homosexuality and about sexual object relations in transsexual people, but refreshingly sensible in relation to psychodynamic, feminist, and other current excesses.

I was unable to find any clear statement about the authors' indications for surgical intervention. This omission may have been deliberate in a book designed to be presentable to patients as well as to staff, but in many ways it is the crux of the matter. I applaud the courage of these Australian colleagues in taking seriously the need of a significant number of transsexual people for surgical intervention as part of a treatment programme, and note that the opportunities for the treatment of people with this problem in the UK are exceedingly limited and not as satisfactory as would be expected in a civilised country.

J. P. WATSON, *Professor of Psychiatry, United Medical and Dental Schools, Guy's Hospital, London*

Recovering From Rape. By LINDA E. LEDRAY. New York: Henry Holt. 1986. Pp 258. $9.95.

This is a readable, relatively short handbook for rape victims and their friends and family. The first and largest part takes the victim and the 'significant other' (by this is meant the victim's boyfriend or other family member) through the stages, both emotional and administrative, which will be experienced after rape. It covers dealing with the police and doctors, the stages of the emotional trauma, and advice on how to recuperate. Each chapter contains separate sections for the victim and the other and looks at the experience from both points of view.

The second part of the book, and in my opinion the least successful, looks very briefly at explanations for rape and ways in which rape might be prevented.

The first part contains no advice with which one would argue. It is clear, and well-illustrated with case histories. The major drawback is that it relates very much to the situation in the USA, and all the supplementary information at the back about Rape Crisis Centres is American. The brevity with which the issues in part two are covered contrasts with the leisurely and usefully repetitive style of the first part, and in my view sits rather uncomfortably alongside it.

With these minor reservations I would recommend this book to my patients.

JULIE A. HOLLYMAN, *Consultant Psychiatrist, Springfield Hospital, London*

The Overactive Child (Clinics in Developmental Medicine No. 97). Edited by ERIC A. TAYLOR. Oxford: Blackwell Scientific Publications. 1986. Pp 269. £16.00.

The idea that a particular child's problematic behaviour can be briefly described as hyperactive is remarkably tenacious. High levels of motor activity, apparent inattention, and a disorganised or unfocused quality of behaviour are prone to elicit the use of the term from clinicians. The extent to which hyperactivity (or similar labels) is an heuristic, reliable or valid term is assessed by this book, and suggestions as to management are made.

The five contributors have helped to create the mass of information which the book explains, and the results of their joint enterprises at the Institute of Psychiatry are quoted in detail in some chapters. Such data, taken with the recent findings of others, point towards various conclusions: a broad concept of hyperactivity has little or no scientific support, and relabelling the problem as attention deficit is unhelpful. The distinction between situation-specific and pervasive overactivity is important: a narrowly defined hyperkinetic syndrome with pervasive overactivity can be identified and multiple causation is the rule, and so assessment and interventions should be broadly based. Drugs are demonstrably powerful treatments.

Perhaps there is little that is new in this, but the value of the book is the way in which it examines habits of thought and assumptions made in conceptualisations. The scholarly approach leads to some sections being over-referenced, and there is some fence-sitting. This is a book which is mainly about being thoughtful. It cannot be definitive, given the state of things, but it is certainly important. Given the good reputation of the *Clinics in Developmental Medicine* series, it will be generally available in paediatric libraries; it ought to be in every psychiatric library, and the wise child psychiatrist will obtain his or her own copy.

PETER HILL, *Senior Lecturer in Child and Adolescent Psychiatry, St George's Hospital Medical School, London*

Differential Therapy of Depression: Possibilities and Limitations. Edited by H. HIPPIUS and N. MATUSSEK. Basel: S. Karger. 1986. Pp 248. SFr186, $103.50.

This book consists of the proceedings of a symposium held in 1984 in West Germany. The symposium was opened by the head of the Federal Health Department, a former psychiatrist, who spoke with specialist insight of the problems posed for his department by pressure for non-medical psychotherapy and against pharmacotherapy and ECT.

The clinical section opens with a review by Pichot examining the idea that response to amitriptyline, imipramine, MAOIs, and ECT can be predicted from symptomatology. He rightly concludes that the evidence is weak, but does not discuss the importance of spontaneous remission and placebo response which may have their own clinical predictors. Four papers follow on clinical experience with fluvoxamine in comparison with other antidepressants; none directly address the issue of whether fluvoxamine is an effective antidepressant. In three papers the numbers of patients studied are so low as to preclude any serious conclusion as to efficacy. A large international multi-centre trial has been carried out, but only the briefest reports have appeared in the literature. It is hard to understand why a full description of this trial does not appear in the volume, much of which concerns fluvoxamine. Wagner *et al* discuss the potentially important issue of the influence of concomitant medication on efficacy and side-effects in the multi-centre fluvoxamine trial, but details of the trial design and results are too brief to permit critical evaluation. They state that fluvoxamine was superior to placebo in the American centres but not in the European centres – the European results are then excluded!

Several contributions (those by Beckmann, Holsboer, Berger, and Fahndrich) examine whether MHPG or 5HIAA in biological fluids, the dexamethasone suppression test or sleep parameters predict antidepressant responses to drugs. The conclusions are generally disappointing, and patient numbers are again low. Some of the chapters are useful reviews. The most interesting new material was Berger's study of sleep in 66 patients with depression. Reduced REM latency and early morning wakening, putative markers of endogenous depression, were as frequent in the neurotic group as in the endogenous.

There are two detailed studies of fluvoxamine on heart and liver function in patients with existing cardiac or hepatic disease.

In the pharmacological section, chapters by Sulser and Brunello highlight the puzzling and, one suspects, important interactions between serotonin and noradrenergic synapses. Direct pharmacological actions of antidepressants are many and diverse, yet all share the ability to induce a delayed decrease in noradrenergic beta receptor function which appears to be mediated by 5HT neurones. This 5HT–beta receptor interaction may be involved in the ability of fluvoxamine to decrease beta-receptor numbers, yet other 5HT re-uptake blockers such as citalopram do not affect beta-receptor numbers.

The book concludes with chapters by Asberg on cerebrospinal fluid 5HIAA and by Matussek on attenuated growth hormone responses to clonidine – perhaps the most consistent biological abnormality associated with depression.

This book is very expensive, and is as much concerned with fluvoxamine as with the subject indicated by the title. Unfortunately the book is not informative on whether fluvoxamine is an effective antidepressant with particular indications. The chapters on the biology of depression and antidepressant drugs are the best, and some of the reviews are useful. However, much of this material is more cheaply available elsewhere.

J. F. W. Deakin, *Senior Lecturer in Psychiatry, University Hospital of South Manchester*

An Introduction to Marital Problems. By Jack Dominian. London: Fontana. 1986. Pp 206. £2.50.

Dominian states in his preface that his aim was to provide an introductory text for the caring professions, and in this he has succeeded admirably. The inexpensive paperback format makes it an ideal course text and I have no doubt it will be widely used for this purpose by the various disciplines involved in health care. Most psychiatric libraries will want a copy on their shelves and it is sufficiently inexpensive for trainees to buy their own.

The first two sections review briefly the contributions from sociological and psychological research and examine the 'family cycle' in the light of current social trends and current knowledge of psychosocial processes within the family. The last section briefly introduces current therapeutic models and practice.

It is a readable book, but is slightly marred by some carelessness in preparation – for example, there are some references in the text which are omitted from the reference list. The author relies heavily on stereotypical descriptions which, on occasion, strike an irritatingly naïve note. These are minor criticisms, and it is clear that this book is a valuable addition to the literature.

O. S. Frank, *Consultant Psychiatrist, Westminster Hospital, London*

Eating Disorders: Effective Care and Treatment. By Felix E. F. Larocca, St. Louis: Ishiyaku Euro-America. 1986. Pp 261. $27.50.

Larocca is the Medical Director of the BASH (Bulimia, Anorexia, Self Help Inc.) treatment and research centre for eating and mood disorders in St Louis, and he has aimed this book at students of the various professions involved in the care of patients with eating disorders.

One third of the book's 16 chapters are on bulimia; there is one on obesity and two on anorexia nervosa, and the remainder cover the anorexia–bulimia nervosa spectrum. These proportions probably accurately reflect current interest. The range of the chapters is unusual: there is one describing dissociative states, in which large extracts of therapeutic dialogue are transcribed, and another on "burnout and eating disorder therapists". The chapter on the oral manifestations of the eating disorders is excellent, with good illustrations and a text which clearly defines dental terminology. Several chapters address the diagnostic difficulties which presently perplex those working in this field; Munoz authoritatively advocates the 'British School' of diagnosis of the eating disorders, as proposed by Fairburn and Russell, and the European influence is continued in two further chapters.

Several aspects of the production of this book were disappointing: my review copy had the last two chapters superimposed on the ghosted print of earlier chapters; the tables and figures in Lacey's chapter were all bunched together at the end; and the absence of illustration in the chapter on the management of obesity resulted in this otherwise interesting chapter becoming incomprehensible when the surgical techniques were described. Throughout the book several references were incomplete.

Despite these caveats this book is of interest to those working within the field of eating disorders and for those in other fields wishing to keep abreast of the recent developments within this rapidly expanding field.

JANET TREASURE, *Research Worker, Institute of Psychiatry, London*

The Psychosomatic Approach: Contemporary Practice of Whole-person Care. Edited by MARGARET J. CHRISTIE and PETER G. MELLETT. Chichester: John Wiley & Sons. 1986. Pp 447. £32.50.

The practice of psychosomatic medicine forms a large part of the workload of a general hospital psychiatrist, and has developed into a sub-speciality of its own – liaison psychiatry – with its own group in the Royal College of Psychiatrists. It is an acceptable area for research both by physicians and psychiatrists, and books, journals, and papers abound in this field. It is against this background that this book has been published.

There are eight sections. The first, 'Contemporary psychosomatics', consists of two chapters: Christie introduces the other chapters in the book while giving a brief overview of the field, and Lipowski clarifies some of the concepts and terminology in this area. He traces the historical development of psychosomatic medicine and discusses semantic issues.

Section two, 'The beginnings of life', covers a number of areas of obstetric and paediatric care. There are chapters dealing with genetic counselling, attitudes to care in labour, and the admission of children to hospital. This is followed by a section on eating behaviour, with a presentation of a multifactorial model of obesity marrying behavioural, physiological, and neurochemical elements and a review of abnormal eating behaviour giving the current state of knowledge about anorexia nervosa and bulimia.

The section on cardiovascular disorders includes chapters on the management of hypertension and the alteration of type A behaviour in patients with coronary artery disease. This is followed by another section on the management of practical clinical areas, namely the treatment of chronic pain and the use of benzodiazepines in the relief of anxiety.

Section six concentrates on non-hospital medicine, with chapters on community medicine, training for primary health care, clinical psychology in general practice, and occupational health. This last chapter covers a number of important areas, including work stress, the relationship between alcohol and occupational factors, and accidents at work. The final chapter in this section deals with the treatment of the home environment of the schizophrenic patient.

The penultimate section deals with the psychosocial aspect of surgery and cancer, and the book ends with a discussion about terminal care.

The editors and authors have produced an excellent book which is clearly laid out, well-written, comprehensively referenced, and is informative and interesting. I would strongly recommend this book to all those involved in the field of health care, especially as it appertains to the interface between medicine and psychiatry.

JEREMY M. PFEFFER, *Consultant Psychiatrist, The London Hospital*

Forensic Psychiatry and Psychology. Edited by WILLIAM J. CURRAN, A. LOUIS MCGARRY and SALEEM A. SHAH. Beckenham: Quest Meridien. 1986. Pp 549. $61.25.

This book is in six parts and comprises 22 individual chapters. The authors are experts in various aspects of the interdisciplinary fields of forensic psychiatry and psychology. The main objective of the book is to improve the quality of forensic expertise within the two disciplines in the American judicial and correctional systems. The intention is to provide a comprehensive review of the current problems and issues in the field, ranging from knowledge about training programmes to increasing the effectiveness of testimonial, consultative, and treatment services.

Each of the six parts deal with a particular aspect of the subject matter. Part 1 focuses on professional training and ethical standards. Major training programmes in various parts of America are extensively discussed, and it is emphasised that these tend to lag behind increased clinical and research activities. An appreciation is made of the important roles of social workers and nursing staff in the forensic field. Part 2, the most detailed section of the book, deals with techniques and procedures for assessment and treatment. Part 3 covers family violence and child abuse, child custody issues, matrimonial problems, and juvenile delinquency. Part 4 contains four chapters on sexually related offences, and Part 5 deals with personality disorder and its legal implications. The final part covers some of the roles of mental health professionals in court proceedings and in prisons.

This book covers a range of topics related to the interface between psychology, psychiatry, and the law. The chapters are of consistently high quality, and although predominantly related to the American judicial system, this is undoubtedly a valuable contribution to forensic psychiatry and psychology literature. The combination of recent research findings, developments in clinical assessment and treatment techniques, and related professional issues are well presented and discussed. The major limitation is the absence of information on the role of the forensic expert in the sentencing stage in court proceedings. For the British psychiatrist and psychologist this seriously limits the value of this otherwise excellent book.

GISLI H. GUDJONSSON, *Senior Lecturer, Institute of Psychiatry, London*

The Penguin Dictionary of Psychology. By ARTHUR S. REBER. London: Penguin Books. 1985. Pp 848. £5.95, $7.95.

This is a remarkable achievement: a dictionary written by one man. It is nearly 1000 pages long and covers 17 000 terms, mainly from psychology and psychiatry but with considerable representation from medicine, philosophy, sociology, linguistics, pharmacology, anatomy, physiology, and an extraordinarily large collection of 'miscellaneous' terms, e.g. peg-word system, a mneumonic technique – the example was chosen by opening the book at random! One author has an advantage over many authors in being less dry and more interesting. On the other hand, as with Dr Johnson, the reader has to live with prejudices, such as a remarkably one-eyed view of parapsychology.

Dictionary and encyclopaedia reading has an intellectually and emotionally compulsive quality to it. Highly recommended both for libraries and the individual reader.

SIDNEY CROWN, *Consultant Psychiatrist, The London Hospital*

Inquiring Man: The Psychology of Personal Constructs (3rd edition). By D. BANNISTER and F. FRANSELLA. Beckenham, Kent: Croom Helm. 1986. Pp 194. £16.95 (hb), £7.95 (pb).

In the Preface the authors say that the third edition of this book, which first appeared in 1971, comes 30 years after Kelly's seminal work *The Psychology of Personal Constructs;* in those 30 years the theory has acquired proven status as a practical tool and as a rich source of new thinking. Over the same period they see that traditional psychology has undergone vital, if cautious, change.

The book retains its original character in that it is a highly readable exposition of the essential theoretical and philosophical bases of Personal Construct Theory. The distinctive Personal Construct approaches to such issues as personality, assessment, development, interpersonal relationships, therapy, and psychological abnormality are all clearly outlined. The coverage of the relevant literature is usefully updated.

As with earlier editions, the presentation relies heavily on a 'compare and contrast' method with other psychological models: behavioural, psychodynamic, psychometric, and cognitive. Unfortunately, in propounding the virtues of Personal Construct Theory the authors are unnecessarily disparaging of these different frameworks. They make no attempt to disguise their disdain for the limitations of these schools of thought.

It must be admitted that Bannister (now sadly deceased) and Fransella have done much by their own efforts to make Kelly's ideas as well known and widely used in this country as they are today. They have been for many years the standard bearers for the radical 'alternative' Personal Construct creed. Despite its at times irritatingly polemical style, the third edition of *Inquiring Man* is still a good, brief, and readable account of the main elements of Personal Construct Theory and can be recommended as such.

TOM TRAUER, *Top Grade Clinical Psychologist, King's College Hospital, London*

Movement Disorders. Edited by NANDKUMAR S. SHAH and ALEXANDER G. DONALD. New York: Plenum. 1986. Pp 406. £49.50.

This is a multi-author book of twenty chapters in five sections, dealing with Parkinson's disease, tardive dyskinesia, Tourette's syndrome, Huntington's disease, and drug-induced disorders. Although the emphasis is on biochemical aspects of each condition, clinical features are discussed in most of the chapters – sometimes in some detail.

The introduction by Stahl is a reasonably clear and up-to-date overview of movement disorders discussed in terms of the neurotransmitter inbalance hypothesis, with useful definitions of clinical terms and a comprehensive list of references to recent literature. The diagrams in this chapter, although somewhat overdone, are helpful in elucidating a difficult field. This chapter can be recommended to clinical neurologists and psychiatrists, as well as to the reader with a specialist interest in brain biochemistry.

The rest of the book is very disappointing. It has all the disadvantages of a multi-author volume without any of its advantages. With one or two exceptions, the chapters in each section are irritatingly repetitive, usually covering the same ground as the previous chapter without offering any new viewpoints. However, some excellent reviews stand out: for example, the chapter on Parkinson's disease by Lieberman & Goldstein is a good review of the biochemical background to the treatment of the illness, and the chapter on drug-induced disorders by Pearce & Clough is another good review from a clinical viewpoint.

The time is ripe for a book on movement disorders, that spans the overlapping interests of psychiatrists, neurologists, and biochemists, perhaps written from one or other point of view. Unfortunately, this book does not quite make it. As it stands, it may have some use as a source of references for specialist readers but is not really viable as a general book on movement disorders, nor as a volume that gives "a more complete understanding" of movement disorders in relation to one another – an aim identified by one of the editors in the preface to the book. It is hardly value for money at its present price.

SUMAN J. M. FERNANDO, *Consultant Psychiatrist, Chase Farm Hospital, Middlesex*

Body and Mind in Old Age and Decay (Sinai Papers number 3). By A. SUNIER. The Netherlands: Van Gorcum. 1986. Pp 307. Dfl. 49.50.

This book is part of a series of three; Sunier is part author of the preceeding works on the Holocaust and problem patients. The title itself is chilling. Before we reach the list of contents, we are offered "guidance": the need becomes increasingly apparent with each page.

Despite the obviously diligent research and effort involved in producing this book, it reads like an inadequate translation of an over-inclusive work. There are frequent irritating spelling errors, and an awkward use of language which at times verges on incoherence. The style is idiosyncratic, dogmatic, and occasionally impenetrable.

What of the contents? The book is divided into two parts. The first two-thirds comprises an unhappy mixture of philosophy and the past findings in dementia. These are loosely woven together. In the second part, the author turns to a group of 70 of his own patients and presents a series of data with an attempt to relate the preceeding discussion with his results, setting himself the immense task of studying "the roots of our knowledge about the body of human subjects", including "molecular biology, cognitive psychology, sociology, and psychoanalysis", to examine the possible causation of dementia. "Our knowledge of the living system" should, he says, be expressed in "a homologous language of growth and decay of structures". There must be a simpler way...

The pathological process of dementia and the resulting adaptation will find expression in a multitude of patterns: so much is evident. Sadly, Sunier, despite his altruistic objectives, takes us little further.

D. KINGHAM, *Senior Psychiatric Registrar, Barrow Hospital, Bristol*

Psychotherapy and the Paranoid Process. By W. W. MEISSNER. New Jersey: Jason Aronson. 1986. Pp 433. $40.00.

The significance of Meissner's contribution lies in his grasp of the underlying structure of paranoid states. Here he explores the therapeutic aspects of the process that he first described in his earlier book, published in 1978. His model of paranoia rests fundamentally on what he describes as the introjective structure, consisting of two polar configurations of both aggression and narcissism. The aggression polarity involves a weak, *victim* introject and a strong, controlling, *victimiser* introject. The narcissistic configuration involves an inadequate, inferior pole, associated with feelings of shame and humiliation, and a grandiose, superior pole. These internalised objects can at different times be felt to be part of the self or part of the object. They are derived from actual early interactions with the parents, and incorporate aspects of the parents' own personality organisation – in Meissner's experience, parents of the paranoid person have often been themselves caught up in pathological aggressor-versus-victim and superior-versus-inferior patterns. He finds that paranoid patients are often very threatened by any attempt to confront them with their rage towards their parents; by projecting negative aspects onto other figures the patient attempts to preserve a good relationship with the primary object, the parent. Usually the paranoid patient is more subjectively in touch with the inferior victim poles, but the opposite poles are always active in repressed or projected form. Depressive patients may also show similar patterns of identifying with the victim introject, but show less projection than the paranoid patient.

Meissner discusses in detail the structure of the therapeutic process. Stage one is to establish the therapeutic alliance through sensitive listening, being particularly attentive to the patient's individuality rather than preoccupied with the therapist's theory. The next stage involves defining the projective system *before* challenging it – this is done through listening to the patient's account of his experience of himself and the world in which he lives, getting as much detail as possible about perceptions and feelings. Then the projective system can be challenged, first by tagging feelings – by adding remarks like "that seems to be how you feel" in order to help distinguish between feelings and reality; initially the patient will offer explanations and conclusions as if they were accepted fact. At this point there can be the crucial shift in focus from the projective system to the introjects. While initially the patient will more easily be in touch with the inferior victim introject, he must gradually be made aware of the superior, destructive, aggressive pole. The derivation of the introjects from actual early interactions can then be clarified. However, the introjective configuration is not easily given up, because it provides the essential core around which the sense of self and other are organised. This motivation must be explored, and the patient then needs to mourn the loss of the internal attachments, which then ushers in a phase of increased transference dependence.

Meissner explores the paranoid process in psychotic, borderline, and narcissistic states, and its role in adolescence, in addiction, in suicide, and in the ageing process. His discussion is scholarly and comprehensive. My one criticism is that in his efforts to be thorough he does not always sufficiently highlight foreground against background. The book is likely to be valuable to all dynamically-oriented psychiatrists and therapists.

PHILIP MOLLON, *Psychotherapist, Dryden Road Hospital, Tyne and Wear*

Current Psychiatric Therapies 1986, Vol. 23. Edited by JULES H. MASSERMAN. Orlando, Florida: Grune & Stratton. 1986. Pp 308. $63.00.

Jules Masserman is a name to be reckoned with in American psychiatry. He is now 79, and this book is the last in the *Current Psychiatric Therapy* series that he intends to edit. I am not familiar with previous volumes in the series, but the last one was described in this journal as making "no scientific case for progress" but as having "some curiosity value". The same could be said of this volume. Seven past Presidents (including the editor himself) and the current President of the American Psychiatric Association have contributed reviews of various fields, but they are conspicuously lacking in new findings or new research. The chapter by Freedman, on the psychopharmacology and psychotherapy of anxiety, is unusual in the number of papers cited, but none are more recent than 1979.

With the exception of a brief chapter by Kolb on the treatment of chronic post-traumatic stress disorders, the contributions about current psychiatric practice of potential application in this country are more concerned with their American practitioners than the treatment. Topics covered include proposals to fix treatment charges according to diagnostic category (discussed by both Talbott and Beigel), women's issues (Nadelson & Notman), and the ever-widening horizons of the malpractice lawyer (two chilling chapters, one by Slawson demonstrating the increasing legal responsibility of the American clinician for the actions of his or her patient, and the other, by Charles, documenting the distress caused to the clinician by a malpractice suit).

A large section of the book is given over to short accounts of their work by psychiatrists from the Bekhterev Psychoneurological Institute, Leningrad. The editor states that "Soviet psychiatrists are as scientifically progressive, ethical, and humanitarian as we try to be in our country", but the impression from this book is that they have become excessively preoccupied by social conformity. The spectre of political schizophrenia haunts the chapters and it is a shock to read that Ponomareff considers that the Institute of Clinical Psychiatry in Moscow is even more over-inclusive in its use of this abused diagnosis.

The remainder of the book is mainly concerned with spiritual matters, although there is one lively description of the ceremony preceding the May and Baker Plenary Address to the World Congress of Community Psychiatry, which involved a Swami and "lovely sari-clad maidens" whom the editor was required to symbolically deflower (his association, not mine).

Potential readers should bear in mind that the editor has interpreted his title, *Current Psychiatric Therapies*, as the Russians interpret schizophrenia and the World Congress of Community Psychiatry interprets inaugural ceremonies: rather loosely.

DIGBY TANTAM, *Senior Lecturer and Honorary Consultant Psychiatrist, The University Hospital of South Manchester*

Psychology and Medical Care (2nd ed). By GERRY KENT and MARY DALGLIESH. Eastbourne: Ballière Tindall. 1986. Pp 342. £7.50.

This book has thirteen main chapters divided into three sections: psychological processes, human development, and doctor-patient communication. The authors are to be congratulated, because they have produced a book which is both factually rich and readable. I liked the presentation of psychotherapeutic theories in the first part, which also contains chapters on memory, intelligence, and the sensory apparatus. Human development is covered in the biological life cycle framework.

There is more than enough here for the undergraduate, and it provides a useful revision for the general professional trainee. The references and suggested further reading give up-to-date information for more detailed research into the literature.

WILLIAM HUGHES, *Consultant Psychiatrist, United Norwich Hospitals*

The Twenty-Minute Hour: A Guide to Brief Psychotherapy for the Physician. By PIETRO CASTELNUOVO-TEDESCO. Washington, DC: American Psychiatric Press. 1986. Pp 172. $12.00.

This is a sensible book which tries to persuade the general physician to listen to his patient and to offer what the author calls "supportive psychotherapy". What the author tries to do is show the physician (by which he means an American general practitioner in private practice) that the work of psychotherapy can easily be fitted into the framework of the usual routine. The level of undertaking is really counselling (Cawley's level 2). He concentrates on the limitations of what can reasonably be achieved by a "non-expert", at consultation and in a subsequent series of ten 20-minute interviews. This may seem luxurious besides the 6 minutes suggested by Balint & Norrell, although hardly enough for most out-patients. Castelnuovo-Tedesco is clear about whom it is reasonable to attempt to help within this time span, what should definitely not be attempted, and on interventive strategies. He is helpful about how to limit the focus to what he calls the present and the future and how to appropriately blinker oneself to the past and the transference.

I think that what is offered is probably less sophisticated than what is available to general practitioners in this country now. It does seem appropriate, however, for physicians and surgeons who might be interested in helping their patients with the emotional conflicts that may manifest themselves as physical disease. (It is heartening to read of a man being positively investigated for the emotional disturbance that might have lead to the perforation of his peptic ulcer!). It would be nice to think of it also in the hands of psychiatrists whose training seems to make it so hard for them to hear any meaning in what their patients say.

I doubt that this book will satisfy the 'converted' or that it will find its way into the hands of the 'unconverted'. I think it might be helpful, however, as a teaching adjunct to those involved in liaison work with physicians, surgeons, and psychiatrists.

R. R. N. CARVALHO, *Consultant Psychotherapist, St Mary's Hospital, London*

Suicide and Depression Among Adolescents and Young Adults. Edited by GERALD L. KLERMAN. New York: American Psychiatric Press. 1986. Pp 383. $24.95.

This book is based on a conference held in 1982 on preventive aspects of suicide and depression among adolescents and young adults. Epidemiological and aetiological factors of suicide are identified and different means of intervention considered, and the possibilities for the prevention of mental illness in this age group discussed. It is sometimes difficult to see any clear order in the sequence of chapters, with different subjects side by side. One chapter is not specifically concerned with adolescence, but with the statistical techniques that can be used to investigate the heritability of major depressive disorders. In contrast is a painful insight into the inner world of Vivienne, an unfortunate victim of suicide at the age of 13, who had written her troubled thoughts in letters and a diary.

Several chapters report on current research. However, the lack of data make them unsatisfactory. There are some useful literature reviews, in particular a clear account of research on personality and psychosocial factors in suicide and depression in adolescence.

The book forms an interesting and thought-provoking collection for anyone having contact with adolescents.

JOYCE ALMEIDA, *Registrar in Child Psychiatry, The London Hospital*

Intensive Family Therapy: Theoretical and Practical Aspects. Edited by IVAN BOSZORMENYI-NAGY and JAMES L. FRAMO. New York: Brunner/Mazel. 1985. Pp 507. $35.00.

In common with most child psychiatrists I practice, and indeed enjoy practicing, family therapy. Unlike many, however, I do not read a great deal of the family therapy literature. I find that too often I become bogged down in a mass of neologisms, or completely unreadable accounts of theoretical models. I have always felt guilty about this, and have wondered whether I might become a better therapist if I read more! I was therefore pleased to receive this book and decided to make an effort to read it through, mainly as an exercise in self-education with a review as a secondary goal. I threw myself into the first chapter and began reading. As I read on I became puzzled by the need of several authors to justify why they practice family therapy at all, as opposed to individual psychoanalytic therapy. I noticed with growing puzzlement that none of the references were more recent than 1963. At that point I turned to the publication details and discovered that this is a reprint of a book originally published in 1965. No reason is given by the publishers or the editors as to why they thought a reprint would be useful 21 years later.

I assume that this is regarded as a seminal work in the development of psychoanalytically-based family therapy. In 1965 it may well have been a most important book, helping those who were treating patients with schizophrenia or other serious psychiatric illnesses through individual psychotherapy to look at the social environment of their patients. With all the developments in family therapy since then and with the growing body of knowledge of the effects of family interactions on the course of psychiatric illnesses, this book can only be regarded as a historical curiosity. I cannot see how this reprint will be of any use other than as an indication of the thinking in the 1950s and 60s of certain analysts who were beginning to transfer their interests from the inner representation of the family to its existence in the real world.

STEPHEN N. WOLKIND, *Consultant Psychiatrist, Maudsley Hospital, London*

Essential Papers on Narcissism. Edited by ANDREW P. MORRISON. New York: New York University Press. 1986. Pp 491. $50.00 (hb), $22.50 (pb).

Why is narcissism important? Kernberg provides an answer: he states that there exists a group of patients whom we might consider as a pure culture of the pathological development of narcissism. These patients on the surface do not present serious disturbed behaviour. He goes on to outline the features of these patients, concluding that they are a subgroup of the borderline group of personality disorders. The importance of identifying this particular group is that they may regress to a psychotic level of functioning, which can come as a surprise if the professionals working with them are not alert to the constellation of symptoms and their significance.

This book draws together various views on narcissism, starting with Freud's classic paper 'On narcissism'.

This is perhaps the most difficult, because of what was for Freud an unusual lack of clarity. But perhaps it is this confusion that enabled subsequent workers to take ideas from it and develop them in the light of their clinical experience. It is a pity the papers are not dated; one assumes they are in chronological order, but this may not be so. Pulver's chapter contains a clear explanation of the concept and the problems of the use of the same word to convey different meanings.

Cooper discusses recent developments, and Kohut's paper is of interest, particularly where he suggests that healthy narcissism may become transformed into humour, creativity, empathy, and wisdom. Kernberg's 'Factors in the psychoanalytic treatment of narcissistic personalities' is a must, and Alice Miller's paper is of interest to general psychiatrists trying to understand depressed patients. Morrison's paper on shame uses Kohut's formulation of the defects in the early development of the structure of the self leading to narcissism, and suggests that shame reflects self-depletion in such patients, and is a painful experience which needs recognition. It is not sufficient to treat the ensuing depression.

There are three papers on treatment: psychoanalysis, supportive psychotherapy, and general psychiatric management. Ideas vary on how to deal with dependence, manipulative behaviour, paranoia, passive-aggressive behaviour, or antisocial behaviour. What is agreed is that if there is to be a fundamental restructuring of these patients, it is long, slow, difficult work.

Meissner's paper attempts to integrate the theoretical problems of narcissism with the problems encountered in treatment. Bromberg's paper may become a classic. It describes the essence of narcissism as currently understood and the pain of the 'mirror and the mask' for patients and those trying to treat them; however, it concludes by offering some hope.

JUDITH TROWELL, *Consultant Psychiatrist, Tavistock Clinic*

Community Surveys of Psychiatric Disorders. Edited by N. N. WEISSMAN, J. K. MYERS and C. E. ROSS. New Brunswick: Rutgers University Press. 1986. Pp 544. $60.00 (hb), $25.00 (pb).

As MacMahon pointed out, 'No problem of definition in medicine is more baffling than that of defining mental illness". One might add that the validity of this assertion is nowhere more apparent than in studies of psychiatric morbidity conducted in community settings. Moreover, the notion of community raises perplexing questions. The frustrations encountered in the struggle for definition, representative sampling, and the specification of appropriate dependent and independent variables, and the efforts to achieve reliable and valid results since Jarvis first attempted to determine the frequency of idiocy and insanity in the mid-19th century, are thoroughly exposed here in 19 chapters by 30 eminent authors. The Midtown Manhatten, Stirling County, New Haven, and NINH Epidemiologic Catchment Area Program studies are dealt with, along with several European surveys. In addition to the editors, the contributors include Dohrenwend, Dunham, Endicott, Helgason, Hollingshead, Jablensky, Regier, Robins, and Wing. Inevitably, the editors concentrate on work done in the USA; little attention is given to the British experience. For example, only three surveys from a general practice setting are referred to, and these only in a Table.

GREG WILKINSON, *Senior Lecturer, Institute of Psychiatry, London*

A Matter of Interest: Clinical Notes of A Psycho-Analyst in General Practice. (The Roland Harris Trust Library Monograph Series No. 2). By KENNETH SANDERS. Strathtay: Clunie Press. 1986. Pp 109. £6.50.

This compassionate little book lives through its case material. It is in truth a matter of interest for GPs rather than in any sense a textbook. It serves as a reminder of the potential available to bring consultations to life by acknowledging the internal world of the patient via 'restrained investigation'. This, if not actually solving the patient's problem, promotes a feeling of warmth and concern, and certainly a satisfying deepening of interest.

Sanders' practice is affectionately placed historically, and some characters introduced in rather an *Under Milk Wood* sort of way. There is poetry in the case cameos: for instance, the Russian "Lady with the miniatures" and her deathly romance with her impotent, alcoholic rear-gunner, Charlie.

Quite a lot of psychoanalytic theory is taken for granted, and sometimes a surprising interpretation stands out rather starkly, with no obvious lesson to be drawn. However, there are lessons in the chapter on Bion: we are led to the idea of rescue rather than action, and the three-dimensional notion of containment.

Throughout, Sanders wisely reminds us of the importance of projection of anxieties from childhood deprivation. There is the delightful case of Sally and her grandmother, where the original encounter was a request for a sedative. The patient was unaware of the complexity of the problem, and the grandmother bewildered, but with the help of Sally's pictures the doctor interpreted safely and helpfully. Could we GPs do more of this?

HILARY GRAHAM, *General Practitioner, Highgate Group Practice, London*

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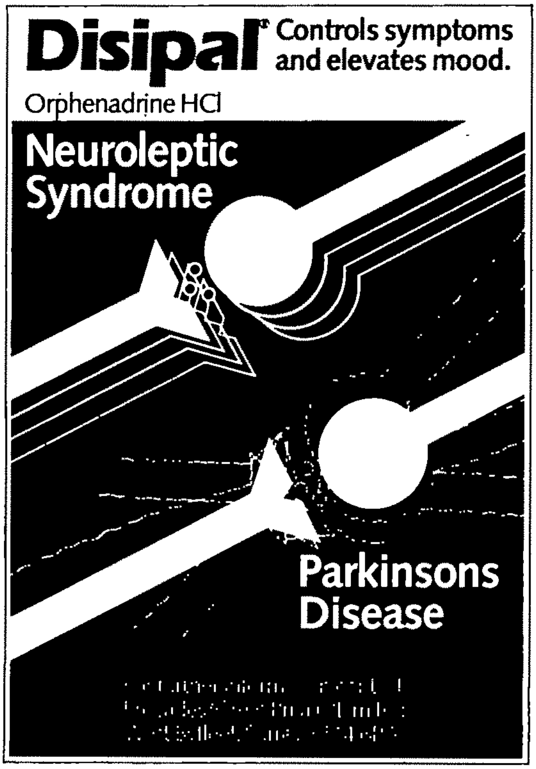

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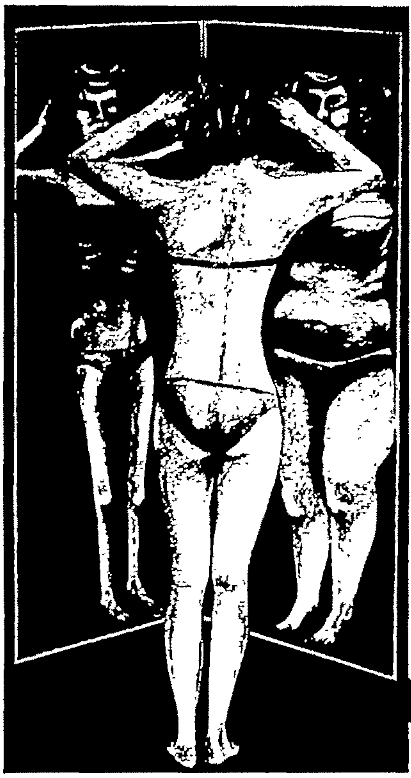

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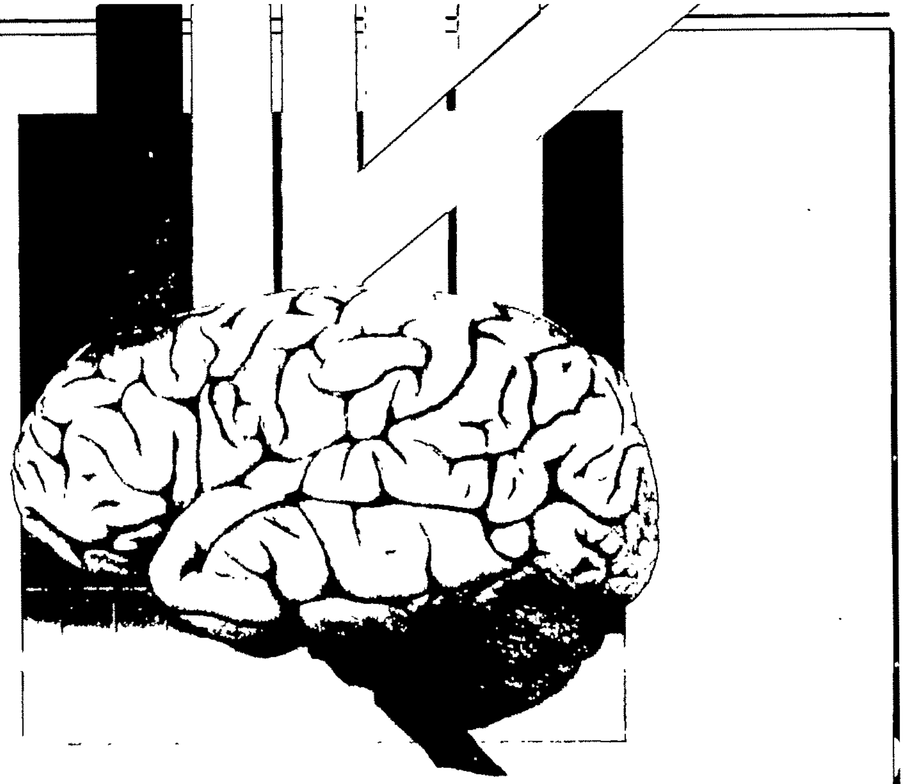

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1984,69: (Suppl 311) 7–30 2 Edwards J G et al Br J Psychiatry 1980; 137. 522–529. 3. Munk-Anderson E Acta Psychiatr Scand 1984, 69 (Suppl 311) 31–41 4 Schiff A A
Med J. 1985, 291 (6506) 1424

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Review Article

Depressive Disorder in Primary Care

C. V. R. BLACKER and A. W. CLARE

Since the pioneering study of psychiatric morbidity in primary care by Shepherd *et al* in 1966, it has become increasingly apparent that a substantial proportion (between 20% and 25%) of patients consulting their GP are suffering from some form of psychiatric disturbance (Goldberg & Blackwell, 1970; Hoeper *et al*, 1979). The composition of this psychiatric morbidity has been shown to be almost wholly affective in nature and largely mild in degree. In their important review Jenkins & Shepherd (1983) recently summarised the now extensive findings relating to overall minor psychiatric morbidity in primary care. However, recent collaborative studies between psychiatrists and GPs have identified that within this dilute pool of minor disorders, lurks a significant but poorly served population of patients suffering from depressive disorders which are by no means minor in degree. A number of crucial issues regarding this depression in primary care emerge which the present paper aims to review. In particular, how common is it, and how severe? How does it present and what, if any, are its special characteristics? What is the precise relationship between depressive symptoms and depressive illness presenting to the GP and what is the relationship between physical illness and depression? And finally, what is the course and outcome of depression in this setting and what are the indications for and effect of treatment?

The extent of the problem

As early as 1947, Watts drew attention to the problem of depression in general practice by showing that 'endogenous depression' was a much commoner condition in that setting than was generally appreciated. Since then numerous studies have attempted to measure and describe this phenomenon but the problems surrounding their definitions of 'caseness' (i.e. at what point a set of complaints becomes serious enough to be considered epidemiologically, sociologically, or clinically significant) have made their interpretation difficult. A popular solution to this problem has been simply to record the actual number of depressive symptoms. Thus Shepherd *et al* (1966) found that, within a range of London practices, 7% of men and 13% of women "usually felt unhappy", 2% and 6% respectively "felt so unhappy that they wished they were dead", 1.8% and 2.5% "always felt miserable and blue", and 2.7% males and 4.4% females "felt that life was entirely hopeless". Other studies of depressive symptoms are summarised in Table I. As can be seen, they demonstrate wide discrepancies between rates for 'depression' so defined, the reasons for which include differences in the way symptoms were elicited, the choice of instrument used to measure them, and in the cut-off scores by which 'caseness' was defined.

As the rates in Table I show, the experience of depression itself is a common one in general practice. Yet examination of such patients shows that only a small proportion of them fulfil diagnostic criteria for depressive disorder, i.e. have a pervasive disturbance of mood, or demonstrable impairment, or any of the constellation of 'associated symptoms' with which the notion of the depressive syndrome is surrounded. The issue of 'caseness' thus moves up from the mere quantification of symptoms to consider their type, severity, and statistical relationship as well as other measures such as degree of associated impairment and help-seeking behaviour as embodied in modern operational diagnostic criteria. In case this is thought to cover unnecessarily familiar territory for readers, it is helpful to recall that GPs are confronted by a

TABLE I
Prevalence of depressive symptoms

Name of study	*n*	*Percentage of patients scoring above shown cut-off score*
Salkind (1969)	80	48 (BDI>11)
		25 (BDI>25)
Burns *et al* (1977)	?	15.6 (SDS>50)
Wright *et al* (1980)	199	41 (SDS>50)
Nielsen & Williams (1980)	526	19.8 (BDI>10)
		3 (BDI>20)
Zung *et al* (1983)	1086	13.2 (SDS>55)
Hankin & Locke (1983)	1921	21 (CES-D>16)

BDI = Beck Depression Inventory, SDS = Zung depression scale, CES-D = Centre for epidemiological studies depressive symptom scale.

TABLE II
Prevalence of 'depressive illness'

Study	*Sample*	*Diagnostic category*	*Rates*
Barber (1981)	Nine Glasgow GPs 1 month	101 new cases of 'depressive' illness'	Incidence = 3.7 cases per GP per month
Porter (1970)	Single practice 2.5 years	93 cases of 'depressive illness'	Prevalence per consulting patients = 1.7% men = 7.8% women Annual incidence per 1000 at risk = 6.5 men = 29.3 women
Johnson & Mellor (1977)	Five Manchester practices. Consecutive cases. Period of sampling not known.	119 new and 88 'in-treatment' cases of 'depressive illness'. Diagnosis confirmed by psychiatrist interview	Prevalence per consulting patients = 3.7% (both sexes)

bewildering range and variety of continuously distributed emotional states which often defy the neat categorisation laid down by existing systems of diagnosis and classification. Current classifactory distinctions between one affective disorder and the next are largely arbitrary and derived from experience with psychiatric-service populations which (because of the peculiar nature of the referral system) are no longer representative of their disease-population of origin. Thus traditional concepts such as the 'endogeneous/neurotic' dichotomy, used to categorise depressive disorder and acquired by GPs in their medical training, are less helpful than they might be. It is not surprising therefore that many GPs have their own ways of defining caseness and labelling disorder (Clare, 1982). In particular, many GPs resort to the notion of a disorder's 'understandability' in defining caseness. However, this creates its own difficulties, not the least of which is that few depressions in any setting are truly 'endogenous'; the majority appear to have arisen in the context of, if not actually caused by, readily discernable life events, illnesses, or social and relationship difficulties of one kind or another. Moreover, as shown in Jenkin's & Shepherd's review, the role these social factors play in the context of depression is complex and very poorly understood. Therefore, the equation 'understandable = appropriate/normal' commonly used by GPs to define caseness in affective disorder is misleading and may well be responsible for much of the apparent diagnostic and therapeutic inertia which seems to afflict them in the management of depressed patients. From the GPs point of view the importance of these arguments is that a more satisfactory definition of caseness will give greater weight to the *form* and *degree* of the ensuing depressive reaction and less to the circumstances against which it arose. Fortunately the operational criteria by which depressive *disorder* is distinguished from all other manifestations of 'depression' have largely been those chosen for epidemiological studies of depression in primary care, the reliability of which have also been greatly enhanced by the more rigorous handling of 'caseness' provided by structured psychiatric interview and diagnostic instruments such as the Present State Examination (PSE: Wing *et al*, 1974) and the Research Diagnostic Criteria (RDC: Spitzer *et al*, 1978). The efficacy of such instruments in controlling for the vagaries of caseness-definition are demonstrated by the greater concordance between the findings from studies which employ them. Nevertheless, such instruments are not infallible and they fail to account realistically for important axes such as personality or physical illness which exert a powerful influence on a clinician's normal case-making decision. The issue of what constitutes a 'case' of psychiatric disorder in primary care is thus still a matter of debate. We therefore turn to consider the prevalence of depressive *disorder*. Studies drawing their data from a retrospective examination of GP case records generally report low prevalence rates for depressive disorder presumably because many cases fail to be detected by GPs (see below). Furthermore not all identified cases are recorded in casenotes, and even when they are, GPs do not always award the patient a specific diagnostic label, or prefer to use alternative labels (e.g. 'anxiety') to describe them (Clare, 1982). Such studies therefore yield low 1-year period prevalence rates for depressive disorder in the range 0.2%–1.5% (percentage at risk, both sexes, diagnoses matched with ICD and DSM) (Watts *et al*, 1964; Wing *et al*, 1967; Bebbington, 1978). Crombie (1974), reporting on the Second National Morbidity Survey of 1970/71, recorded that 3.1% of consulting patients in the UK were suffering from neurotic depression giving a 1-year consultation rate of 35.5/1000 at risk (a substantial increase on the previous survey). The reasons for this increase are unclear but may include a true increase in prevalence during that time, or a change in public attitudes making it more likely that depressed people will seek help from GPs, or that GPs have greatly increased their diagnostic vigilance and recording skills. Other studies of depression, not dependent upon the retrospective examination of case records are summarised in Table II.

More reliable studies of depressive disorder which bypass the problems of case-detection by GPs include those of Wright *et al* (1980) and Nielsen & Williams (1980). In both studies patients screened for depressive symptoms (see Table I) were subjected to a clinical diagnostic interview with one of several psychiatrists. On the basis of these interviews Wright returned a very high rate for RDC depressive disorder of 17% of consulting patients. However, only 32% of those eligible for diagnostic interview agreed to receive it and only two of these were men. Nielsen found that 10% of consulting patients fulfilled a diagnosis of 'Depressive Neurosis' (DSM-II) whilst on Feighner criteria 6.8% were judged to have primary depression and 4.9% secondary depression.

Using the structured psychiatric interview and diagnostic techniques referred to earlier, Casey *et al* (1984) made a study of 'conspicuous' psychiatric morbidity referred by GPs from a single centre over the course of a year. On the basis of diagnostic interviews using the PSE the authors report that 'nearly half' of the sample (approximately 3.3% of consulting patients) had depressive disorder. Comparison with other studies suggests that this figure is on the low side, the probable reason for which is the well-known problem of non-recognition of psychiatric disorder by GPs. Casey's figure for overall psychiatric morbidity, too, is on the low side (7% of consulting patients compared to the 15% plus reported by Shepherd and others) giving further grounds for suspecting that many patients went unrecognised by the diagnosing GPs.

Hoeper *et al* (1979), using a familiar two-stage design (the GHQ and the Schizophrenia and Affective Disorder Schedule—Lifetime Version, Endicott & Spitzer, 1979), returned point prevalence rates for RDC depressive disorder out of 1972 consulting patients as follows: major depressive disorder (5.6%), intermittent depressive disorder (5%), and minor depressive disorder (3.4%). It should be born in mind that these three categories are not mutually exclusive since patients may have more than one disorder simultaneously. These prevalence rates are, however, adjusted for the 30% of cases which, on interview, the GHQ was found to have failed to detect. In this study patient recruitment was low: 15% declined to complete a GHQ and 30% of GHQ-positive patients declined to be interviewed. The importance of obtaining high recruitment in epidemiological studies of this sort concerns the nature of the patients so missed. It is our experience that primary care patients who are most reluctant to participate in questionnaires and interviews are very likely to be suffering from some form of emotional distress (e.g. bereavement) or psychiatric disorder (typically alcoholism, personality disorder, and major depression). Their non-inclusion would therefore exert a powerful influence upon prevalence rates. Other criticisms of Hoeper's study include the use of social workers to make the diagnostic interviews and the shortness and timing of their 3-month sampling frame (January and March—a time of year notorious for its association with high rates of physical illness and general distress). Short sampling-frame studies fail to account for the well-known seasonal fluctuation in depression, and also produce disproportionately large numbers of chronic cases (and those who, because of the presence of other factors—such as personality, loneliness, or physical illness—tend to consult more frequently). Not surprisingly therefore, 80% of Hoeper's sample were later found to be suffering from chronic disorders.

Thus the definitive study of prevalence of depressive disorder in primary care remains to be done although we hope shortly to publish our own findings in which many of these methodological problems were taken into account (Blacker & Clare, in prep.). In the meantime, one may tentatively conclude that point prevalence rates for RDC Major and Minor depressive disorder of approximately 5% each in consulting patients are not far from the mark. There are suggestions from other investigators that this figure is similar in other centres despite their differences in geographical location. The importance of this finding is that depressive disorder is therefore one of the more common clinical problems encountered by GPs in the course of their work, the severity and characteristics of which we shall now consider.

Severity of depression in general practice

It is frequently stated that primary care depressives are milder, more 'neurotic', more 'reactive', and less 'biological' than those found in psychiatric settings. Certainly such patients are very well represented in primary care but the question remains whether depressions in this setting are always of this type—or whether cases of more severe depressive illness also exist but currently escape referral to psychiatric services? Of course, the proportion of severe-to-mild disorders in a particular practice will probably be influenced by its geographical location because of the effects of demographic and social variables upon prevalence (Shepherd *et al*, 1966). Individual GPs bias and accuracy, therapeutic orientation and referral attitudes will also exert an effect upon the proportion of cases deemed 'severe'. The traditional view of a positive relationship between severity and likelihood of referral to psychiatric services is largely derived from experience with patients suffering from psychotic illnesses. The situation in depression is unclear. A number of authors have reported a marked differential of severity between psychiatric-service and general practice depressives. In comparison with depressed psychiatric in-patients, Davies & Blakshi (1973), Johnson (1973, 1974), and Fahy (1974) found that primary care depressives were milder and borderline in quality. Sireling *et al* (1985*b*) compared 'GP depressives' with psychiatric out-patient depressives selected for inclusion in a drug trial and concluded that 'general practice depressives were considerably less severely ill than psychiatric out-patients, with fewer depressive symptoms and shorter illnesses, as well as less primary and less endogenous depressions'. However, these conclusions are partially forced upon them by their sampling: depressed patients already receiving antidepressants at the time of entry, or who were felt to be so depressed as to require (or have recently required) a psychiatric opinion, were excluded. Thus, in this otherwise valuable study, many of the more severely depressed would have been removed before the essential comparisons were made. In addition, one-fifth of the so-called 'general practice depressives' did not have depression at all but were found to be suffering from anxiety states or other conditions. Exceptionally severe cases of depression are likely to gravitate quickly into psychiatric care but it is important to bear in mind that depressed patients found within psychiatric services represent a distillation or 'creaming off' of cases from a comparatively dilute pool of disorders in the community. Thus, if taken as a whole, these two populations will always demonstrate a differential of severity due more to the effects of sample composition than anything else. After controlling for this, Brown *et al* (1985) found considerable overlap between the severity of depressive disorders in psychiatric out-patients and in the community. What distinguished the two, it seemed, was not the severity of disorder but the way symptoms were expressed. Their findings support the notion that GPs tend to make a decision to refer depressed patients not so much on the basis of the illness severity but on the presence of additional 'complicating' factors such as personality difficulties, alcohol abuse, non-response to treatment, or threatened self-harm. Fahy (1974), too, found that severity of depression was not a major factor in a GPs decision to refer. Studying the question of severity of general practice depression by comparison with psychiatric-service patients may not, therefore, be the best way to proceed. The additional complicating factors, which referred patients appear to have, make independent contributions to the measurement of disorder severity and so confuse the picture. The alternative solution is to obtain a large and representative sample of general practice depressives and make a within-group study of depression severity using a hierarchy of caseness as defined by various diagnostic and classification systems. There are, of course, ways of defining severity other than the type number and statistical relationship of individual symptoms. In clinical practice the degree of associated social impairment or disability is often a decisive factor although this invites confusion with other variables such as personality and social support. In a large sample of over 200 consecutive cases of depressive disorder, Johnson & Mellor (1977) found that half of these general practice patients were unable to continue with their normal lives. They concluded that the traditional view of general practice depression as a mild and self-limiting condition was therefore mistaken.

Only a proportion, probably less than 10%, of depressed patients in primary care are referred to psychiatric services. As will be shown later only a small proportion currently receive what would be regarded as an adequate course of treatment. Quite what happens to the depressed patients who thus escape referral and who therefore remain 'locked' and untreated within the community is not known. The worrying statistics with regard to GPs' contacts with suicidal patients (Barraclough *et al*, 1974), chronicity of disorder (see below), and the emerging association between depression and alcohol and drug dependency in the community (Brown *et al*, 1985) suggest that the current complacency with regard to the severity of GP depression should be resisted.

Sociodemographic factors and depression in general practice

The importance of sociodemographic variables in the study of any disorder lies in the identification of groups of patients who may be considered to be 'at risk'. The impression gained from exposure to general practice is that young, working-class, women are responsible for the lion's share of the community's depression. However, this is largely due to the effects of consultation processes since young women are over-represented in this setting. Tischler *et al* (1975*a,b*) reported that single, unemployed people living on their own over-used GP services when compared with other groups in the general population from which they were drawn.

Previous studies of depressed patients in general practice have reported a variety of often conflicting findings whose interpretation is difficult. Often, little effort is made to define 'caseness' and the usual inclusion of chronic relapsing disorders (which constitute a substantial proportion of cases seen in this setting) may lead to biases in reported sociodemographic findings (e.g. civil status and employment) because of the negative effects such disorders have upon relationships. Studies of single practices or those drawn from similar areas will generally report sociodemographic associations which reflect the composition of the populations they serve; thus studies conducted in inner city areas will report high rates for unemployment or unmarried status. Case-records studies or those using GPs will also be subject to bias because of the known effect that sociodemographic variables exert upon GPs' case-detection abilities; for example Marks *et al* (1979) demonstrated that doctors are more likely to detect psychiatric disorder in women than in men. Studies in which only the patient is interviewed may be biased because of the perceptual distortions which being depressed confers; for example, Henderson (1981) showed that depressed people tended to devalue or underestimate the true nature of their supportive relationships. Lastly, few studies have controlled for other social variables when examining for associations between depression and individual sociodemographic factors an exception to which being that of Hesbacher *et al* (1975).

Because of the uncertain and conflicting nature of the evidence it is not proposed to discuss individual sociodemographic factors and depression in great detail. Two factors, however, do merit attention—sex and age.

Sex

All the studies of depression in general practice quoted so far report a sex distribution in which women far outnumber men. General population studies report a sex-ratio for various depressive disorders of approximately 2:1 (e.g. the recent ECA programme, Robins *et al*, 1984; Myers *et al*, 1984) but in general practice this ratio rises to 3:1 or even 4:1 (Porter, 1970; Dunn & Skuse, 1981 & Sireling *et al*, 1985*a*). The reasons for this sex differential are complex and poorly understood but genetic factors probably make only a small contribution. Jenkins & Shepherd (1983), in their review, suggest that the greater prevalence of minor psychiatric morbidity amongst women may have an environmental basis but illness-behaviour is also an important consideration. Consultation rates are higher in women than men for almost all forms of complaint and women report more symptoms and are more likely to seek help for minor complaints of any kind than men (Shepherd *et al*, 1966). Primary care studies of depressive symptoms shown in Table I are unanimous in finding that women tend to have higher scores on self-report questionnaires than men. Men, on the other hand, are less inclined to identify themselves as psychiatrically ill or to communicate their feelings to others (Horwitz, 1977), a finding confirmed by general population studies showing that men are less likely than women to seek professional help when depressed (Weissman & Klerman, 1977 & Roberts & Vernon, 1982). It is well known that men have higher rates than women for suicide and alcoholism both of which are also well known to be associated with depression. Moreover, family studies of alcoholic men have shown that their non-drinking sisters have a higher than expected incidence of depressive illness (Winokur *et al*, 1971). Thus it may be wrong to identify women as being especially 'at risk' from depression if men express it in different ways (e.g. through violence or substance abuse, so earning an inappropriate label of 'personality dis-order'). In the light of the link between illness behaviour, alcoholism, and depression in men it is worth noting that a recent general population (Robins *et al*, 1984) and primary care (Casey *et al*, 1984) study both reported the usual excess of depression amongst women and alcoholism and personality disorder amongst men but found that the prevalence of *total* psychiatric disorder was the same in both sexes.

Age

Depressive illness has been traditionally thought of as a disorder of middle age and onwards, a concept supported by Dunn & Skuse (1981) who examined patient case-records from a single practice for a 20-year period. They found that women became progressively more prone to depression (or consult for it) as they grew older. However, this positive association between depression and older age may be complicated by other factors such as course and outcome: Dunn & Skuse showed that women were less likely than men to recover once depressed—and so more likely to have chronic disorders. The 'gathering' effect of those with chronic disorders will tend therefore to favour older depressives in cross-sectional studies in which chronicity is not controlled for. Not all primary care studies, however, support the idea of the predominantly older depressive. Mazer (1967) in a five-practice study in Massachusetts, noted that the peak age group for women complaining of disorders which were predominantly 'psychoneurotic' or affective in nature, was 25–34 years. (Men 'peaked' about a decade later). So marked was this finding that 12% of the women in this age range had consulted their GP with a psychiatric complaint at some point during the 3 months of the study. Thereafter the prevalence rates by age fell off rapidly suggesting either that many of these disorders do not persist or that these patients become disillusioned and cease seeking help from the GPs. Other primary care studies also support Mazer's findings showing that younger adults seem to be especially at risk from depression (Porter, 1970; Berndt *et al*, 1983 & Sireling *et al*, 1985*a*). Of course illness-behaviour must be acknowledged since older patients may be reluctant to disclose that they are depressed. Weissman *et al* (1981), in a general population study of depressive disorder, found that younger patients were three times more likely than older age-groups to consult a GP when depressed. Nevertheless illness-behaviour is unlikely to be the only factor behind this association between depression and younger age in primary care. Several studies record that younger adults have more depressive symptoms and symptoms of a greater intensity than older groups e.g. Craig & Van Natta (1979). Recent and reliable general population studies also show that it is predominantly younger adults who are now developing the major depressions. All three sites in the massive NIMH study in the USA (Robins *et al*, 1984) obtained the same unexpected finding that lifetime prevalence rates for major depressive disorder was significantly greater in the younger as opposed to the older respondents. The peak age-group in this case was 25–44. The conclusion from these findings is that younger adults now seem especially likely to develop major depression, and more likely to consult a GP when they do so. On the other hand they are also more likely to recover once depressed than older patients and less likely to experience further episodes over the following decades. The significance could be that depression is a phenomenon which is on the increase, or that general population and primary care studies are now 'unearthing' a previously unknown phenomenon of depression in the young. Older patients who have chronic or recurrent depressions and who are selectively referred to psychiatrists may represent a later, second, prevalence-peak.

Social class and socioeconomic status

These often reflect the characteristics of the practices studied and it is not surprising that studies of psychiatric disorder in different primary care settings are in disagreement as to whether certain classes of patient are more prone to depression than others. Nevertheless several primary care studies are in agreement that patients from lower socioeconomic classes appear to be especially 'at risk' of depression. Wright *et al* (1980) found that the single most important predictor of score on a self report depression questionnaire was socioeconomic status: the higher the status or educational attainment the less likely it was that the person would rate themselves as depressed. This finding was confirmed by Hesbacher *et al* (1975) and Nielsen & Williams (1980) both of whom took the precaution of controlling for other social variables first. Of course studies such as these may be picking up variations between the classes in terms of response-set, not actual depression. Class is a very complex variable and previous efforts to study class in relation to populations which include housewives and the increasing numbers of unemployed or never-employed suffer from the usual difficulties of definition. More detailed and sophisticated analyses of individual variables which are linked with class (such as income, the use of leisure time, and social networks) are now required.

Civil status

Primary care studies reporting associations between civil status and depression are wildly contradictory and no conclusions can be drawn. Thus Porter (1970) found that single women were almost entirely spared from depression whilst married ones were particularly at risk. Berndt *et al* (1983), conversely, found that the highest scores on the Beck Depression Inventory were obtained from the single, divorced, and widowed whilst the never-married had the lowest scores of all. Nielsen & Williams (1980) showed, predictably, that any severing of marital ties was associated with higher depression scores on the BDI, whilst Craig & Van Natta (1979) found that the never-married had the highest rate of depressive symptoms and the married, the lowest.

Civil status, social support, social stresses, life difficulties and life events are all important topics in depression but few or none of the studies of depression in primary care have examined them. They thus fall outside of the scope of this article and readers are referred to the recent reviews by Jenkins & Shepherd (1983) and Paykel *et al* (1984) in which such topics receive extensive coverage. Therefore, we turn now from issues of caseness, prevalence, severity, (i.e. what general practice depression is like) to consider those which directly concern the GP-patient axis; in particular the decision to consult, presenting symptoms, diagnosis by GP, and treatment.

The decision to consult

General population studies show that the majority of people who develop depressive disorders seek help from a GP. Although the situation is somewhat different in the USA where one may bypass the GP and contact a mental health professional directly, Weissman and Myers (1978) still found that 85% of community respondents had in fact turned to their

GP for help. In the UK, 68% of Brown & Harris' (1978) sample of depressed women and 88% of Wing *et al*'s (1981) community sample had sought help from their GP in the previous year. Confirmatory evidence of this help-seeking behaviour in connection with primary care is shown by Widmer & Cadoret (1978) who found that consultation frequencies increased as patients developed depression.

Severity of depression is one factor in leading to a decision to consult. Weissman & Myers (1978) found that the more severely depressed a person was as measured by their score on a self-report questionnaire, the more likely they were to have sought help, and measures of depression and anxiety, found that severity of symptoms or emotional distress gave the best discrimination between patients who had or had not consulted their GP. Only 9% of the non-consulting depressed were as severely depressed as 70% of those who had consulted the GP. In addition those who were classified as 'ill' as well as being emotionally distressed were significantly more likely to have gone to the GP although they claimed that the overlap was small and of little practical significance. The authors posited a link between major life events, a tendency to greater severity of psychological symptoms, and likelihood of consulting a GP.

Not all those who suffer from depression will necessarily choose to consult their GP for help, particularly if the GP in question has been found to be unhelpful in the past. In particular, as already discussed, men and those in older age groups seem to be less inclined to consult a GP when depressed—whether or not out of stoicism is not clear. The importance of the findings with regard to decision to consult is that the burden for delivering care to the majority of society's depressed falls to the GP. It remains to be seen how such patients present and how they might be recognised.

Physical symptoms and the presentation of depressive disorder

Depressed people may consult their GP in one of two ways: either complaining of depression or presenting with other complaints which are more physical in nature. It is frequently reported that general practice depressives are especially likely to present with complaints which are partly or wholly somatic in nature. Watts (1970), reporting on a large series of 773 depressed primary care patients, showed that only "a small number" presented with symptoms that were "clearly depressive in type". The ten symptoms most commonly presented were:

1. tiredness, shortage of energy, feelings of being weak and run-down
2. headache
3. anxiety or tension
4. depressed mood
5. backache
6. insomnia
7. pains in the chest
8. dyspepsia
9. giddiness
10. pains in the trunk, arms and legs.

It is interesting to note that pain was responsible for 35% of the presenting symptoms. Nevertheless, some of these complaints (namely 1, 3, 4, and 6) might be legitimately regarded as part of the depressive syndrome thereby reducing the force of Watts' conclusions. Such somatic symptoms may of course originate from several sources. They might be vegetative symptoms belonging to the depressive syndrome, part of the psychological 'vocabulary' of patients who habitually somatise their feelings, psychogenically determined hypochondriacal symptoms such as 'pain', or the exacerbation of actual physical illness by underlying depression. Examination of the literature shows that the problem in presentation of depression seems not to be that depressive features are absent, or that these disorders are true 'masked depressions', but that the key symptoms are obscured by a great deal of somatic 'noise'.

Thus Goldberg & Blackwell (1970) found that only 7.8% of predominantly affective disorders presented with symptoms which were entirely psychological in nature whilst the majority had a combination of psychological and somatic symptoms. Likewise Widmer & Cadoret (1978) demonstrated that 26% of primary care depressives had somatic symptoms which featured prominently in their presentation (only 9% had purely physical complaints). Widmer divided these somatic complaints into:

1. ill-defined 'functional' complaints
2. pain of undetermined aetiology in a wide variety of sites
3. 'nervous complaints' including mainly tension and feelings of anxiety.

Nevertheless, two-thirds of Widmers' sample of 154 depressives presented non-somatically.

'Caseness' and severity in the context of presentation of depression is likely to be very important but is seldom, or never, properly defined. In addition, there is lack of rigorous care of the definition of what is a 'presenting symptom'—whether it is the first complaint uttered by the patient, the main 'theme' of the consultation, or the complaint which the GP or the investigator considers to be the most important. The definitive study on depression in this setting has yet to be done, although at least three separate studies are now underway and should be appearing in due course. The conclusion to date is, that although a proportion of depressed persons in primary care present somatically, this does not justify an assumption that many do. Experience teaches that the majority of general practice patients with depressive disorders can be quite easily identified providing the usual clinical enquiries about both cognitive and physiological components of the syndrome are made. There is nothing mysterious about general practice depression in this context. The difficulties more often arise when the various symptoms which go to make up a diagnosis of depression are also shared by physical illnesses of one sort or another.

Depression and physical illness

The relationship between depression and physical illness is well established. Not only do chronic, painful, disfiguring or incapacitating illnesses induce low spirits in people who have them, but certain illnesses are also known to be powerful depressors of mood in their own right, most notably the endocrine, metabolic, infective, and neoplastic diseases. Not surprisingly, since a large number of people who go to GPs are suffering from physical disorders, the association between physical and depressive illness in primary care, as on a medical ward, is a frequent and strong one. Nielsen & Williams (1980) reported that 46% of medically ill primary care patients scored over 13 on the Beck Depression Inventory compared to only 9% of those without such illnesses. Wright *et al* (1980), too, found that the risk of depression was increased for GP patients who had co-existing medical illness. However, Sireling *et al* (1985*b*) found, contrary to expectation, that only 36% of their GP depressives had physical illnesses and in only 20% was this judged to be related to the mood disturbance. A converse association between depression and physical illness may be due to the increased vulnerability to physical illness that psychiatric disorder exposes people to. Many psychiatrically ill patients have low incomes, poor diets and hygiene, poor housing, and are obliged to live in crowded hostels where transmittable illnesses are sometimes rife. Shepherd *et al* (1966) showed that psychiatric patients, especially those with chronic conditions, tended to have more general physical morbidity than other primary care consulters. A 'significant associated medical factor' was found in 44% of those with psychotic and 23% of those with neurotic illnesses. In addition some people appear to be specially susceptible to both physical and psychiatric illness. This hypothesis was tested by Eastwood & Trevelyan (1972) who made careful independent assessments of both psychiatric and physical illness in primary care patients using objective data including laboratory investigations. Of those with psychiatric disorders, 17% were also found to have physical disorders (often unknown to the patient) compared with only 2.4% of the non-psychiatrically ill control group. They concluded that both types of disorder tended to 'cluster' in some individuals. In such cases the psychiatric disorder tended to be chronic and to precede the onset of the physical disorder. The older the patient, the greater this tendency was found to be. However, this association between depressive and physical morbidity may not be a direct aetiological one:

(1) Neurotic or depressed patients consult more frequently than non-depressed and have a low threshold for reporting physical complaints when they do, thereby making this association more apparent than real.
(2) There may be third common factors such as age. As has been already discussed, prevalence rates for chronic and recurrent depressive disorder rise with increasing age as do rates for physical morbidity; thus the association between the two could be due to chance.
(3) Another explanation may be that the presence of physical illness may delay recovery from psychiatric disorder. This is supported by Mann *et al* (1981) who found that primary care neurotic patients, still disturbed at follow-up 1 year after entry, were more likely to be suffering from physical illnesses than those who had recovered. From the epidemiologists point of view the importance of this finding is that patients who have both types of illness will tend to be over-represented in samples of consulting patients.

In cases in which symptoms are shared between depression and physical illness and distinguishing between them proves difficult, other, non-core syndromal, symptoms such as anxiety or a previous psychiatric history of depression assume greater importance. However, even with the best of intentions, some patients will defy confident diagnostic allocation and will ultimately require a trial of treatment.

Non-detection of Depressive Disorder by the GP

It has been amply demonstrated in a number of studies that GPs fail to detect between 33% and 50% of cases of psychiatric disorder presenting to them. The same rates for non-detection apply too to depressive disorder as shown by Bebbington (1978); Houpt *et al* (1979); Hoeper *et al* (1980); Nielsen & Williams (1980); Hankin & Locke (1983) and Zung *et al* (1983). One might expect the effect of co-existing physical illness on GPs' case-recognition abilities to be a powerful one and Freeling *et al* (1985) noted that major depressives missed by their GP were more likely to be suffering from physical illnesses to which the depression seemed to be related. Other investigators have concentrated more on the sociodemographic characteristics of unrecognised patients. Hankin & Locke (1983) and Hoeper *et al* (1984) in the USA, Marks *et al* (1979) in the UK, and Chancellor *et al* (1977) in Australia all confirm that GPs tend to under-diagnose psychiatric disorder in males, the young and old, those with higher education, and those who are unmarried. Conversely, females, those from the lower socioeconomic classes, and who are separated, divorced or widowed (and unemployed), are more likely to be correctly identified by the GP when psychiatrically ill. (Probably because of small sample size, Freeling *et al* (1985) failed to demonstrate any sociodemographic differences between recognised (10) and unrecognised (14) patients with depressive disorder). It is likely that the influence that such sociodemographic variables play in affecting case-recognition is mediated through the GPs' notions about what constitutes a 'typical' case which is itself dependent upon previous experience with large numbers of young depressed women. The reasons for GPs failure to recognise cases of psychiatric disorder have been discussed at length by Goldberg and Huxley (1980) who divide them into 'patient factors' (chief of which is the tendency to 'somatise' psychological symptoms (see above)), and 'GP factors' (which include diagnostic 'bias' and 'accuracy'). However, a third source of non-recognition of disorder which is seldom alluded to, concerns the nature of the primary care system itself. The approach to diagnosis and management of patients in primary care is radically different from that in hospital-based settings. The average duration of a consultation in primary care is about 7 minutes, during which time the GP must select which aspects of the patients complaints he regards as important. Primary complaints may have to be pursued at the expense of secondary or tertiary complaints. In addition, GPs often spread diagnostic decisions over several consultations and defer making a firm diagnosis (especially when evidence is uncertain) until a patient has presented with the same symptoms on several occasions. This pragmatic policy probably derives from GPs' previous experiences with large numbers of psychological disturbances which are essentially short-lived and self-limiting. Evidence for the effect of the consultative process upon case-detection, however, is provided by studies showing that GPs are more likely to pick up cases of psychiatric disorder that are chronic, already known to them, or in which the patient is a frequent consulter (Shepherd *et al*, 1966; Locke *et al* 1966; Marks *et al*, 1979; Hankin & Locke, 1983). Thus studies yielding high rates of hidden psychiatric morbidity in which the diagnostic efficiency of GPs are examined on the basis of a *single* index consultation, may not do justice to GPs. A more accurate measure of 'hidden' psychiatric morbidity might therefore be obtained if investigators took *serial* patient consultations and then examined the diagnostic efficiency of GPs at the end of a defined number (e.g. three). It is also important, because of the way GPs proceed to diagnosis, to allow them a degree of flexibility in their response to case-recognition since GPs may fail to award a specific diagnostic label but will often record a psychological observation (if asked) which indicates that they are not completely unaware of patients' mental states. In a WHO collaborative study, Gastpar *et al* (1981) found that GPs had little difficulty in differentiating between depressed and psychologically normal patients but differentiation between depressed patients and those with other psychiatric disorder proved more difficult. Thus, in many studies, failure to recognise a disorder may be confused with failure to award a specific label. As was shown to the case in the decision to consult, there appears to be a relationship between syndrome-severity and the likelihood of a case being detected and correctly identified by the GP. Hankin & Locke (1983) reported that the mean depression score of a group of GP-identified depressives as measured by a self-report questionnaire (the CES-D) was significantly higher than for the non-identified depressives. As part of Sireling's study (see above) Freeling *et al* (1985) found that their small sample of 24 major depressives who were 'missed' by the GPs were 'less obviously depressed' and less likely to complain of depression. They were also more likely to have feelings other than normal sadness, and to report mood changes which were responsive to external cues. Although patient factors are not unimportant, case-recognition is still largely dependent upon the clinical vigilance on the part of the GPs. In the light of this the study by Goldberg *et al* (1980), showing that GPs case-recognition can be improved by giving extra tuition in interviewing skills, has direct relevance.

Treatment of depression in primary care

Watts (1966) suggested that a motivated GP should be able to treat '90%' of his depressed patients at home, emphasising that this was not only less costly than specialist care but far better for the patient since it maintained important ties with the family and community. For the vast majority of persons in the community who, develop depressive disorder 'treatment' will continue to come in the form of interventions from their GP since only a small proportion of those who consult their GP are ever referred to psychiatric services (a situation which is unlikely to change in the future). The treatment options available to the GP fall, broadly speaking, into three groups: psychotropic medication, some form of psychotherapeutic or social intervention, and referral to psychiatric services. The first two alternatives tend to be envisaged at present on an 'either/or' rather than an 'either/and' basis which probably diminishes their overall efficacy. However, for many depressed patients consulting their GP, the decision to treat will often end in a prescription of psychotropic medication. General Practitioners have been criticised in the past for using antidepressants and anxiolytics in an inappropriate and cavalier manner (Weissmann & Klerman, 1977; Weissman *et al*, 1981) and in doses which are low or which psychiatrists consider to be 'subtherapeutic' (Johnson, 1974). Some of this may be excused on the grounds that differentiation between kind and degree of emotional disorder in this setting can be difficult. GPs often base their management decisions on the strength of the primary presenting symptoms rather than the patients membership of a particular diagnostic group. That this affects choice of treatment was demonstrated by Downing & Rickels (1974) who showed that GPs tended to prescribe, in situations of mixed symptoms, according to the predominance of anxiety or depressive symptoms. 'Mixed' cases in which both symptoms featured equally, tended to come off badly in terms of obtaining appropriate treatment, and this was particularly so when a stressful social origin to the depression could also be identified. Sireling and his colleagues (1985*a*) reported that the more severe and less reactive the depressions were, the more likely it was that the GPs would prescribe antidepressants. The degree of stress did not appear to influence the GPs' decisions to prescribe antidepressants although, interestingly, the presence of sleep disturbance did, which suggests that the antidepressants were being prescribed either for their sedative effects or that complaints of sleep disturbance are an effective alerting signal to the GP of the presence of more serious depression. That GPs are not the only non-psychiatrist clinicians who have difficulty in allocating appropriate treatment to patients with depression is shown by Raft *et al* (1975). Studying hospital physicians, they noted that the presence of hopelessness, loss of interest, or the 'physiological' symptoms of depression tended not to alert the physician that the patient was depressed unless they were also accompanied by visible sadness. The doctors in this study justified their decision to prescribe minor tranquillisers on the basis that the patient appeared 'nervous' or had 'anxiety', a similar finding to that of Clare & Williams (1981) who also found that, where anxiety was judged to be the main complaint by the GP, an anxiolytic was almost invariably prescribed. In the light of studies reporting low rates for antidepressant prescribing in general practice one must acknowledge that GPs are sometimes inhibited by the knowledge that a previous prescription of antidepressants (and referral to a psychiatrist) must be declared on life insurance forms and that patients' premiums are raised as a result.

What about efficacy of antidepressant treatment in general practice? Well, despite that fact that Goldberg & Blackwell (1970) report generally good responses to treatment, trials of antidepressants in primary care are rare and very little is known about the efficacy of standard interventions in standard disorders in that setting. Thompson *et al* (1982) includes a brief review of the few treatment trials there have been in this setting and shows that the majority are flawed for one reason or another. Her own study, however, demonstrated the clear efficacy of tricyclics (amitriptyline) on her general practice depressives. One problem which continues to afflict antidepressant treatment in general practice and probably militates against its wider and more effective use are the patients' low compliance rates. Thompson found that 20% of her patients stopped taking the prescribed antidepressants after only 3 weeks, the main reasons for which seemed to be (1) the presence of unwelcome side-effects, (2) worsening of depressive symptoms, or (3) early recovery. Johnson (1981) found that 60% of his series of treated depressed patients also dropped out after 3 weeks for much the same reasons. Other sources of non-compliance were the GPs failure to warn patients about the delay in therapeutic response, the possibility of side-effects, and the need to continue taking the medication regularly. Patient attitudes also affect compliance: Johnson found that patients who believed that their impressions were responses to identifiable life stresses regarded medication as an unsuitable choice of treatment. Other patients feared becoming dependent on medication, a fear which has only been fuelled by recent media coverage on the benzodiazepines. It is therefore probable that a considerable part of the failure of treatment-efficacy with antidepressants in general practice is due to basic failures of understanding and communication between doctor and patient. This is not to assume that all or even many cases of depression seen in general practice require physical forms of intervention. There are moral objections to patients being prescribed psychotropic medication as an alternative to more appropriate (but also more time-consuming) psychotherapeutic or social measures. Lader (1975) criticises the use of medication in situations of stress-related disorder in that they inhibit the patient's own efforts to organise themselves and meet the stresses. However, this argument cuts both ways. It is just as possible that some depressed patients recover, not because of any direct pharmacological activity of medication, but because the benefits in sleep improvement or lowered anxiety, which such medication indirectly affords, enables them to make better use of their other resources. In such situations low-dose antidepressant or anxiolytic or hypnotic medication may be more 'effective' than is generally believed. Medication aside, many depressed patients who attend their GP receive some form of simple counselling and advice (Shepherd *et al*, 1966). The power and efficacy of such interventions must not be dismissed even though GPs have received no formal training in their use. Formal psychotherapy requiring specialist training is probably prohibitively time-consuming for most GPs even though shortened forms, specifically designed for primary care (such as the '20-minute hour'), have been advocated. Balint's views on the 'massive need for psychotherapy' in this context and the therapeutic usefulness of the doctor–patient relationship are well known. However, these ideas have received criticism from some quarters. Sowerby (1977) argues that much of general practice is concerned with problems for which formal psychotherapy is quite unnecessary and that simple advice and reassurance is probably all that is necessary for such patients. However, this view is in danger of running to the opposite extreme. We seriously doubt whether many primary care patients with depressive disorder will derive much lasting benefit from "simple advice and reassurance" alone. With this in mind it is valuable to note the study into the efficacy of social work intervention in depressed women by Corney (1984). In this controlled and randomised primary care trial Corney was unable to demonstrate any preferential benefit in terms of outcome at 6 months for patients allocated to a social worker. A small group of women with acute chronic depressions and marital difficulties did appear to derive some benefit but the overall effect was disappointing and found to be strongly related to the patients motivation and the amount of purely practical assistance they received. Cognitive therapy, although still in the process of being evaluated as a treatment for depression, may turn out to be a useful tool for GPs especially as the skills required to conduct it are relatively easily acquired. Critics remark that it is still too time-consuming for most GPs. Although Teasdale *et al* (1984) make favourable reports about the efficacy of a combination of cognitive therapy and antidepressants, instead of drugs alone, in the treatment of primary care major depression, the actual amount of therapist attention such patients received was of the order of 15 hours. (In addition, the reported gain over the drugs-only group was lost, with three out of 12 patients suggesting that the benefits from cognitive therapy were mainly short-term.) If GPs lack the time to conduct the essential non-drug therapies for depression then who should conduct them? A number of suggestions have been made, and one enjoying a current vogue is the direct deployment of visiting psychiatrists in primary care. The arguments for and against this are reviewed by Mitchell (1985) although the use of trained psychiatrists (outside of an educative and supervisory role) may not be an entirely efficient use of resources. Other sources of psychiatric care in this setting including community psychiatric nurses, psychologists, psychiatric trainees, and psychiatric social workers, have all been suggested although the arguments relating to the appropriate use of scarce resources remain. It is possible that with the burgeoning of the voluntary organisations geared towards counselling and group therapy, the use of non-NHS counsellors and other psychotherapists, providing a service for which the busy GP does not have sufficient time, may become an increasing feature of general practice in the future. It is important, however, for such organisations to define what it is they are trying to deliver and to whom, and to be prepared to be flexible in their clinical approach and relations with statutory services. For the time being we conclude that considerably more could be achieved within the existing system for the many patients in primary care who are suffering from major depressive disorder and for whom antidepressants would be an appropriate course of management. Such patients have most claim on our concern because their need is greater, their disorders are more severe, and because simple psychosocial manoeuvres will be less effective. The improved deployment of standard courses of antidepressants by GPs would, we believe, go a long way towards relieving the misery of a great many patients in this setting.

Course and outcome of depressive disorder in primary care

Studies of psychiatric disorder in primary care have reported that approximately 50% of those cases identified at a single screening exercise have been ill for at least a year, i.e. 'chronic' (Shepherd *et al*, 1966; Kedward & Cooper, 1969; Wing *et al*, 1978, 1981). The problem facing the interpretation of such claims is the lack of specification of the disorders so included in the analysis. Psychotic disorders, personality disorders, and many phobic disorders are generally chronic, whilst the very much more common forms of affective adjustment disorder or situational disturbance are generally short-lived. Accordingly the proportion of acute to chronic disorders reported in such studies will depend greatly upon the composition of the sample. The choice of sampling frame, too, is important since, as discussed before, it affects the ratio of acute to chronic cases. The bulk of the 80% chronicity identified from Hoeper's sample (Regier *et al* (1985) over the course of 3 months was accounted for by personality, psychotic, and anxiety disorders, not depressive disorder. In their sample of 207 consecutive cases of depressive disorder Johnson & Mellor (1977) obtained a ratio of chronic to acute cases of 2.5 to 1. In terms of course and outcome, non-primary care follow-up studies of depressive illness demonstrate that about 15% of acute episodes go into partial remission or remain chronic (Robins & Guze, 1972; Paykel *et al*, 1974; Weissman & Klerman, 1977). Thus, with time, there will be a 'gathering' effect of older, chronic, patients who, because of their higher consultation frequences, will 'haunt' general practice. One might expect depression outside of specialist settings to run a different course but retrospective studies based on the examination of GP case-records have their weaknesses and cannot be entirely relied on. Watts (1970) reported that 21% of a selected sample of 93 'endogenous depressives' had, over 22 years, become chronic and were resistant to treatment. A further 30% had died although one-third of these had recovered by the time of their death. It must be recalled that all these patients had at one time been in-patients, and many had been ill before antidepressant medication had become available. Dunn & Skuse (1981), reporting on a 10-year case-record follow-up of neurotic patients, found that one in six women and one in 20 men with depressive neurosis had developed truly chronic illnesses, the women being especially at risk from this. Cooper (1972) invited eight GPs to identify patients who, in the course of one month, presented to them with chronic psychiatric illnesses. Of the cases so selected, 42% were diagnosed (by the GPs) as having 'depressive neurosis'. One of the few prospective outcome studies of depression in general practice is that of Johnson & Mellor (1977). Of 119 newly diagnosed patients with depressive disorder, 69% were recovered after 4 months and a further 10% much improved, thereby confirming the report of Goldberg & Blackwell (1970) and others that affective illness in general practice has, on the whole, rather a good prognosis. The group of 88 patients in Johnson & Mellor's study who had already been receiving treatment for depression from their GP at entry, and who tended to have had their depression for longer, had fared less well since 44% of them were still unable to resume their normal lifestyle at 4 months. The authors suggest that a small proportion of new cases of depression in general practice eventually become chronic (as shown by non-primary care studies) and are often quite seriously handicapped as a result. Recognition by, and treatment from, the GP must surely play a part in affecting outcome although it is hard to interpret findings which suggest that they do since both factors may be dependent on other variables which exert their own effects upon outcome. Freeling *et al* (1985) reported that although only a few of the GP-recognised depressives had completed even a minimal course of antidepressants, they had all fared better at three months' follow-up than the unrecognised group. However, the identified depressives differed from the non-identified in having had shorter illnesses at the time of diagnosis so that other factors probably play a part too. Johnstone & Goldberg (1976) overcame this difficulty by randomly allocating patients who were suffering predominantly from affective disorder and who had been 'missed' by their GPs to one of two treatment groups. In one group patients were 'notified' to the GP and in the other group the patients diagnostic status was kept concealed. The 'notified' cases did significantly better in the short term and at 1-year follow-up although the differences became less marked as time went by, suggesting that the main benefits from notification (i.e. GP-recognition and treatment) appeared to be in the short term. The differences in outcome between the groups was less pronounced in those patients with milder disorders presumably due to the effects of spontaneous resolution. Chronicity of depression in primary care may therefore be something to do with non-detection (and therefore non-treatment) of existing cases by GPs but it must be recognised that even under the best conditions a small proportion of new cases will fail to recover and run a chronic course. The reasons for this are unclear but may be due to continuing psychosocial stresses or fewer personal resources in the patients who do. There is evidence to suggest that social factors do play a part in affecting the course and outcome of minor psychiatric illness in primary care. In a prospective study, Mann *et al* (1981) found that 22% of patients pursued a variable intermittent course during the follow-up year, whilst 23% failed to recover at all. Here, the major predictive factor of outcome was the overall severity of disorder at the outset but the presence of social stresses, and the perceived quality of social life (in particular family and marital life) turned out to be further discriminating factors.

As regards the course of depressive illness in primary care, 100% of the women but only one-third of the men in Watts (1970) series of ex in-patients had experienced a further episode of depression which required in-patient treatment suggesting that (as shown Dunn & Skuse 1981) women were especially vulnerable to depression and/or further attacks of the same and less likely to make a full and lasting recovery once identified as depressed. Only 20% of Watts's depressed patients had had a single attack of depression in the whole 22 years. Porter (1970) found that 34% of his 93 patients with 'depressive illness' had a further recurrence within only a year of diagnosis. However, Widmer & Cadoret (1978), examining case-records for 154 patients suffering from depression between 1948 and 1971 (mean follow-up period of 6 years), found that 65% of them had been recorded as having only had a single episode of depression, 26% two episodes, and 9% three or more. The median episode length of these depressions was 4 months and only 13% had suffered episodes lasting longer than 2 years. It is clear from the great variation between these studies that the characteristic course and outcome of primary care depressions is not known, some authors claiming how transient and self-limiting they are whilst others point to their chronicity and resistance to treatment. The severity of disorder, composition of the group followed up, and the length of follow-up are all crucial to the interpretation of such findings. Several new follow-up studies of course and outcome of depression in primary care (including our own) are now underway and will be published in due course.

Conclusion

After Adjustment disorder, Depressive disorder is the commonest formal psychiatric disorder in primary care constituting some 8–10% of consecutive-patient consultations. The findings of recent studies suggest that the traditional notion that general practice depression is a mild, self-limiting—and therefore clinically unimportant—condition, is largely mistaken. The bulk of depressed patients presenting to GPs are young and/or female but related social stresses are ubiquitous and do not appear to distinguish between types or degree of associated depressive response. The problems of 'caseness' and their relation to detection of disorder by the GP are such that, although 90% of persons suffering from depression remain within the primary care sector and never enter psychiatric services, a sizeable proportion of them remain undiagnosed and/or inadequately treated. The prognosis is ultimately a good one for many such patients but this failure of service-provision has important consequences for the patient and his/her family in the meantime. Moreover, this has consequences for the GP in that untreated depressed patients impose an increased burden upon primary care services. The size of this unmet morbidity has important implications for the future development of psychiatric services too. The problems of presentation and 'caseness', syndrome severity and profile, relationship to physical illness, and the efficacy of standard psychiatric interventions in primary-care depression all require further study.

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References

BARBER, J. H. (1981) Depressive illness in general practice. *Acta Psychiatrica Scandinavica* (suppl), 441–446.

BARRACLOUGH, B., BUNCH, J., NELSON, B. & SAINSBURY, P. (1974) A hundred cases of suicide: Clinical aspects. *British Journal of Psychiatry*, **125**, 355–373.

BEBBINGTON, P. E. (1978) The epidemiology of depressive disorders. In *Culture, Medicine and Psychiatry*, (ed. A. M. Kleinman), 297–341. Dordrecht, the Netherlands, D. Reidel Publishing Co.

BERNDT, S. M., BERNDT, D. J. & BYARS, W. D. (1983) A multi-institutional study of depression in family practice. *Journal of Family Practice*, **16**, 83–87.

BROWN, G. W. & HARRIS, T. (1978) *Social Origins of Depression*. London: Tavistock Publications.

——, CRAIG, T. K. J. & HARRIS, T. O. (1985) Depression: disease or distress? Some epidemiological considerations. *British Journal of Psychiatry*, 147, 612–622.

BURNS, B. J., ORSO, C. & JACOBSEN, H. (1977) Utilisation of health and mental health out-patient services in organised medical care settings. *Final report, National Institute for Mental Health*, Contract No. 278-76-0027.

CASEY, P. R., DILLON, S. & TYRER, P. J. (1984) The diagnostic status of patients with conspicuous psychiatric morbidity in primary care. *Psychological Medicine*, 14, 673–683.

CHANCELLOR, A., MANT, A. & ANDREWS, G. (1977) The general practitioner's identification and management of emotional disorders. *Australian Family Physician*, **6**, Research report.

CLARE, A. W. (1982) Problems of psychiatric classification in general practice. In *Psychiatry and General Practice*, (eds A. W. Clare & M. Lader). London: Academic Press.

—— & WILLIAMS, P. (1981) Factors leading to psychotropic drug treatment. In *The Misuse of Psychotropic Drugs*. London: Gaskell Books.

COOPER, B. (1972) Clinical and social aspects of chronic neurosis. In *Psychosocial Disorders in General Practice*, (eds P. Williams & A. W. Clare). London: Academic Press.

—— & FRY, J. (1969) A longitudinal study of psychiatric morbidity in a General Practice population. *British Journal of Social Medicine*, **23**, 210–217.

CORNEY, R. H. (1984) The effectiveness of attached social workers in the management of depressed female patients in general practice. *Psychological Medicine* (suppl), **6**.

CRAIG, T. J. & VAN NATTA, P. (1976) Presence and persistence of depressive symptoms in patient and community populations. *American Journal of Psychiatry*, **133**, 1426–1429.

—— & VAN NATTA, P. (1979) Influence of demographic characteristics on two measures of depressive symptoms. *Archives of General Psychiatry*, **36**, 149–154.

CROMBIE, D. L. (1974) Changes in patterns of recorded morbidity. In *Benefits and Risks of Medical Care*, (ed. D. Taylor). London: Office of Health Economics.

DAVIES, B. & BLAKSHI, T. (1973) Course of depression: a comparison of depression in general practice and hospital. In *Classification and Prediction of Outcome of Depression*. Stuttgart: F. K. Schattauer.

DOWNING, R. W. & RICKELS, K. (1974) Mixed anxiety and depression: fact or myth? *Archives of General Psychiatry*, **30**, 312–317.

DUNN, G. (1985) Records or psychiatric morbidity in general practice: the National Morbidity Surveys. *Psychological Medicine*, **15**, 223–226.

—— & SKUSE, D. (1981) The natural history of Depression in General Practice: stochastic models. *Psychological Medicine*, **11**, 755–764.

Eastwood, M. R. & Trevelyan, M. H. (1972) The relationship between physical and psychiatric disorder. *Psychological Medicine*, **2**, 363–372.

Endicott, J. & Spitzer, R. L. (1979) The use of the Research Diagnostic Criteria and the Schedule for Affective Disorders and Schizophrenia to study Affective disorders. *American Journal of Psychiatry*, **136**, 52–56.

——, ——, Fleiss, J. L. & Cohen, J. (1976) The Global Assessment Scale. *Archives of General Psychiatry*, **33**, 766–771.

Fahy, T. J. (1974) Depression in Hospital and in General Practice: a direct clinical comparison. *British Journal of Psychiatry*, **124**, 240–242.

Freeling, P., Rao, B. M., Paykel, E. S., Sireling, L. I. & Burton, R. H. (1985) Unrecognised depression in general practice. *British Medical Journal*, **290**, 1880–1883.

Gastpar, M., Gilsdorf, U. & Gastpar, G. (1981) Diagnosis of Depression in General Practice. In *Prevention and Treatment of Depression*, (eds T. A. Ban, R. Gonzalez, A. S. Jablensky, N. A. Sartorius & F. E. Vartanian). Baltimore: University Park Press.

Goldberg, D. & Blackwell, B.(1970) Psychiatric illness in general practice: a detailed study using a new method of case identification. *British Medical Journal*, **2**, 439–443.

—— & Huxley, P. (1980) *Mental Illness in the Community*. London: Tavistock Press.

——, Rickels, K., Downing, R. & Hesbacher, P. (1976) A comparison of two psychiatric screening tests. *British Journal of Psychiatry*, **129**, 61–67.

——, Steele, J., Smith, C. & Spivey, L. (1980) Training family practice residents to recognise psychiatric disturbances. Final report, Contract No. ADAMHA 278-78-003(DB). South Carolina: Department of Biometrics and Family Practice. Medical University of South Carolina.

Hankin, J. & Locke, B. (1983) Extent of depressive symptomatology among patients seeking care in a prepaid group practice. *Psychological Medicine*, **13**, 1000–1007.

Henderson, S. (1981) Social relationships, adversity, and neurosis: an analysis of prospective observations. *British Journal of Psychiatry*, **138**, 391–398.

——, Duncan-Jones, P., McAuley, H. & Ritchie, K. (1978) The patient's primary group. *British Journal of Psychiatry*, **132**, 74–86.

Hesbacher, P. T., Rickels, K. & Goldberg, D. (1975) Social factors and neurotic symptoms in family practice. *American Journal of Public Health*, **65**, 148–155.

Hoeper, E. W., Nycz, G. R., Cleary, P. D., Regier, D. A. & Goldberg, I. D. (1979) Estimated prevalence of RDC mental disorder in Primary Medical Care. *International Journal of Mental Health*, **8**, 6–15.

——, ——, Regier, D. A. (1980) Diagnosis of mental disorder in adults and increased use of health services in four out-patient settings. *American Journal of Psychiatry*, **137**, 207–210.

——, ——, Kessler, L. G., Burke, J. D. & Pierce, W. (1984) The usefulness of screening for mental illness. *Lancet*, **i**, 33–35.

Horwitz, A. (1977) The pathways into psychiatric treatment: some differences between men and women. *Journal of Health and Social Behaviour*, **18**, 169–178.

Houpt, J., Orleans, C. & George, L. K. (1979) *The Importance of Mental Health Services in General Health Care*. Massachusetts: Cambridge.

Hurry, J., Tennent, C. & Bebbington, P. (1980) Selective factors leading to psychiatric referral. *Acta Psychiatrica Scandinavica*, (suppl.) **285**, 315–323.

Ingham, J. G. & Miller, P. M. (1979) Symptom prevalence and severity in a general practice population. *Journal of Epidemiology and Community Health*, **33**, 191–198.

Jenkins, R. & Shepherd, M. (1983) Mental illness and general practice. In *Mental Illness: Changes and Trends*, (ed. P. Bean). Chichester: Wiley.

Johnson, D. A. W. (1973) Treatment of depression in general practice. *British Medical Journal*, **2**, 18–20.

Johnson, D. A. W. (1974) A study of the use of antidepressant medication in general practice. *British Journal of Psychiatry*, **125**, 186–192.

—— (1981) Depression: treatment compliance in general practice. *Acta Psychiatrica Scandinavica*, (suppl.) **63**, 447–453.

—— & Goldberg, D. (1976) Psychiatric screening in General Practice: a controlled trial. *Lancet*, **i**, 605–608.

—— & Mellor, V. (1977) The severity of depression in patients treated in general practice. *Journal of the Royal College of General Practitioners*, **27**, 419–422.

Kedward, H. B. & Cooper, B. (1969) Neurotic disorders in urban practice: a three-year follow-up. *Journal of the College of General Practitioners*, **12**, 148–163.

Lader, M. (1975) The social implications of psychotropic drugs. *Royal Society of Health Journal*, **95(b)**, 304–305.

Locke, B. Z., Krantz, G. & Kramer, M. (1966) Psychiatric need and demand in a prepaid group practice programme. *American Journal of Public Health*, **56**, 895–904.

Mann, A. H., Jenkins, R. & Belsey, E. (1981) The twelve-month outcome of patients with neurotic illness in general practice. *Psychological Medicine*, **11**, 535–550.

Marks, J. N., Goldberg, D. & Hillier, V. F. (1979) Determinants of the ability of general practitioners to detect psychiatric illness. *Psychological Medicine*, **9**, 337–353.

Mazer, M. (1967) Psychiatric disorders in General Practice: the experience of an Island community. *American Journal of Psychiatry*, **124**, 609–615.

Mitchell, A. R. K. (1985) Psychiatrists in Primary Health Care Settings. *British Journal of Psychiatry*, **147**, 371–379.

Myers, J. K., Weissman, M. M., Tischler, G. L., Holzer, C. E. *et al* (1984) Six-month prevalence of psychiatric disorders in three communities. *Archives of General Psychiatry*, **41**, 959–967.

Nielsen, A. C. & Williams, T. A. (1980) Depression in ambulatory medical patients. *Archives of General Psychiatry*, **37**, 999–1004.

Paykel, E. S. (1974) Recent life events and clinical depression. In *Life Stress and Illness* (eds E. K. Gunderson *et al*). Springfield, Illinois: C. C. Thomas.

——, Klerman, G. L. & Prusoff, B. A. (1974) Prognosis of depression and the endogenous-neurotic distinction. *Psychological Medicine*, **4**, 57–64.

——, Rao, B. M. & Taylor, C. N. (1984) Life stress and symptom pattern in out-patient depression. *Psychological Medicine*, **14**, 559–568.

Porter, A. M. W. (1970) Depressive illness in a general practice: A demographic study and a controlled trial of Imipramine. *British Medical Journal*, **1**, 770–778.

Raft, D., Davidson, J., Toomey, T. C., Spencer, R. & Lewis, B. (1975) In-patient and out-patient patterns of psychotropic drug prescribing in non-psychiatrist physicians. *American Journal of Psychiatry*, **132**, 1309–1312.

Regier, D. A., Burke, J. D., Manderscheid, R. W. & Burns, B. J. (1985) The chronically mentally ill in primary care. *Psychological Medicine*, **15**, 265–273.

Roberts, R. & Vernon, S. (1982) Depression in the community. *Archives of General Psychiatry*, **39**, 1407–1409.

Robins, E. & Guze, S. (1972) Classification of affective disorders: the primary—secondary, the endogenous, and the neurotic—psychotic concepts. In *Recent Advances in the Psychobiology of the Depressive Illnesses*. Published by the Department of Health, Education, and Welfare. (HSM) 70-9053.

Robins, L. N., Helzer, J. E., Weissman, M. M., Orvaschel, H. *et al* (1984) Lifetime Prevalence of specific psychiatric disorders in three sites. *Archives of General Psychiatry*, **41**, 949–958.

Salkind, M. R. (1969) Beck Depression Inventory in General Practice. *Journal of the Royal College of General Practitioners*, **18**, 267.

Shepherd, M., Cooper, M., Brown, A. C. & Kalton, G. (1966; 2nd ed. 1981). *Psychiatric Illness in General Practice*. Oxford/New York: Oxford University Press.

Sireling, L. I., Paykel, E. S., Freeling, P., Rao, B. M. & Patel, S. P. (1985*a*) Depression in general practice: case thresholds and diagnosis. *British Journal of Psychiatry*, **147**, 113–118.

——, ——, —— & —— (1985*b*) Depression in general practice: clinical features and comparison with out-patients. *British Journal of Psychiatry*, **147**, 119–126.

Sowerby, P. (1977) Balint re-assessed. *Journal of the Royal College of General Practitioners*, **27**, 583–589.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research Diagnostic Criteria: rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.

Teasdale, J. D., Fenell, M. J. V., Hibbert, G. A. & Amies, P. L. (1984) Cognitive therapy for major depressive disorder in primary care. *British Journal of Psychiatry*, **144**, 400–406.

Thompson, J., Rankin, H., Ashcroft, C. W., Yates, C. M. *et al* (1982) The treatment of depression in general practice. *Psychological Medicine*, **12**, 741–751.

Tischler, G. L., Henisz, J. E., Myers, J. K. & Boswell, P. C. (1975*a*) Utilization of mental health services. Patienthood and the prevalence of symptomatology in the community. *Archives of General Psychiatry*, **32**, 411–415.

——, Heinisz, J. E., Myers, J. K. & Boswell, P. C. (1975*b*) Utilization of mental health services II. Mediators of service allocation. *Archives of General Psychiatry*, **32**, 416–418.

Tyrer, P. (1978) Drug-treatment of psychiatric patients in General Practice. *British Medical Journal*, **ii**, 1008.

Watts, C. A. H. (1947) Endogenous depression in general practice. *British Medical Journal*, **1**, 11.

—— (1966) *Depressive Disorders in the Community*. Bristol: John Wright.

—— (1970) A long term follow-up of mental hospital admissions from a rural community. *Journal of the Royal College of General Practitioners*, **20**, 79.

——, Conte, E. C. & Kuensberg, E. V. (1964) Survey of mental illness in General Practice. *British Medical Journal*, **2**, 1351–1359.

Weissman, M. & Klerman, G. L. (1977) The Chronic Depressive in the community: unrecognized and poorly treated. *Comprehensive Psychiatry*, **18**, 523–532.

—— & Myers, J. K. (1978) Rates and risks of depressive symptoms in a US urban community. *Acta Psychiatrica Scandinavica*, **57**, 219–231.

——, —— & Thompson, W. D. (1981) Depression and its treatment in a US urban community. *Archives of General Psychiatry*, **38**, 417–421.

Widmer, R. B. & Cadoret, R. J. (1978) Depression in primary care: changes in pattern of patient visits and complaints during a development depression. *Journal of Family Practice*, **7**, 293–302.

Wing, J. K. (1976) A technique for studying psychiatric morbidity in in-patient and out-patient series and a general population sample. *Psychological Medicine*, **6**, 665–671.

——, Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

——, Mann, S. A., Leff, J. P. *et al* (1978) The concept of a 'case' in psychiatric population surveys. *Psychological Medicine*, **8**, 203–217.

——, Bebbington, P. E., Hurry, J. & Tennant, C. (1981) The prevalence of the general population of disorders familiar to psychiatrists in hospital practice. In *What is a Case? The Problem of Definition in Psychiatric Community Surveys*, (eds J. K. Wing, P. E. Bebbington & L. N. Robins). London: Grant McIntyre.

Wing, L., Wing, J. K. & Hailey, A. (1967) The use of psychiatric services in three urban areas: An international case register study. *Social Psychiatry*, **2**, 158–167.

Winokur, G., Tsuang, M. & Crose, R. (1971) The Iowa 500: affective disorder in the relatives of manic and depressed patients. *American Journal of Psychiatry*, **139**, 209–212.

Wright, J. H., Bell, R. A., Kuhn, C. C., Rush, E. A., Patel, N. & Redmon, J. E. (1980) Depression in Family Practice patients. *Southern Medical Journal*, **73**, 1031–1034.

Zung, W. W. K., Magill, M., Moore, J. & George, D. T. (1983) Recognition and Treatment of Depression in a Family Medicine Practice. *Journal of Clinical Psychiatry*, **44**, 1.

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Review Article

The Neuroleptic Malignant Syndrome, So-called

A Survey of the World Literature

A. M. P. KELLAM

Because of its rarity, the neuroleptic malignant syndrome has been studied in a survey of the world literature. Similar conditions which occurred before the advent of neuroleptics have also been included, as they may indicate that the drugs are not totally to blame. It is also apparent that with modern treatments, the condition is not usually lethal. The implications of this for theories of dopaminergic disturbances underlying the mental illnesses involved are considered. An overall picture of the syndrome, its treatment and outcome, is presented from over 200 published references. The spread of recognition of the condition throughout the world and especially across linguistic barriers has been followed.

The neuroleptic malignant syndrome (NMS) is generally agreed to consist of, first, a diffuse rigidity of the muscles often with extrapyramidal signs such as cogwheeling or more rarely with the waxy flexibility of catatonia. Secondly, there is an alteration of consciousness towards either stupor (with negativism) or coma (with confusion). Thirdly, there is a neurovegatitive dysfunction, of which the first and most prominent feature is a sudden pyrexia accompanied by sweating, tachycardia, and sudden falls in blood pressure. Other clinical features are reported (e.g. dyspnoea) but seem to occur more irregularly and, in many cases, may well be secondary. There is often a leucocytosis and the serum creatinine phosphokinase (CPK) is characteristically elevated. I have adopted a pyrexia of more than 37.5°C, and an abnormality of movement, preferably either parkinsonian or catatonic, as the criteria for inclusion as a case of NMS.

The neuroleptic malignant syndrome closely resembles advanced catatonia. Similar syndromes of unexplained fulminating hyperpyrexia, usually described in the setting of catatonia have been regularly described since the beginning of the nineteenth century, especially in the French psychiatric literature as "délire aigué" or "foudroyant, ('acute' or 'fulminating psychosis' are approximate translations) (Fodéré, 1817; Calmeil, 1832). Bell (1849) first described the syndrome in English as "Bell's Mania". Ladame (1919) used these terms to describe essentially similar cases, as did Claude & Cuel (1927). The second of their three cases was described as showing "Hypertonie" (hypertonia), in the setting of a manic episode which ended in a fatal hyperpyrexia. This is the first description of a neuromuscular abnormality in the condition and shows it occurred long before the introduction of neuroleptic drugs. Stauder (1934) described "Tödliche Katatonie" (acute lethal catatonia) as a syndrome marked by hyperpyrexia and catatonia with excitement or stupor and waxy flexibility. The cases usually ended fatally and, as in Claude & Cuel's cases post-mortem changes, if any, were minimal.

The syndrome gained widespread acceptance on the continent, but tended to be denied in the UK. Mayer-Gross *et al* in their influential textbook (2nd ed. 1960) wrote "Sudden death in acute catatonia has drawn the attention of workers hoping to find lesions indicating an underlying metabolic dysfunction. Critical examination of the post-mortem findings in such cases demonstrated as the immediate cause of death a concurrent somatic illness which could not be clinically diagnosed because of the patients' abnormal behaviour." More recently Levinson & Simpson (1986) stated "The cases of extrapyramidal symptoms with fever are too heterogenous to justify the assumption of a unitary and 'malignant' syndrome". They assembled 39 reported cases and after excluding those with medical problems concluded that in only 14 were "no medical causes of fever identified."

Mahendra (1981) even argued that catatonia may always in reality have been an organic psychosis not especially related to schizophrenia, and that this is why catatonia started to disappear in the 1930s well before the advent of neuroleptic drugs. Against this, photographs of catatonic posturing continued to appear in the standard English Textbooks well into the 1950s. There were 17 cases of fairly classical catatonic schizophrenia showing waxy flexibility and partial or complete akinesia and mutism out of 43 patients with stupor in my study between 1967 and 1970 (Kellam, 1970).

More recently, Tolsma (1967) described similar cases as "acute pernicious psychosis" and urged the importance of early diagnosis and treatment with ECT. Gabris & Muller (1983) under the title "La Catatonia dite pernicieuse" (translated as 'The so-called lethal catatonia') examined the records of 1690 patients admitted to the Hôpital de Céry in Lausanne between 1961 and 1981. They identified 270 as having catatonia. Of the 72 patients who had died, 24 were in the catatonic group, and they were especially over-represented in the cases where death had occurred before the age of 50. In one surviving case it appeared that NMS had preceeded the administration of any neuroleptics which here proved beneficial. Mann *et al* (1986), have reported 292 cases of 'lethal catatonia' from the literature since 1960. They conclude that it is a syndrome which may develop in association with both functional and organic illnesses, and that "NMS may be considered a neuroleptic-induced iatrogenic form of lethal catatonia". In all cases hyperpyrexia appears to be the life-threatening problem and usually the ultimate cause of death, unless bronchopneumonia or some other terminal complication supervenes.

It seems probable that a catatonic syndrome has been regularly recorded in schizophrenia since well before the condition was delineated. Certainly, catatonia can occur as a result of numerous other causes, the next most frequent probably being various organic states. The syndrome, however, occurred sufficiently frequently in schizophrenia for it to give its name to a subtype of that diagnostic grouping. In schizophrenic patients at least, this syndrome may rarely take on a fulminating course leading to a potentially fatal hyperpyrexia, which seems clinically indistinguishable from NMS as it is now described, except for the absence of parkinsonian features. The resemblance of these catatonic conditions to NMS is especially striking if the possibility is accepted that the extrapyramidal signs of NMS could be due to the neuroleptic drugs now almost universally involved. This raises the question as to whether catatonia may itself sometimes become a life-threatening condition, and whether in some current cases of apparent NMS, neuroleptics are not pathogenic.

If the syndrome seen in catatonic schizophrenia, other catatonias and the NMS are indeed the same, then it would seem probable that all of them can be produced by a sudden disturbance in the dopaminergic systems of the brain. From this it would follow that alterations in dopaminergic functioning probably occur in this form of schizophrenia, and may well be of quite fundamental importance in its pathogenesis.

In addition to occurring without the involvement of neuroleptics, the NMS can also clearly occur in conditions which are definitely not schizophrenic nor even psychotic, when there is exposure to neuroleptics. Examples of the former group, psychotic but not schizophrenic; are mania, (Goekoop & Carbatt, 1982); hypomania, (Town, 1982); depression (Sumiyoshi *et al*, 1983), two cases; bipolar affective illness (Smith & Carter, 1984); and manic depressive psychosis (Spring & Frankel, 1981; Singh, 1981) three cases. Examples of the latter non-psychotic group: include character disorder, (Bourgeois *et al*, 1971); psychopathy, (Dumont *et al*, 1978); acute grief reaction, (Kaskey & Nasr, 1979); and heroin addiction, (Bleichner *et al*, 1981), all conditions which have never been associated with catatonia. Two cases even occurred in well patients given neuroleptics as pre-operative medication, (Moyes, 1973) and (Konikoff *et al*, 1984).

Some cases not involving neuroleptic drugs occurred in organic states, Huntington's Chorea, (Burke *et al*, 1981); and Parkinson's disease, (Toru *et al*, 1981) and (Fujitake *et al*, 1984), but only when dopamine agonists which were used to treat parkinsonian symptoms were stopped abruptly. In addition Fujitake *et al* (1984) reported eight cases, Toru *et al* (1981) two, and Sechi *et al* (1984) one case of NMS following the abrupt cessation of L-DOPA with, in some cases, the abrupt cessation of amantadine. It has also been recorded as being caused by several other drugs or drug combinations which lower the activity of the dopaminergic receptors. For example Ritchie (1983) reported a case following an overdose of dothiepin and phenelzine; Eiser *et al* (1982) a case involving amitritypline and thioridazine in therapeutic doses; and Burke *et al* (1981) a case involving tetrabenazine (a reserpine-like drug which depletes monoamine stores) and α-methyl-para-tyrosine, again in therapeutic doses. These cases all suggest that sudden alterations in dopaminergic activity can themselves produce NMS.

Judging by the recent increase in articles on NMS in the English language medical literature, there has been a recent upsurge of interest. There was a delay between the first reports in the French language which appeared fairly regularly from 1960 onwards, and those in the English language journals. The first description of NMS as a complication of neuroleptic therapy, was by Delay *et al* (1960) in French. The paper appeared in the proceedings of the Société Médico-Psychologique for December 21, 1959, in a communication on the use of the then new drug haloperidol. It was described as a neurovegetative complication, as opposed to the neurological complications discussed in the next section. The first description in English was by Delay & Deniker (1968) when the term appears as a subheading in a chapter on drug-induced extrapyramidal syndromes in *Handbook of Clinical Neurology*.

In the English language journals the first reference to Delay & Deniker's description of NMS was by Metzler (1973) when he recognised it as a complication of treatment with depot fluphenazine enanthate. Four further papers before 1980 mentioned the term whereas 12 others describing similar cases between 1970 and 1979 made no reference to this work. Since 1980, case descriptions have become far more frequent as has the use of the term "neuroleptic malignant syndrome', probably following the article by Carroff (1980). This delay throws an interesting light on the spread of ideas in a science which aims to be international. Articles have also appeared in the journals of most other European countries with the exception of Scandinavia, and in journals from India, Japan, Australasia, and South Africa, as well as, of course, America.

In the survey by Burke *et al* (1981) 14 cases from the literature were reported plus one of their own. Since then, there has been a large increase in the number reported and I have collected about 300 cases from 164 references and five personal communications. Of these 251 have contained sufficient information to be included in the compilations. These which have had to be excluded because of insufficient information nearly all dated from the nineteenth century and the early years of the twentieth century. The cases quoted here from the French literature are a sample only, because of the difficulty in obtaining French journals in the UK.

Neuroleptic malignant syndrome has a distinct section in an abridged French textbook on psychiatry, (Lemperier & Feline, 1982), but it is not mentioned in any British textbooks including the recently published *Handbook of Psychiatry* (Shepherd *et al*, 1982). It appears in the American *Modern Synopsis of Comprehensive Textbook of Psychiatry IV*, (Kaplan & Sadock, 1985) but is not in the third edition.

Table I shows the number of papers and case descriptions appearing by year, and also the languages in which they were published.

During a 2-year survey of all cases of stupor in central South Wales between 1968 and 1971 (Kellam, 1970), there was one case meeting the criteria of NMS. This level of incidence seems to have continued and that it is certainly not much more than one case per million population every 2 or 3 years. The annual incidence, therefore, should lie between one case per million and one case per five million population. This would give the average psychiatrist about an even chance of seeing a case during his working lifetime.

In their letter to the *New England Journal of Medicine*, Cohen *et al* (1985) suggested that the annual incidence of the syndrome amongst persons being treated with neuroleptics could be as high as 1.4% of treated persons. In the subsequent article Pope *et al* (1986), identified seven definite or probable cases among 1239 patients admitted to their hospital. They estimated that, of these 39% or 483 were exposed to neuroleptics and thus justified their high rate of incidence. Other estimates of the incidence amongst neuroleptic treated cases have been made by Caroff (1980), 0.5% and Shalev & Munitz (1986), 0.4%. The last authors reviewed retrospectively 1250 cases admitted to their ward for neuroleptic treatment since 1972 and found five cases.

There appear to be no definite statistics showing the percentage of the general UK population receiving neuroleptics at any one time, nor for the number of new courses of neuroleptics started each year. Freeman & Alpert (1986) in their survey of the prevalence of schizophrenia in Salford found a prevalence for 1974 of 6.8 cases per 1000 population. This would suggest that between 5 and 10 per 1000 of the population start a course of neuroleptics in any one year and therefore using the work of Cohen *et al* and Pope *et al* that there should be an annual incidence of between 70 and 140 cases of NMS per million. Using the work of Caroff & Shalev, and Munitz it would be between 20 and 50 cases per million. Support for the lower suggested range of incidence comes from surveys such as Sukanova (1985) who among 4000 patients treated with neuroleptics on her service in Czechoslovakia found six cases of the syndrome, over 6 years. This gives an annual incidence of one case per 670 patients treated, or between eight and 15 cases per million population. These are all very different estimates from mine. For this reason there is a need to try and collect prospectively all the cases occurring in a large population.

It has often been said that NMS most commonly affects young men: out of 247 cases collected (with data on sex available), 92 were female. Table II shows the cases divided by sex and into different age-groups. The number of cases in each group is followed by the percentage of the total in brackets. For comparison, the percentage in the same age- and sex-group for the population of Wales is also shown; these are taken from the Registrar General's mid-year estimate for 1985. There does appear to be an over-representation of young men aged between 16 and 44.

TABLE I

A chronological summary of the description of cases of acute lethal or fulminating catatonias and neuroleptic malignant syndromes from 1832

Year	*Author(s)*	*No. of cases*		
1832	Calmeiel, L. F.	Cases in general (French)		
1849	Bell, L. D.	10 cases		
1919	Ladame, Ch.	51 cases (French)		
1927	Claude, H. & Cuel, J.	3 cases (French)		
1934	Stauder, K. H.	3 cases (German)		
1946	Shulak, N. R.	1 case		
1950	Aronson, M. J.	2 cases		
	In English Papers	*Cases*	*In French papers*	*Cases*
1951	1	0		
1952	1	1		
1958	1	1		
1959	2	1	1	2
		(1st description in French as "Syndrome Malin")		
1962			1	4
1965	1	55	1	1
1967	1	5		
1968	Described as "NMS" by Delay & Deniker			
1970			1	0
1971			4	8
1972	3	3	1	1
1973	6	8		
1974	1	4		
1975	2	0		
1976	3	0 (1 case in Italian)	1	1
1977	4	39 (3 cases in Japanese)	4	11
1978	6	3	2	4
1979	4	5 (2 cases in German)		
1980	5	1	1	1
1981	11	11 (1 case in Hebrew)	2	3
1982	12	10 (1 case in Rumanian)	2 +(1 paper in German)	7
1983	35	36 (2 cases in Japanese)	3	3
1984	35	30 (1 case in German)	2 (8 cases in Japanese)	1
1985	19	22 (6 cases in Czech. 2 cases & 2 references in German)		
1986	9	13		

The survival rate has been quoted at about 20% in many articles: Cohen *et al* (1985) quoted a fatality rate of 14.3%. The Committee on Safety of Medicines (CSM) (1986), stated that of 17 cases reported to them, five had been fatal.

In my compilation the reports from the 1960s and earlier showed a 76% mortality, but at this time a fatal outcome was possibly the usual feature leading to the case being recorded. In the cases from the 1970s, the mortality rate had fallen to 22.7% and, for the cases from the 1980s, further to 14.9%. This may reflect the successes of modern treatment methods rather than just less serious cases being recorded in the literature. Table III shows the relationship between the highest pyrexia recorded and a fatal outcome; unfortunately it could be taken to support either view.

It has been suggested that an elevated serum creatinine phosphokinase (CPK) is found in NMS (Metzler, 1973). This is measured in international units: where quoted, the normal range seems rather variable, but usually less than 100 *IU*/litre. In one

TABLE II
Distribution of cases by age and sex

Age	*Under 15*	*16–24*	*25–34*	*35–44*	*45–64*	*Over 65*
Male cases	4(2%)	37(15%)	49(20%)	25(10%)	35(14%)	5(2%)
Welsh population	10.9%	7.4%	6.5%	6.4%	11.1%	6.1%
Female cases	3(1%)	14(6%)	19(7%)	16(6%)	33(13%)	7(3%)
Welsh population	10.3%	7.3%	6.4%	6.4%	11.6%	9.6%

TABLE III
Pyrexia and mortality

Temperature (°C) over	*37.5*	*38*	*39*	*40*	*41*	*42*	*Fever or hyperthermia*
1970s	2	10	11	19(5)	14(7)	3	7(3)
1980s	12	37(3)	32(2)	36(5)	23(6)	9(5)	17(3)

The cases have been tabulated according to the maximum recorded temperature in degrees centigrade, i.e. the 37.5°C column contains all the cases between 37.5 and 37.9°C; the 38°C column all those between 38 and 38.9°C. After the figures for total cases the number of cases with a fatal outcome is shown in brackets.

possibly related condition, malignant hyperpyrexia, there is also a gross elevation of CPK but this is with a considerable proportion of an abnormal isoenzyme. This isoenzyme can be shown to occur both during the illness and at times when the patient is unaffected as well as amongst some of his relatives. It is believed that this indicates a liability to the disease when anaesthetics are administered. In the cases from the 1970s, elevated levels of CPK were recorded in ten cases, seven between 100 and 999 *IU*/litre and three cases between 1000 and 9999 *IU*/litre. For the 1980s, the numbers of cases recorded for the same groups were as follows, 38 between 100 and 999 *IU*/litre, 28 between 1000 and 9999 *IU*/litre and 14 over 10 000 *IU*/litre. In addition there were six cases where the CPK was only described as elevated.

In the cases from the 1970s, cases of organic mental illnesses showed the highest mortality, four out of seven; these cases included those with Parkinson's disease. Cases diagnosed as schizophrenia also had a higher mortality seven out of 43; whereas there was only one fatality among eight cases diagnosed as manic depressive psychoses, and only one out of seven cases diagnosed as non psychotic illnesses (mainly psychopathies and addictions). In the 1980s the mortality rates improved overall: nine of 75 for the schizophrenias and two of 26 for the organic psychoses. The apparent worsening, nine of 33 for the manic depressive psychoses, and four out of 13 for the group of non-psychotic diagnoses, is probably due to the small size of these groups.

Other possibly related conditions include malignant hyperthermia and heat stroke. Malignant hyperthermia (MH) is a genetically transmitted (autosomal dominant) condition. The inherited liability is to an abnormal muscle reaction to anaesthetic agents producing a similar clinical picture to NMS. The abnormal muscle reaction in addition to being shown by an atypical CPK can be demonstrated by subjecting isolated preparations of the patient's muscle to anaesthetics and caffeine *in vitro*. Recently, Caroff *et al* (1983) has shown a similar reaction to fluphenazine in a preparation from a patient who had recovered from NMS; this reaction was quite distinct from those of controls and also MH subjects. Muscle from this patient reacted to halothane but not caffeine, whereas if it had come from an MH patient it would have reacted to both and more strongly. This work was felt to suggest that there might be a closer relationship between these two conditions than had previously been the view, and of course suggests that the mechanism might not be entirely central. Dantrolene is the treatment of choice for MH but is not always effective in NMS. When dantrolene was only partially effective in NMS it reduced the rigidity and with it the fever which may be produced at the level of the muscles, but not always the confusion, stupor, tremor, or the psychotic symptoms, which are probably more central in origin. In heat stroke and anticholinergic poisoning, the skin is hot and dry and rigidity and tremor, absent which should make differentiation possible. These clinical features are summarised in Table IV.

TABLE IV

Clinical features of catatonia, malignant hyperpyrexia, and neuroleptic malignant syndrome

	Confusion	*Akinesia mutism*	*Rigidity*	*Tremor*	*Pyrexia*	*Neuroleptics involved*
Catatonia	Common	Mandatory	Usual	Possible	Absent	Possible (except in acute lethal)
NMS	Usual	Usual	Essential	Usual	Essential	Mandatory
Malignant hyperpyrexia	—	—	Usual	Usual	Essential	Absent

In the 1970s, depot neuroleptics were involved in 21 cases, with eight fatalities, and oral neuroleptics in 45, with seven fatalities. In the 1980s, the corresponding figures for depot neuroleptics were 28 cases, with eight fatalities, and for the oral drugs 109, with 17 fatalities. During this period there were 21 cases not associated with neuroleptic drugs three of which ended fatally. Nearly all of these cases appeared to be caused by the sudden cessation of dopamine agonists.

Among the injected neuroleptics, there were 16 cases attributed, at least in part, to fluphenazine enanthate with six fatalities; 20 to fluphenazine decanoate, with three fatalities; two to injected haloperidol, five to injected thioxanthines with two fatalities and five to pipothiazine palmitate.

Among the oral drugs, phenothiazines were recorded in 74 cases, with ten fatalities, butyrophenones in 89 cases, with ten fatalities, a thioxanthine in 14 cases, with one fatality, pimozide in three cases and substituted benzamides in seven cases, with four fatalities. Cases produced by non-neuroleptic drugs have only been recorded since 1980. They include reactions apparently to tetrabenazine (one case who survived); monoamine oxidase inhibitors (again one case who survived and one who died); trycyclic antidepressants (three cases - one died); and cessation of dopamine agonists (14 cases - again one died). In many of these, more than one drug may have been involved in a single reaction, and this will have been recorded against each of them.

Finally, the cases have been analysed for the treatment used and their success or failure as recorded in the case records. Between 1970 and 1979, 69 cases were treated by supportive measures, with sometimes the addition of benzodiazepines in three cases, ECT in two cases, and antiparkinsonian drugs in seven cases. Of these, 17 died. One case was noted as treated successfully with anticholinergic drugs.

In the 1980s, in the group receiving supportive treatment only fatalities occurred in 19 of 69 cases, in the benzodiazepine-treated group three cases of 14 died, and in the dantrolene-treated group two cases out of 33 died. Sometimes, an active treatment was not successful and an alternative had to be used. Sometimes treatments such as dantrolene cured the rigidity and hyperpyrexia, but left psychotic symptoms needing further treatment such as ECT, and this, as well as the patient's death, is described as failure of treatment in some cases.

Dantrolene was used in 34 cases and failed in five; bromocriptine was used in 13 cases and failed in one; anticholinergic drugs were used in ten cases and failed in two; amantadine was used in ten cases and failed in one; L-DOPA was used in nine cases and failed in three. Finally ECT was used in eight cases. In a series of eight cases from Japan, (Fujitake *et al*, 1984), antiparkinsonian medication was stopped, producing the syndrome; which was then successfully treated by restarting it. They have not been included in the above figures.

In two cases dantrolene was noted to cure the hyperpyrexia but not the rigidity or the mental disturbance. Intravenous administration of lorazepam has also been shown to be of value in treating catatonic symptoms, and of the four cases reported by Frichione *et al* in 1983, two were apparently suffering from NMS.

From the point of view of treatment, most patients will already have been prescribed neuroleptics, and it would seem essential to stop them immediately and at least for the duration of the pyrexial phase, as there is no way of knowing that they are not doing harm. I have adopted the French practice, whenever I use large doses of neuroleptics or fear the onset of the condition, of maintaining a 4-h temperature chart and making any unexplained rise above 37°C an absolute indication to withhold the drug until the case can be investigated further.

These results show that active treatment is associated with a much lower mortality than supportive treatment alone or combined with benzodiazepines or anticholinergics. The conclusion of the CSM (1986) that "Apart from withdrawal of the drug, treatment is essentially supportive," is also refuted. Probably the best treatment at present would be to combine dantrolene with either amantadine or bromocriptine, or both. If these failed, intravenous lorazepam would be worth a trial, especially if catatonic symptoms were prominent. If dopamine agonists have cured the rigidity and hyperpyrexia but psychotic symptoms persist, then ECT may well be the treatment of choice. ECT may well only have been dangerous when before the advent of effective symptomatic treatments with dantrolene and dopamine agonists, it had to be used on hypertonic, hyperpyrexial patients. Recent experience using it later has apparently been more successful (Greenberg & Gujavarty, 1985). In any event, a patient developing NMS should be moved immediately into medical intensive care and supportive measures should also be used.

In the pre-neuroleptic era, three of 12 cases were recorded as in stupor, and seven as confused; three showed catatonia, two parkinsonism, and two rigidity. Between 1959 and 1968, two out of ten cases were recorded as confused and four as in stupor; three were noted to be catatonic and two parkinsonian, with one recorded as rigid with no indication as to which type. In the cases from the 1970s, 18 out of 66 cases were recorded as confused, and 26 as in stupor; nine were noted as being catatonic, and 29 as parkinsonian with 18 only recorded as rigid and two as flaccid. In the 1980s, 68 out of 161 cases were confused, and 47 stuporose; 79 were parkinsonian, 18 catatonic, 49 rigid, and three recorded as flaccid.

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I am grateful to Messrs Lundbeck, who searched their records and could find no recorded cases of NMS in Scandinavia; Messrs Jannsen for considerable help in providing references, and to my secretary Mrs J. Clarke. I should be grateful if any member of the College coming across a case of NMS (a fever over 37.5°C accompanied by motor disturbances) would notify me. I will request either a loan of the case notes or the completion of a very short form.

References

Aronson, M. J. & Thompson, S. V. (1950) Complications of acute catatonic excitement, a report of two cases. *American Journal of Psychiatry*, **107**, 216–220.

Bell, L. V. (1849) On a form of disease resembling some advanced stages of mania and fever. *American Journal of Insanity*, **6**, 97–127.

Bleichner, G., Squara, P. & Parent, A. (1981) Hypertonia and malignant hyperthermia due to morphine and neuroleptic. *Lancet*, **1**, 386–387.

Bourgeois, M., Tignol, J. & Henry, P. (1971) Syndromes malins et morts subites au cours des traitements par neuroleptiques simples et retard. (Sudden malignant and fatal syndromes in the course of treatment with simple and depot neuroleptics). *Annales Medico-psychologiques*, **129**, 729–746.

Burke, R. E., Fahn, S., Mayeux, R., Weinberg, H., Louis, K. & Willner, J. H. (1981) Neuroleptic malignant syndrome caused by dopamine depleting drugs in a patient with Huntingdon's chorea. *Neurology (NY)*, **31**, 1022–1026.

Calmiel (1832) In *Dictionaire de Medicine ou Repetoire General des Sciences Medicales sous la rapport theorique et practique*. Paris 1832–1846. (Cited extensively by Ladame C.H., *vide infra*, original work not yet traced.)

Caroff, S. N. (1980) The neuroleptic malignant syndrome. *Journal of Clinical Psychiatry*, **41**, 79–83.

——, Rosenberg, H. & Gerber, J. C. (1983) Neuroleptic malignant syndrome and malignant hyperthermia. *Lancet*, **i**, 244.

Cohen, B. M., Baldessarini, R. J., Pope, H. G. & Lipinski, J. F. (1985) Neuroleptic Malignant Syndrome. *New England Journal of Medicine*, **313**, 1293.

Claude, H. & Cuel, J. (1927) Notes anatomo-clinoque sur trois cas de délire aigu. (Anatomo-clinical notes on 3 cases of fulminating psychosis.) *Encéphale*, **22**, 628–632.

Committee On Safety Of Medicines (1986) Neuroleptic Malignant Syndrome – an underdiagnosed condition? *Current Problems*, **18**, 2.

Delay, J. & Deniker, P. (1968) Drug induced extrapyramidal syndromes. In *Handbook of Clinical Neurology, Vol. 6. Diseases of the Basal Ganglia*, eds P. J. Winken & G. W. Bruyn, New York, Elsevier North Holland Inc.

——, Pichot, P., Lempériere, T., Ellisalde, B. & Peigne, F. (1960) Un neuroleptique majeur non phenothiazinique et non reserpinique, l'haloperidol, dans le traitement des psychoses. (A non phenothiazine, and non reserpine major neuroleptic, haloperidol, in the treatment of psychoses.) *Annales Médico-psychologiques*, **118**, 145–152.

Dumont, A., Minjat, M., Blanc, J. L. & Vignon, H. (1978) Le syndrome malin aux neuroleptqies (neuroleptic malignant syndrome). *Annales Anaesthesie France*, **19**, 777–781.

Eiser, R., Neff, M. S. & Slifkin, R. F. (1982) Acute myoglobinuric renal failure, a consequence of the neuroleptic malignant syndrome. *Archives of Internal Medicine*, **142**, 601–603.

Freeman, H. & Alpert, M. (1986) Prevalence of schizophrenia in an urban population. *British Journal of Psychiatry*, **149**, 603–611.

Fodéré. *Traité du délire. t II.* (1817) p. 407. (Cited by Claude & Cuel, *vide infra* original not yet traced.)

Fricchione, G. L., Cassem, N. H. & Hooberman, D. (1983) Intravenous lorazepam in neuroleptic induced catatonia. *Journal of Clinical Psychopharmacology*, **3**, 338–342.

Fujitake, J., Kuno, S. & Nishitani, H. (1984) Neuroleptic malignant syndrome-like state in eight patients with parkinsonism, *Rinsho Shinkeig'aku (Clinical Neurology)*, **24**, 371–378.

Gabris, G. & Muller, C. (1983) La catatonie dite pernicieuse. (The so called lethal catatonia). *Encéphale*, **9**, 365–385.

Goekoop, J. G. & Carbaat, P. A. Th. (1982) Treatment of neuroleptic malignant syndrome with dantrolene. *Lancet*, **ii**, 49–50.

Greenberg, L. B. & Gujavarty, K. (1982) The neuroleptic malignant syndrome; review and report of three cases. *Comprehensive Psychiatry*, **26**, 63–70.

Kaplan, I. H. & Sadock, B. J. (eds) *Modern Synopsis of Comprehensive Textbook of Psychiatry* IV. Baltimore: Williams & Wilkins.

Kaskey, G. B. & Nasr, S. (1979) A side effect of rapid neuroleptisation. *American Journal of Psychiatry*, **136**, 1232.

Kellam, A. M. P. (1970) A survey of stupor with special reference to the aetiology, incidence and differential diagnosis. M.D. Thesis. University of London.

Konikoff, F., Kuritzky, A., Jerushalmi, Y. & Theodor, E. (1984) Neuroleptic malignant syndrome induced by a single injection of haloperidol. *British Medical Journal*, **289**, 1228–1229.

Ladame, C. H. (1919) Psychose aigue idiopathique ou foudroyante. (Acute or fulminating idiopathic psychoses.) *Schweizer Archive für Neurologie und Psychiatrie*, **5**, 3–28.

Lemperiere, T. H. & Feline, A. D. (1977) *Psychiatrie de l'Adulte. (Adult Psychiatry).* Paris: Masson.

Levinson, D. F. & Simpson, G. M. (1986) Neuroleptic-induced extrapyramidal symptoms with fever. *Archives of General Psychiatry*, **43**, 839–848.

Mahendra, B. (1981) Where have all the catatonics gone? *Psychological Medicine*, **11**, 669–671.

Mann, S. C., Caroff, S. N., Bleier, H. R., Welz, W. K. R., Kling, M. A. & Hayashida, M. (1986) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1381.

Mayer-Gross, W., Slater, E. & Roth, M. (1960) In *Clinical Psychiatry* 2nd ed. London: Cassell.

Meltzer, H. Y. (1973) Rigidity, hyperpyrexia and coma following fluphenazine oenanthate. *Psychopharmacologia (Berlin)*, **29**, 337–346.

Moyes, D. G. (1973) Malignant hyperpyrexia caused by trimeprazine. *British Journal of Anaesthesia*, **45**, 1163–1164.

Pope, H. G., Keck, P. E. & McElroy, S. L. (1986) Frequency and presentation of neuroleptic malignant syndrome in a large psychiatric hospital. *American Journal of Psychiatry*, **143**, 1227–1233.

Ritchie, P. (1983) Letter. *British Medical Journal*, **287**, 561.

Sechi, G. P., Tanda, F. & Mutani, H. (1984) Fatal pyrexia after the withdrawal of levodopa. *Neurology (Cleveland)*, **34**, 249–251.

Shalev, A. & Munitz, H. (1986) The neuroleptic malignant syndrome: agent host interaction. *Acta Psychiatrica Scandinavica*, **73**, 337–347.

Shepherd, M. *et al* (1982) In *Handbook of Psychiatry*, Cambridge: Cambridge University Press.

Shulak, N. R. (1946) Exhaustion syndrome in excited psychotic patients. *American Journal of Psychiatry*, **102**, 466–475.

Singh, G. (1981) The malignant neuroleptic syndrome (a review with report of three cases) *Indian Journal of Psychiatry*, **23**, 179–183.

Smith, J. A. & Carter, J. H. (1984) Neuroleptic malignant syndrome with a positive weil-felix test. *American Journal of Psychiatry*, **141**, 609.

Spring, G. & Frankel, M. (1981) New data on lithium and haloperidol incompatibility. *American Journal of Psychiatry*, **138**, 818–821.

Stauder, K. H. (1934) Die tödliche katatonie. *Archiv für Psychiatrie und Nervenkrankheiten*, **102**, 614–634.

Sukanova, L. (1985) Maligni neurolepticky syndrom. (Neuroleptic malignant syndrome). *Ceskoslovenska Psychiatrie*, **81**, 91–95.

Sumiyoshi, A., Oguchi, T., Takahashi, A. & Miura, S. (1983) Syndrome malin induced by tricyclic antidepressants. *Hiroshima Journal of Anaesthesia*, **19**, 15–20.

Tolsma, F. J. (1967) The syndrome of acute pernicious psychosis. *Psychiatria, Neurologia, Neurochirgia*, **70**, 1–21.

Toru, M., Matsuda, O., Makiguchi, K. & Sugano, K. (1981) Neuroleptic malignant syndrome-like state following a withdrawal of anti-parkinsonian drugs. *Journal of Nervous and Mental Diseases*, **169**, 324–327.

Town, I. G. (1982) Haloperidol, Neuroleptic malignant syndrome. *New Zealand Medical Journal*, **2667**, 199.

A full list of all the other references collected, including all the cases used in compiling the tables and other figures, a table showing the clinical features of the unpublished cases and a bibliography of other articles reviewing or discussing the Neuroleptic Malignant Syndrome is available from the author on request.

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Communicating with Cancer Patients

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In a prospective study, 100 cancer patients and their relatives were separately interviewed using a structured interview to examine whether the diagnosis of cancer should be communicated to patients and relatives; the reasons for doing so or not, the emotional reactions of patients and families; any change in the attitude of relatives; behaviour of patients and patient's expectation, on diagnosis, of the course of their illness. The majority of patients, who knew their diagnosis (71%), wanted to be told the truth. The majority of relatives wanted to know the truth themselves (81%) without the patients being told (77%). Patients did show immediate emotional reactions like anxiety and dejection, however they appeared to accept the diagnosis. Findings suggest that in an Indian setting the attitude of family members is more positive. The role played by sociocultural factors in the acceptance of the truth is discussed.

The word 'cancer' connotes an incurable disease followed by a distressing and inevitable death which is an ultimate challenge to the knowledge and skills of physicians, and a disturbing reminder of their limitations. Some doctors experience their failure to save the patient as a personal defeat and humiliation. They may respond with feelings of guilt, shame and resentment. To cope with his own emotions, the doctor may simply avoid the patient or become awkward or detached when they meet. The doctor's withdrawal tends to increase the patient's sense of helplessness and loneliness. Often the patient, his family and the doctor, attempt to maintain the denial of impending dissolution and an awkward game of mutual deception and avoidance of facing the facts takes the place of open communication.

The issue to be faced and settled by all concerned is that of communication of the diagnosis and its consequences. The perennially controversial question is whether or not to tell the truth. It is remarkable that extensive studies conducted among physicians and layman, respectively, reveal almost diametrically opposing views on this issue: 80–90% of healthy subjects questioned as well as cancer patients, responded that they wished to be told that they had cancer or another fatal illness (Cappon, 1962; Kelly & Friesen, 1950). However, 40% of dying patients who were asked if they wanted to be told when they would die, responded that they did not (Cappon, 1962).

Of 219 physicians questioned by Oken (1961), 90% said that they did not disclose diagnosis of cancer as a usual policy. In a general poll of 5000 American doctors, 22% said that they never told patients that they had cancer. Yet doctors usually affirm that they would personally want to be told if they were critically ill (Oken, 1961).

Whether or not the patient is told that he has cancer, he sooner or later guesses the truth from non-verbal clues. How this problem should be handled is a difficult issue often confronted by psychiatrists. The question is not only whether to tell, but who should do it, how, when and what should be told. Practical difficulties such as these, led to the present investigation.

Aims of the study

A prospective study was carried out at SMS Hospital Jaipur (India) to find out:

(a) whether diagnosis of cancer should be communicated to patients and relatives
(b) why the diagnosis of cancer should be revealed or not to patients and relatives
(c) the emotional reaction of patients and their relatives when patients know that they are suffering from cancer
(d) if patients perceive a change in their own behaviour after they know the diagnosis
(e) whether there is any change in the attitude of relatives towards the cancer patients on knowledge of diagnosis and if so, what is the change
(f) whether knowledge of having cancer is related to the patient's expectation of the course of the illness.

Method

In a prospective study, 100 consecutive patients, with an established diagnosis of cancer, attending the radiotherapy out-patient department of SMS Hospital, Jaipur, were included. The assessment of patients was done any time within 2 to 4 weeks of establishment of the diagnosis. The patients and their relatives were interviewed separately. Out of 100 patients included in the study, 33 had cancer of the head and neck organs, 32 had involvement of the genitourinary system, and 35 had cancer of sites other than those mentioned above.

All the available 77 relatives were interviewed, using a semi-structured interview schedule. This was designed to elicit the responses of relatives on disclosure of diagnosis both to them and the patients and their emotional reactions towards patients on knowledge of diagnosis (Appendix 1). All 100 patients were interviewed using another interview schedule. This was designed to elicit from patients a) what they thought they were suffering from; b) how they knew about it; c) their emotional reactions to their diagnosis; d) any change in their behaviour (as perceived by themselves) after the onset of illness; e) any change in the behaviour of others towards them; and f) their own expectation of outcome to treatment (Appendix 2). Special care was taken to see that at no time during the interview with the patients, were the words 'cancer', or, 'malignancy' used – or any indirect clue given to indicate their diagnosis. However, 48 patients already knew that they were suffering from cancer – either from the treating physician – or from the case files where diagnosis of malignancy/cancer was written – or they guessed from the fact that they were being treated at the cancer out-patient department – or they guessed it from behaviour towards them. These 48 patients were asked whether they thought their diagnosis should be revealed to them or not – and the reasons for their answers.

We designed the semi-structured interview schedules used for this study in a pilot study for interview of patients and relatives separately. Both of us were well versed with the technique of administering the interview schedule. Rates of literacy were judged according to the educational status of the informant; a person who had no schooling and did not know how to read and write was rated as 'illiterate', while those who had primary education were rated as 'low literate'. A rating of 'moderately literate' was given to those who had finished their school education (12th standard); and those with degree qualifications etc. were rated as 'highly literate'.

The responses were recorded and the results analysed.

Results

Of 100 cancer patients, 51% were ignorant about their illness and only 48% knew about their diagnosis and its outcome, while one patient could not be interviewed since he had a secondary metastasis in the brain (Table I). Of the 48 patients who knew their diagnosis, 23 clearly accepted the fact, while 25 were reluctant and used denial as a defence. It was observed that the majority of the illiterate patients (63%) did not possess the relevant knowledge, most of the low literate (42%) tended to use denial more frequently, while moderately and highly literate patients expressed their knowledge, and denial was infrequently used.

An enquiry into the immediate emotional reactions of patients on knowledge of their diagnosis, revealed that anxiety was the predominant immediate emotional reaction (77%), followed by feelings of dejection (42%); 8% did not initially accept the diagnosis, 4% felt that their anxiety was relieved and 6% reported no specific change. These emotional reactions were not found to be related to the site of cancer, while they appeared to have some relationship with age. The higher the age-group, the greater percentage of patients reported anxiety – while feelings of dejection were found to be maximum in the age-group 30–50 years, and comparatively less common in patients under 30 and over 50 years of age.

When patients were asked whether they perceived any change in their own behaviour after the onset of illness,

TABLE I
Interviews with cancer patients

Patients' knowledge about the illness (distribution according to literacy status)

Responses	*Illiterate* *n*=57	*Low literacy* *n*=12	*Moderately literate* *n*=19	*Highly literate* *n*=2	*Total* *n*=100
No knowledge	42(63%)	3(25%)	6(32%)	—	51
Knowledge	10(15%)	4(33%)	8(42%)	1(50%)	23
Denial of illness	14(21%)	5(42%)	5(26%)	1(50%)	25
Information not available	1	—	—	—	1
Whether truth should be told	*n*=24	*n*=9	*n*=43	*n*=2	*n*=48
Yes	17(71%)	6(67%)	10(77%)	1(50%)	34(71%)
No	2(8%)	1(11%)	2(15%)	—	5(10%)
Undetermined	5(21%)	2(22%)	1(8%)	1(50%)	9(19%)

57% gave an affirmative answer. Changes included preoccupation with illness (77%), loss of interest in daily routine (51%), irritability (40%), increased/reduced intensity of involvement in family affairs (23%). More than one type of change was reported by many patients.

Patients were also asked how they perceived the attitude of relatives towards them. It was found that 46% of the patients felt no change, while 52% felt it; 48 out of 52 patients (92%) reported that relatives were paying more attention to them.

The subsequent emotional state of patients and their perceptions of the course and outcome of their illness was examined. A comparison of responses in those who knew and those who did not know about their diagnosis showed that the majority (53%) of those who did not know or denied it, were hopeful about the outcome while only 29% of those who know, were hopeful about the outcome; 19% withheld expression of their emotional reaction, 17% expressed helplessness, 12% showed features of depression and 41% were not sure of the course and outcome.

When patients were asked if the truth should be told to them, out of 48 respondents who knew of their illness 71% were of the opinion that they should be told, 10% said the truth should not be told, while 19% were indecisive on this issue. The literacy level of the patients, unlike their knowledge of the disease, did not affect their opinion on this issue.

Of those who wanted the diagnosis revealed to them, 68% were of the opinion that it would increase their sincerity, and co-operation with treatment, 29% said that it would help them accept reality, 6% said that they would be in a position to plan for their dependents, while 3% said they could fulfil their last wishes. (More than one answer was given by some respondents.)

Of the five patients who said that the truth should not be told, 60% felt that the revelation would make them feel depressed and 40% anxious. Two of the respondents could not attribute any specific reasons for what they believed. (Two patients gave more than one response.)

We also explored the relationship between the presence of psychiatric morbidity and duration of illness, and found that higher psychiatric morbidity was found among patients with 1–3 years duration.

Interview with relatives

The majority of relatives of cancer patients were of the opinion that the disease and its outcome should be revealed to the relatives (81%) but should not be revealed to the patient (77%) (Table II). A small percentage (3%) of those who thought that the relatives should be informed, expressed the view that distant relatives should not be informed as this would lead to distancing the patient. Only 12% of the relatives were of the opinion that they should not be told while only 10% were of the opinion that the patient should not be told. A small percentage (10–13%) of relatives were indecisive on this issue. The literacy status of respondents was not found to have any bearing on this question.

Of those 62 relatives who said that the truth should be told to them, 73% felt that it would lead to better patient care in terms of medical facilities, early initiation of treatment and sympathetic attitudes from others, while 18% thought that the truth should be told so that the patient could be separated from others; 13% felt that it would assure the relatives about the progress of disease and 8% thought it would help relatives make financial and social arrangements (if need be) for the patient. One thought that it was his duty to know the truth, while another gave no specific reason for knowing.

The nine relatives who felt that the diagnosis should not be revealed to them, thought that knowing the diagnosis and its outcome would lead to a feeling of helplessness, sadness and thus a lowering of their morale.

The majority of relatives (87%) thought that the diagnosis and its outcome should not be told to the patient because it would make him more worried and restless (71%), apprehensive and fearful (49%) and would lead to an early deterioration of patient's general condition (15%). (Some respondents gave more than one response.)

The small percentage of relatives (13%) who wanted the patient to be informed about his disease and its outcome felt that this should be done to remove the patient's fears,

TABLE II
Interviews with relatives of cancer patients

Whether truth should be told
(responses according to literacy status of respondents)

Response	*Illiterate* *n* = 22	*Low literacy* *n* = 17	*Moderately literate* *n* = 30	*Highly literate* *n* = 8	*Total* *n* = 77
To the relatives?					
Yes	13(59%)	14(82%)	28(93%)	7(88%)	62(81%)
No	5(23%)	1(6%)	2(7%)	1(22%)	9(12%)
Undetermined	4(18%)	2(12%)	—	—	6(7%)
To the patient?					
Yes	3(14%)	—	4(13%)	1(13%)	8(10%)
No	14(64%)	14(82%)	24(80%)	7(87%)	59(77%)
Undetermined	5(22%)	3(18%)	2(7%)	—	10(13%)

apprehensions and curiosity (50%) while 25% felt that he should be told so that he himself could take steps to prevent the spread of disease to other family members. Another 25% felt that sooner or later the patient would find out about his disease so one should not hide the facts from him.

An assessment of relatives' emotional reactions towards cancer patients revealed that 86% felt that they should be affectionate and give emotional support to the patient, 10% expressed their helplessness while 9% were indifferent and were just performing their duties towards the patient to meet his daily physical needs.

Discussion

It is evident from the above findings that only 48% of the patient population knew about their illness, which is quite different from those of Albeit (1959) and Peck (1972) who reported that about 80% of their subjects knew their diagnosis. A possible reason could be that these studies were conducted in western countries where the literacy rate of the general population is higher.

When diagnosis is revealed, individuals exhibit different immediate emotional reactions, of which anxiety is predominant. These findings are quite similar to those reported by Bhatt (1963), Bronner-Huszar (1971), Peck (1972), Krant (1972), Hintan (1966), and Patel *et al* (1980). In this study, although a clear depressive reaction was not seen, a feeling of dejection and helplessness was reported by 42.5% of patients. Bhatt (1963) reported such feelings in 15.4% of her patients. It is also interesting to note that other immediate reactions like guilt feelings reported by western authors (Abrams, 1951; Harley *et al*, 1951; Abrams & Finesinger, 1953; Peck, 1972) were not seen so commonly in this sample.

Bhatt (1963) reported that knowledge of illness leads to two different kinds of emotional reactions – immediate as well as delayed, and found that depression and insecurity were the most common (53%) delayed reaction in her sample. Similar results have also been found in the present sample. Possible reasons for similarities in these findings are that patients in both studies came from similar cultural backgrounds and that acceptance of disease is known to be affected by the cultural setting and prevailing attitudes towards the illness.

The attitude of relatives towards patients when they know the diagnosis was found to be most sympathetic and they paid more attention to the patients. No discrepancy was found between patient's perception and relative's expression. These findings were similar in many respects to those reported by Bhatt (1963) where 42% of patients were reportedly treated with deep sympathy by their families and friends. A feeling of being neglected was reported by only two patients in the present study. Although the knowledge of diagnosis made relatives experience anxiety and panic they reported that this did not affect their attitudes towards patients.

Most of the patients (71%) in this series who knew of their diagnosis were of the opinion that truth should be told to them. These findings support the observations of Kelly & Friesen (1950); Aitken Swan & Easson (1959); Albeit (1959); Bo *et al* (1960); Gilbertson & Wangenstten (1962); Hintan (1966); and Peck (1972).

It has also been observed that the maximum number of psychiatric symptoms can be identified in those patients who have suffered from cancer for 1–3 years (94%) or at least for more than 6 months. The frequency of psychiatric symptoms becomes less after 3 years. Probably as the time passes the patient tries to accept his life as such, and makes realistic adjustments to his illness, with altered goals in life.

The above findings indicate that Indian patients tend to accept the diagnosis of cancer rather easily. The authors feel that the concept of death prevalent in Indian culture based on the philosophy of 'Geeta' where the soul is accepted as immortal and it is believed to transfer through death from one to another human/species (Bhagwat Geeta translated by Swami Adidevanand, 1973) plays a significant role in the easy acceptance of the diagnosis and planning of the rest of life. It is probably the cultural background, close emotional family ties and the tendency in the Indian population to come together at times of crisis which cause these responses about why the truth should be told.

Concerning the questions who should reveal the diagnosis, and how and when, we subscribe to the view of Krant (1972) that communication should be the responsibility of someone close to the patient and his family. Time must be allowed for the facts to be absorbed and questions to be raised. The patient should not be told that there is nothing more than can be done for him. The way the news is broken should depend upon patient's personality, intelligence, religion and the indirect clues he provides about how he is likely to deal with the disclosure. Some patients should not be told until a strong relationship with a member of the therapeutic team has developed. Clearly a general rule of thumb has no place here.

References

ABRAMS, R. D. (1951) Guilt reactions in patients with cancer. *Cancer* 3, 474–482.

—— & FINESINGER, J. F. (1953) Guilt reactions in cancer patients. *Cancer*, 6, 474–479.

AITKEN SWAN, J. & EASSON, E. C. (1959) Reactions of cancer patients on being told their diagnosis. *British Medical Journal*, March 21, 779–783.

Albert, C. (1959) Reactions of cancer patients on being told their diagnosis. *British Medical Journal*, March 21, 779.

Bhatt, U. (1963) *Socioeconomic Problems of Cancer Patients.* Bombay: The Indian Cancer Society.

Bo, G., Gerd, L., Philip, S. (1960) The patients with inoperable cancer from the psychiatric and social stand point (A study of 101 cases). *Cancer*, 13, 1206–1209.

Bronner-Huszar, J. (1971) The psychological aspects of cancer in Man. *Psychosomatics*, 12, 133–138.

Cappon, D. (1962) Attitudes of and towards the dying. *Canadian Medical Association Journal*, 87, 693–700.

Gilbertson, V. A. & Wangensteen, O. (1962) *Should the Doctor tell the Patient that the Disease is Cancer.* New York: American Cancer Society.

Hintan, J. M. (1966) Facing death. *Journal of Psychosomatic Research*, 10, 22–28.

Harley, C., Shands, J. E., Finesinger, Stanley, C. & Abrams, R. D. (1951) Psychological mechanisms in patients with cancer. *Cancer*, 4, 1159.

Kelly, W. D. & Friesen, S. R. (1950) Do cancer patients want to be told? *Surgery*, 27, 822–826.

Krant, M. J. (1972) The organised care of the dying patient. *Hospital Practice*, 7, 101–108.

Oken, D. (1961) What to tell cancer patients? *Journal of the American Medical Association*, 175, 1120–1128.

Patel, J. J., Singh, B. K. & Gawadia, M. L. (1980) Psychological manifestations in cancer patients. *Indian Journal of Clinical Psychology*, 7, 147–150.

Peck, A. (1972) Emotional reactions to having cancer. *American Journal of Roentgenology*, 114, 591–599.

Plumb, M. & Holland, J. (1981) Comparative study of psychological functions in patients with advanced cancer – II. Interviewer-rated current and past psychological symptoms. *Psychosomatic Medicine*, 43, 243–254.

Swami Adidevanand (translator) (1973) Bhagwad Geeta, Mangalore India: Mangalore Trading Association Pvt. Ltd.

Appendix 1
Questionnaire for the relatives of cancer patients

Question 1 What do you know about cancer?

2 Suppose one person in the family has cancer and another person has some other physical illness, will the attitude of the family members be the same towards both? *Yes/No*

3 In your opinion what is society's attitude towards a cancer patient?
  - a) That he has got an illness which is incurable.
  - b) That he needs other people's sympathy
  - c) That he has some communicable disease and he should be isolated.
  - d) Any other.

4 If someone known to you has cancer what do you think the relatives should be told about it and i seriousness? *Why*

5 In your opinion should the patient himself be tol about the disease and its seriousness? *Yes/N* *Why*

6 If someone in your family has cancer what would b your reaction towards him/her?
  - a) Show your helplessness
  - b) Become indifferent
  - c) Just perform duties towards the patient to me his physical needs.
  - d) Give him emotional support/and affection
  - e) Any other.

Appendix 2
Questionnaire for patient

Question 1 What do you think you are suffering from?

2
(A) How did you come to know about it?
(B) What was your reaction when you came to kno about the diagnosis?
  - a) Did you become very serious?
  - b) Did you become very anxious?
  - c) Was your anxiety relieved?
  - d) Did you feel dejected?
  - e) Did you accept it?
  - f) Was there any other reaction?

3 Did you find any change in your behaviour after th onset of illness? *Yes/N*
If *Yes* –
  - a) Do you feel more involved in family affairs?
  - b) Do you feel uninvolved in family affairs?
  - c) Have you become irritable?
  - d) Have you lost interest in daily routine?
  - e) Are you mostly preoccupied with your illness?
  - f) Is there any other change?

4 Do you find any change in your family member' attitude towards you after the illness? *Yes/N*
If *Yes* –
  - a) Do they pay more attention to you?
  - b) Do they pay less attention than you expect?
  - c) Are they rejecting?
  - d) Are they indifferent?
  - e) Are they hostile?
  - f) Is there any other change?

5 Do you feel yourself socially cut off after the onse of illness? *Yes/N*

6 Do you find yourself physically weak after the onse of illness? *Yes/N*

7 How long do you think it should take you to get well'

8 (In case the patient knows the nature of his illness? Do you think it should be revealed to the patients tha they are suffering from cancer?

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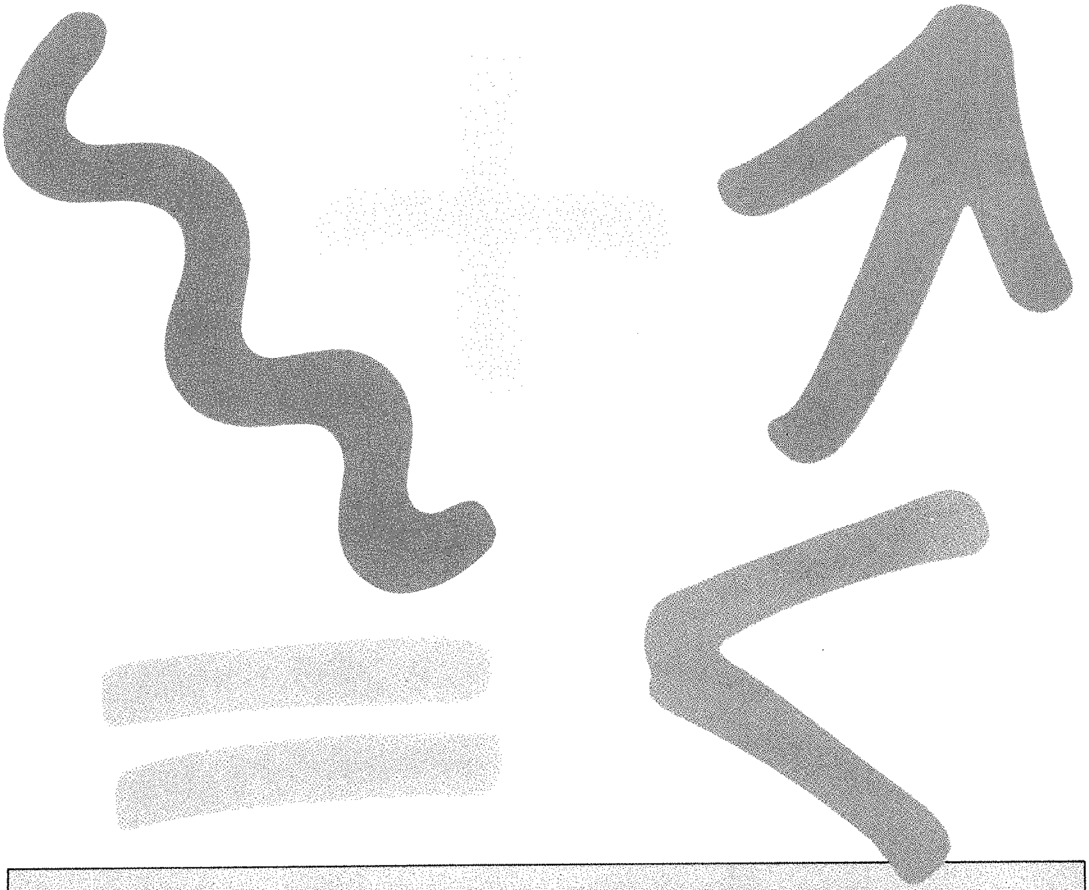

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REFERENCES

1. Johnson D A W and Malik N A. Acta Psychiat Scand 1975; 51: 257-267
2. Pinto R, Bannerjee A and Ghosh N. Acta Psychiat Scand 1979; 60: 313-322
3. Hamilton M, Card I R, Wallis C G and Mahmood M R. Psychopharmacol 1979; 64: 225-229
4. Chowdhury M E H and Chacon C. Comp Psychiat 1980; 21: 135-139
5. Haslam M T, Bromham B M and Schiff A A. Acta Psychiat Scand 1975; 51: 92-100
6. Knights A et al. Brit J Psychiat 1979; 135: 515-523
7. Kelly H B, Freeman H L, Banning B and Schiff A A. Int Pharmacopsychiat 1977; 12: 54-64
8. Based on Basic NHS prices at September 1986.

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Ethnic Differences in Self-poisoning

A Comparison of West Indian and White Groups

J. MERRILL and J. OWENS

The characteristics of West Indian patients admitted to a Birmingham hospital following deliberate self-poisoning were compared with those of White self-poisoners. West Indian patients were more likely to be young, female and single. They were less likely to have previously self-poisoned, have received previous psychiatric treatment, or be suffering from a personality disorder or alcoholism, but were more often followed-up by a social worker. Rates of self-poisoning for those aged under 25 were similar but for those aged over 25 were lower for West Indians than Whites.

Despite an abundance of American papers comparing Black and White suicide attempts (e.g. Pederson *et al*, 1973; Tardiff *et al*, 1981; Baker, 1984), few British studies have addressed themselves to this subject.

Bagley & Greer (1972) compared suicidal behaviour in 25 Blacks with controls. They found that Blacks were younger and more likely to suffer from acute situational stress. They were less likely to be sociopathic, addicted to drugs or alcohol, suffer from serious psychiatric illnesses or receive social or psychiatric support. The 'Black' group was heterogenous, comprising West Indians, Asians, Africans and Cypriots.

Burke (1976*b*) studied case notes of West Indians admitted to Birmingham hospitals after attempting suicide between 1969 and 1972. He found that West Indians were under-represented and that they were young, rarely abused drugs or alcohol and rarely repeated the attempt. He drew attention to an association between recent pregnancy and attempted suicide in West Indian females.

A pilot study from this centre (Wright *et al*, 1981) found that West Indian self-poisoners were more likely to be young, single and female than Whites. The mean annual rate of increase in self-poisoning between 1975 and 1979 was higher for West Indians than for Whites.

In the present study a comparison is made of demographic, social and psychiatric characteristics of West Indian and White self-poisoning. From such data the rate of self-poisoning is calculated for each group.

Method

The study was carried out at Dudley Road Hospital, Birmingham. The hospital serves a catchment area of 194 720, 22.3% having birthplaces outside the UK, 5.6% being born in the West Indies. Patients attending the casualty department after deliberately taking more than the prescribed dose of a medical substance, or after taking substances not intended for human consumption, were routinely admitted to the West Midlands Poisons Unit pending psychiatric assessment and completion of a comprehensive coding sheet. Between January 1 1979 and December 31 1981, 130 West Indians and 975 Whites were admitted to the Poisons Unit following deliberate self-poisoning. The West Indian (Afro-Caribbean) group consisted of 55 West Indian-born patients (42.3%) and 75 patients of West Indian parentage who were born in the UK (57.7%). The White group consisted of all whites born in England, Scotland and Wales. When patients were admitted on more than one occasion during the 3 years of the study, details of their first admission only were included. Patients of mixed ethnic origin were excluded as were patients borns in Northern and Southern Ireland, the latter because of Burke's finding that Irish-born attempted suicides in Birmingham were over-represented (Burke, 1976*a*).

Coding sheet data on the two ethnic groups were compared by means of the χ^2 test using Yates' correction for all items except the age distribution for which the Mann-Whitney *U* test was used. The two groups were well matched for social class, 87.8% of the Whites and 89.2% of the West Indians being in Social classes IV and V according to the Registrar General's classification (OPCS, 1970). Characteristics found to be age-dependent (previous psychiatric treatment, psychiatric diagnosis, psychiatric follow-up and pregnancy) were compared for those aged under 35 years only. Comparison of those aged over 35 years was precluded due to the small number of older West Indians. 'Marital problems' were recorded if, on interview, the patient indicated that difficulties in their marriage were at least part of the reason for self-poisoning. Note was made of the presence or absence of a general factor of cultural or racial conflict which here is called 'culture conflict'. This was identified only when patients volunteered information on conflict.

Overall rates were calculated for patients born in England, Scotland and Wales (UK-born) and the West Indies (West Indian-born) using the Small Area Statistics for the hospital catchment area (OPCS, 1981*b*). Age-specific rates were calculated by extrapolating from the age group/country of birth analysis for the West Midlands Metropolitan County (OPCS, 1981*a*).

Further details of patients, assessment and comparisons have been previously described (Merrill & Owens, 1986).

Results

Sex, age and civil status

See Table I. Compared with the White group (383 males and 592 females, ratio 1:1.5), the West Indian group (35 males and 95 females, ratio 1:2.7) had a significant excess of females ($P < 0.01$). The mean age of both male and female West Indians was much younger than that of the White group. This was due to an excess of older White patients and the modal age group for West Indian males being younger than for White males. Both male and female West Indians were significantly more likely to be single. When compared with White patients West Indian females were significantly less often 'divorced and separated' or widowed but the same was not true when comparing West Indian and White males.

Marital problems and culture conflict

Marital problems among those married were reported by a similar proportion of West Indians (males 66.7%, females 52.9%) and Whites (males 48.3%, females 49.1%). Culture conflict was apparent only in single females (1.4% of West Indians and 1.8% of Whites). All of these were due to parental disapproval of relationships with boys of a different colour.

Previous self-poisoning and psychiatric treatment

A previous self-poisoning was significantly more common in White patients (males 33.2%, females 34.0%) than West Indian patients (males 14.3%, $P < 0.05$, females 15.8% $P < 0.001$). West Indian females aged under 35 years were significantly less likely than Whites to have received previous psychiatric treatment (West Indians 10.2%, Whites 20.1%, $P < 0.05$). The difference for males however was not significant (West Indians 21.4%, Whites 30.5%).

Psychiatric diagnosis

Significantly fewer West Indian females aged under 35 years were diagnosed as suffering from a psychiatric illness (West Indians 17.4%, Whites 29.3%, $P < 0.05$). Among males there was no significant difference (West Indians 42.9%, Whites 23.3%). The actual diagnoses made were similar for each group, depression being the most common. Personality disorder was diagnosed in significantly fewer West Indian than White females (West Indians 6.3%, Whites 22.1%, $P < 0.005$) but not males (West Indians 14.3%, Whites 25.7%, NS). Although there was no significant difference in the incidence of alcoholism between ethnic groups when male and females were compared separately, when added together alcoholism was found to be significantly more common in White patients (West Indians 1.5%, Whites 6.6%, $P < 0.05$).

Follow-up

The type of psychiatric follow-up offered to the groups was similar. In-patient treatment was given to 5.6% of White males, 4.4% of White females, 7.1% of West Indian males and 4.5% of West Indian females. Out-patient treatment was given to 31.1% of White males, 26.2% of White females, 32.1% of West Indian males and 30.7% of West Indian females. Significantly more West Indians of both sexes were followed up by the psychiatric social worker

TABLE I

Age and civil status of self-poisoning patients compared by ethnic group

	White		*West Indian*		*Significance (White vs WI)*	
	Male	*Female*	*Male*	*Female*	*Male*	*Female*
Age						
(*n*)	(383)	(592)	(35)	(95)		
<35 years: %	66.8	66.4	80.0	92.6		
35–54 years: %	24.0	22.5	14.3	6.3	$P < 0.005$	$P < 0.0005$
>55 years: %	9.1	11.2	5.7	1.1		
Mean age: years	32.0	31.3	24.8	20.4		
Modal age group	25–29	15–19	15–19	15–19		
Civil status						
(*n*)	(364)	(575)	(32)	(94)		
Married: %	33.8	32.3	12.5	21.3	$P < 0.025$	$P < 0.05$
Single: %	50.6	47.3	84.3	74.5	$P < 0.0005$	$P < 0.0005$
Divorced & separated: %	12.4	12.3	3.1	4.3	NS	$P < 0.05$
Widowed: %	3.3	8.0	0.0	0.0	NS	$P < 0.01$

attached to the unit (West Indian males 14.3%, White males 3.7%, $P<0.025$; West Indian females 28.4%, White females 12.4%, $P<0.0005$).

Others

Other items included the incidence of self-poisoning and suicide in relatives and friends of the patient, the accumulation of debts, a history of violence by and against the patient in the previous year, and living in overcrowded conditions. None of these revealed significant differences between groups. Significantly more West Indian females had been separated from their parents before the age of 10 years. Separation from the father occurred in 23.2% of West Indians and 12.0% of Whites ($P<0.01$) and separation from the mother in 16.8% of West Indians and 9.1% of Whites ($P<0.005$). Teenage pregnancies were significantly more common in West Indians (9.1%) than Whites (1.8%, $P<0.05$) but overall pregnancy in under 35-year-old females was not significantly greater among West Indians (6.8%) than Whites (3.3%). Only three of the 13 pregnant Whites and one of the six pregnant West Indians were married; all four had marital problems.

Rate of self-poisoning

The annual rates of self-poisoning for males and females are shown in Table II. Overall rates were significantly lower for West Indian males than for White males and West Indian males aged between 25 and 34 were significantly under-represented. The annual rate of self-poisoning for females over 45 years was significantly lower for West Indians. Rates for patients aged over 25 years were significantly lower for both West Indian males ($P<0.001$) and females ($P<0.005$).

Discussion

This study provides reliable data on the prevalence of self-poisoning among West Indian-born and UK-born residents at one of Birmingham's general hospitals. Self-poisoning among males was almost twice as prevalent for UK-born as compared to West Indian-born and this finding was significant. Among females the West Indian-born prevalence was less than that for UK-born but the difference was not significant. The study compares social and demographic factors among these populations and gives data on psychiatric diagnosis and follow-up.

While much of the data used in this study is highly reliable (e.g. sex, age, marital status and follow-up) some items (e.g. cultural and marital problems, previous self-poisoning and previous psychiatric treatment) were dependent on the information supplied by the patient and others (e.g. alcoholism, psychiatric illness and personality disorder) both on the information supplied and the assessor's interpretation of it. One author (J.O.) assessed the majority of patients and supervised the assessment of the remainder.

The calculated rate of self-poisoning is subject to inaccuracies. An unknown number of patients came from outside the hospital's catchment area and some cases of self-poisoning from within the catchment area will have attended other hospitals. Census data contains information on the country of birth but not on ethnic group, the number of persons born in the UK to West Indian parents is not known. Furthermore, the UK-born group in the present study includes non-white patients of West Indian and Asian parents. It is possible that a number of West Indians were wrongly classified by place of birth but every effort was made to reduce this likelihood. It is of interest to note that among the 130 West Indian patients in the present study the male/female ratio of those born in the West Indies (17:38) is not significantly different from that of patients born to West Indian parents but of British birth (18:57). Finally, there is a small number of self-posioining patients (which in our view is less than 5% of the total) who refused hospital admission. Once again the ethnic distribution of such cases is not known.

The under-representation of West Indian males and older West Indian females, the predominance of unmarried and the decreased frequency of

TABLE II

Rates of self-poisoning (per 100 000 per year) compared by ethnic group

Age groups: years	*UK-born patients*		*WI-born patients*		*UK-born rates*		*WI-born rates*	
	Male	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
<16	25	86	1	1	44	156	606	518
16–24	130	280	6	23	392	862	352	1072
25–34	126	125	3	8	438	446	129*	257
35–44	60	79	4	4	240	318	108	93
>45	71	120	3	2	98	133	35	30*
Overall	412	690	17	38	190	299	103**	233

Significance (West Indian compared against White rates) *$P<0.05$, **$P<0.025$

alcoholism, personality disorder and previous self-poisoning support the findings of previous research (Bagley & Greer, 1972; Burke, 1976*b*; Wright *et al*, 1981).

Although pregnancy in those aged under 20 years old was significantly more common among West Indian than White patients, pregnancy rates for young West Indians in the general population are higher than for whites (Joint Working Party on Pregnant Schoolgirl Mothers, 1979). Similarly the increased occurrence of early parental separation found in West Indian patients seems likely to be a reflection of the common practice of grandparents raising children in the West Indian community and of children migrating after their parents have settled in the UK.

The increased likelihood of West Indians being followed up by the social worker is not easily explained. The social problems that West Indians had were similar to those of Whites and were commonly due to financial difficulties, poor housing and family disputes. It was felt that rather than these problems being more common in West Indians than Whites, the West Indians were perceived as being less likely to have the social support networks of friends, relatives and parents to help them cope.

Self-poisoning rates for West Indian-born and UK-born under the age of 25 were similar. However, rates for West Indian-born males and females aged over 25 years were significantly lower than for UK-born. No recent rates of self-poisoning in the West Indies are known but from Burke's finding of an overall female attempted suicide rate of 16 per 100 000 per year in Trinidad and Tobago (Burke, 1974) it seems likely that West Indians in Britain are much more likely to attempt suicide than native West Indians. The annual rates of attempted suicide among West Indians aged 16–64 years in Birmingham between 1969 and 1972 were 56 per 100 000 for males and 180 per 100 000 for females (Burke, 1976*b*). Although the rates found in the present study are higher, a reversal of the 31.5% mean annual increase in West Indian self-poisoning found by Wright *et al* (1981) has occurred. Over the three years of the present study a mean annual decrease in West Indian rates of 1.8% was similar to that of 1.2% for Whites.

Many of the characteristics of West Indian self-poisoners resemble those of Asian self-poisoners who have been compared previously with the same group of White patients in a previous paper (Merrill & Owens, 1986). Compared to Whites both West Indian and Asian groups had a preponderance of young females, lack of past psychiatric treatment and more often received social work follow-up. Further, Asian and West Indian females were both less likely to be diagnosed as psychiatrically ill or having a personality disorder. Interestingly however, culture conflict was common among Asians but not West Indians.

The findings of this research support the conclusions of the study by Pederson *et al* (1973) on White and Non-White suicide attempters in the United States that "in future research on suicide attempts, it would seem imperative to consider race as a separate factor".

Acknowledgements

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References

Bagley, C. & Greer, S. (1972) 'Black suicide': a report on 25 English cases and controls. *Journal of Social Psychology*, **86**, 175–179.

Baker, F. M. (1984) Black suicide attempters in 1980: a preventative focus. *General Hospital Psychiatry*, **6**, 131–137.

Burke, A. W. (1974) Socio-cultural determinants of attempted suicide among women in Trinidad and Tobago. *British Journal of Psychiatry*, **116**, 489–491.

—— (1976*a*) Attempted suicide among the Irish-born population in Birmingham. *British Journal of Psychiatry*, **128**, 534–537.

—— (1976*b*) Socio-cultural determinants of attempted suicide among West Indians in Birmingham: Ethnic origin and immigrant status. *British Journal of Psychiatry*, **129**, 261–266.

Joint Working Party On Pregnant Schoolgirls And Schoolgirl Mothers (1979) *Pregnant at School*. London: National Council for One Parent Families.

Merrill J. & Owens, J. (1986) Ethnic differences in self-poisoning: a comparison of Asian and White groups. *British Journal of Psychiatry*, **148**, 708–712.

Office Of Population Censuses & Surveys (OPCS) (1970) *Classification of Occupations*. London: HMSO.

—— (1981*a*) *Census 1981. County Report – West Midlands*. London: HMSO.

—— (1981*b*) *Census 1981. Small area statistics*. London: HMSO.

Pederson, A. M., Awad, G. A. & Kindler, A. R. (1973) Epidemiological differences between white and nonwhite suicide attempters. *American Journal of Psychiatry*, **130**, 1071–1076.

Tardiff, K., Sweillam, A. & Jacque, C. (1981) Suicide and race. *Journal of Psychiatric Treatment and Evaluation*, **3**, 275–278.

Wright, N., Trethowan, W. N. & Owens, J. (1981) Ethnic differences in self-poisoning. *Postgraduate Medical Journal*, **57**, 792–793.

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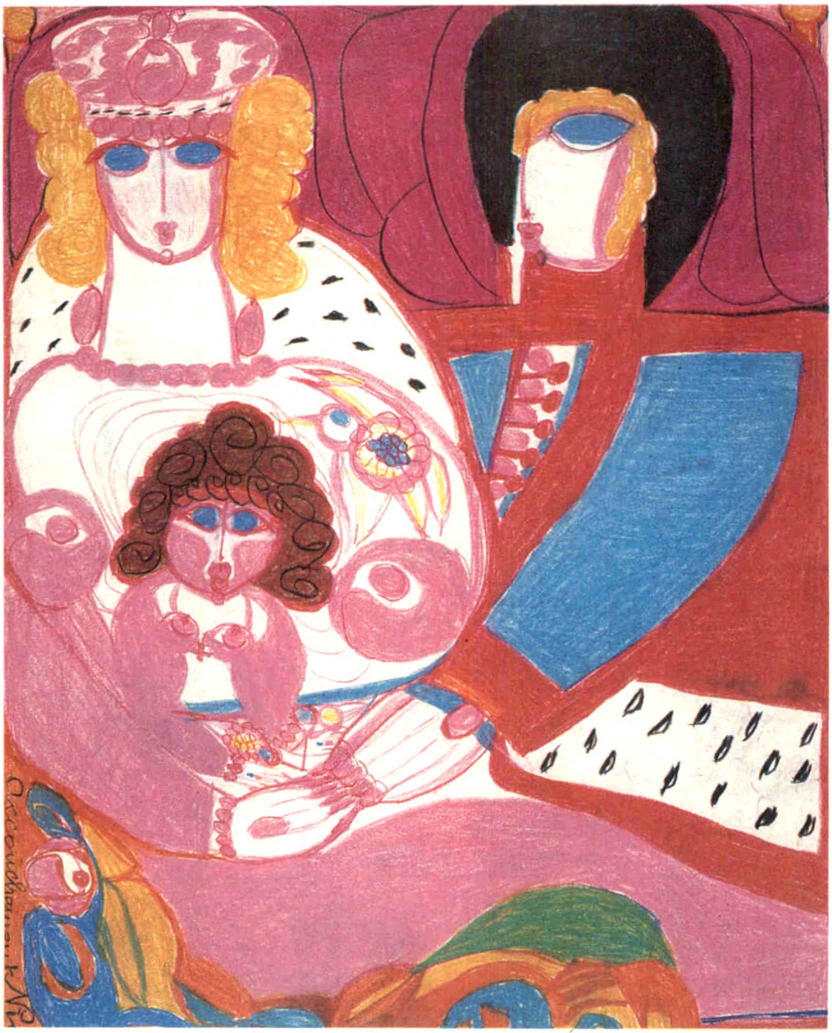

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References: 1. Pakes G. E. In 'Therapeutics Today' Ed D. A. W. Johnson. ADIS Press. (1982). 2.
2. Youssef H. A. Acta Therapeutica. (1983) **9**, 215-225

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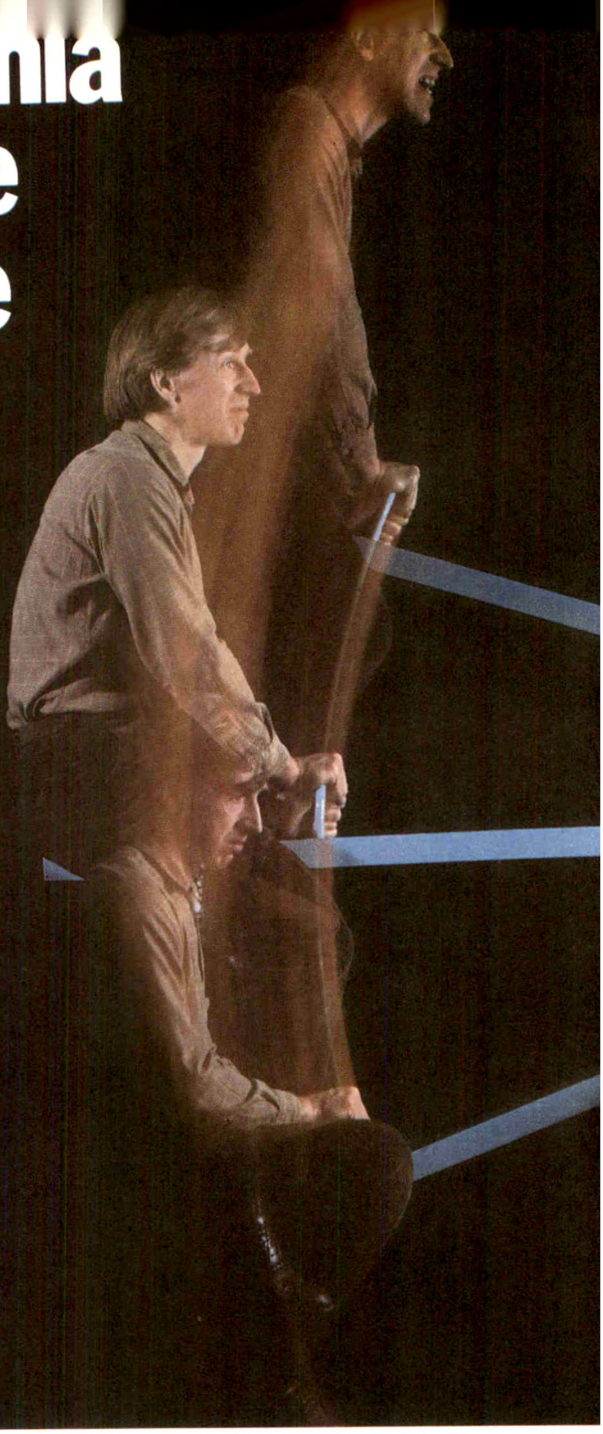

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British Journal of Psychiatry (1987), **150**, 769–773

Phenomenology of Intoxication with Toluene-based Adhesives and Butane Gas

A. C. EVANS and D. RAISTRICK

The phenomenology of solvent inhalation was investigated comparing a group of young people who misused toluene ($n=31$) with a group misusing butane ($n=12$). Marked changes of mental state were invariable. Most users reported elevation of mood and hallucinations but a rich variety of phenomena was elicited. Nearly one-quarter of subjects had the potentially dangerous delusion of believing they were able to fly or swim. In the toluene group thoughts were more likely to be slowed, time appeared to pass more quickly and tactile hallucinations were more commonly reported than in the butane group. Withdrawal phenomena and tolerance were also looked at.

Epidemiological research has shown that 3–5% of 15-year-olds in the UK have misused volatile substances at some time (National Opinion Poll, 1982; Plant *et al*, 1984). Impressions suggest that the numbers are rising and that this figure is a gross underestimate of the number currently misusing. It has been said that dependence and tolerance may develop (Press & Done, 1967; Watson, 1982) but withdrawal states are reported to be rare (Barker & Adams, 1963; Press & Done 1967) although several papers have defined a withdrawal syndrome (Lindstrom, 1960; Merry & Zaccariadis, 1962; Nylander, 1962; Ackerley & Gibson, 1964; Watson, 1979). Deaths from misuse of volatile substances in the UK have been surveyed (Anderson *et al*, 1985). A common cause of death is accident or trauma while intoxicated, but all volatile substances can cause fatality probably by direct cardiotoxicity.

There has been very little research into the phenomenology of volatile substance misuse. Press & Done (1967) wrote that the effects of solvent sniffing are greater than those of alcohol ingestion and are closer to those seen with LSD. Tolan & Lingl (1964) found that the effects of gasoline sniffing compared closely with the psychotic syndromes produced by mescaline, LSD, and psilocybin. Lawton and Malmquist (1961) reported hallucinations in gasoline sniffers as did Ackerley & Gibson (1964) in lighter fuel sniffers.

More recently, the various clinical features of solvent intoxication have been described as being similar to those of alcohol intoxication with initial stimulation followed by depression. Other symptoms are said to include euphoria, blurring of vision, tinnitus, slurring of speech, ataxia, feelings of omnipotence, headache, abdominal pain, anorexia, nausea, vomiting, jaundice, chest pain, bronchospasm, impaired judgement, irritability and excitement, and it is said that less often, a delirious state is seen with clouding of consciousness, illusions, and hallucinations (Francis *et al*, 1982; Watson, 1982; Sourindhrin & Baird, 1984). It has been reported that chronic users describe transient symptoms of toxic psychosis which often have an affective component (Skuse & Burrel, 1982). The primary reason for the present study was to produce a systematic account of the phenomenology of volatile substance misuse.

Method

The sample consists of 43 subjects, of whom 18 were the total referrals for volatile substance misuse to the Leeds Addiction Unit between January and August 1985. The remainder were drawn from the three CHE establishments (Community Homes with Education on the premises) administered by the DHSS in Leeds, for young offenders. These 25 were young people identified by the staff of the CHEs as having particular problems with volatile substance misuse.

All subjects had a 40–60 minute interview with one researcher (A.E.). Subjects were asked to recall recent episodes of intoxication and a detailed phenomenological analysis was made. This part of the interview was open-ended and as far as possible descriptions were recorded verbatim for phenomenological allocation later. Data on withdrawal symptoms and tolerance were also collected.

Results

Thirty-one subjects used toluene-based adhesives as their preferred substance and 12 used butane gas. The mean age of the toluene group was 16 years, while the mean age of the butane group was 15 years.

The majority of both the toluene (74%) and butane (67%) groups had been misusing for more than a year, putting them into the chronic class of volatile substance abuser. Nearly 50% of both groups had misused on a daily basis. All subjects had experimented with several solvents before deciding on the preferred one.

Phenomenology

The results are summarised in Table I.

Reports on general behaviour and talk are based on observations of others as well as on subjective experiences. Most users said that they could make phenomena occur if they thought about them, but that experiences were also spontaneous.

General behaviour

Behaviour was reported to have been disturbed in the majority of cases and most said that they behaved as though they were drunk. Those who said they were in control of themselves were usually the more experienced users.

Talk

In general, talk was either slow and slurred or faster than normal, the latter being commonest with the toluene group. A word frequently used by them to describe their speech was "blurred". Other descriptions used were "missing letters out", "talking in unknown language", "baby talk", "voice sounds like someone else".

TABLE I

Summary of phenomena experienced during intoxication with toluene and butane

	Toluene (*n=31*) %	*Butane* (*n=12*) %	
General behaviour changed	70	75	
Talk slow	58	50	
Talk fast	23	8	
Thoughts slow	48	17	
Thoughts fast	13	33	
Mood – elated	97	100	
Mood – depressed	29	17	
Mood – fearful	65	42	
Illusionary misinterpretations	74	50	
Tactile hallucinations	42	8*	$\chi^2=4.449$
Auditory hallucinations	87	75	
Visual hallucinations	97	83	
Gustatory hallucinations	3	0	
Time slow	23	67**	$\chi^2=7.402$
Time fast	55	0***	$\chi^2=10.88$
Colours bright	26	8	
Colours dark	26	25	
Perception of body form	71	58	
Micropsia	19	17	
Macropsia	26	25	
Delusional ideas	55	33	
Delusions of control	19	0	
Delusions of flying	16	25	
Delusions of swimming	3	0	

*$P<0.05$; **$P<0.01$; ***$P<0.001$.

Thought

Thoughts were most commonly slow and difficult in the toluene group or fast and easy in the butane group. Phrases used to describe the fast form of thought were "think a lot better", "just so much easier", "as if you're really brainy", "thoughts feel free".

Words used to describe slowness and difficulty in thinking were "wasn't able to think", "thoughts blocked by something", "mind went blank", and "thoughts slipping away – like a vapour going through my brain". One patient said "I say it in my mind and like an amplifier in my mind it echoes for about ten minutes".

Mood

Mood was nearly always altered and often differently in the same session. Most users experienced getting high which they described as "feeling good", "no worries", "feel brilliant". Many users experienced unpleasant mood states, especially fear. The fluctuations in mood were invariably a response to the content of other abnormal experiences.

Illusionary misinterpretations

There was often no clear distinction between illusionary and hallucinatory phenomena. Readily available objects were commonly the catalysts for an illusionary creation, which could develop to an hallucination. Thus one youngster said that trees "become scarey, the branches go all curvey and seem like they're grabbing out to get you. They look like they're alive and their notches look like eyes and mouths". A girl said that when she looked in the fire she saw hands and animals and faces coming out of the flames. Another description was "the gravestones changed into orange aliens with big, round heads, pointed ears and one eye in the centre. They came towards me. I shot them with a long white gun with two buttons on the side. The trees changed into spaceships, long silver things, with three legs and a door". A boy described wall paper patterns moving around like footballers knocking balls to each other. His friend saw it first, then told him about it and then he could see it. Another said "I take my toy soldiers, that's the best thing. I put them on the floor and they grow and talk to me, or they stay small and walk about on my hand. I had a toy ambulance and it grew to its proper size and two plastic men grew and became proper men and put me in the back to take me away because I was glue sniffing".

Glue bags were frequently reported to change shape – "my bag changed into a hat – I put it on my head and had glue all over me." "I was sitting in front of a wall. A big shiny egg appeared in the glue bag; it was talking. A hole came in the wall and I pushed the egg through. My teacher walked in – she looked like a rabbit; she asked 'Have you got the Queen's birthday present?' She sent me home for glue sniffing. I was Alice in Wonderland." Other objects often altered similarly. "My bed changed into a dog, and barked." "I saw the TV moving towards me - it tried to eat me." "I look on a blank wall and draw on it and it comes alive. A house I drew came alive and came out and got big. I went in to the house and had fun. There were lots of toys inside and I played."

Hallucinatory experiences

Tactile hallucinations were much commoner in the glue group. A girl said "something came into my throat - a long, greasy, slimy worm trying to climb out". A boy described sitting on a sofa, "I could feel knives sticking in my legs and my back".

Auditory and visual hallucinations were experienced by nearly all youngsters in both groups. A buzz was heard by most of them when they first began sniffing, but this commonly wore off after the first few weeks and is not included as an auditory hallucination. Several people reported that trees started talking to them. A girl heard voices talking about what she was doing and telling her to do things. "You are sitting on the bed, you are standing up, get up, pack your bags". Another girl heard a voice in a graveyard telling her to cut her wrists; she did so.

The visual hallucinations described were florid and colourful. A boy saw a little cat putting its head through the fence. "I looked over the fence and saw loads of witches with black hats and cloaks in a garden running around. They were having a fancy dress party. I asked where the cat was and they said I was it. I looked down and I had a tail and was black. I was a cat. I started screaming and ran and jumped over the fence".

One glue user saw how the Universe was formed. "It was like a little ball dropped out of someone's pocket and there was something like an electric light bulb which gave day and night." Another always saw a white box in front of his face telling him what he was going to dream about. "It is about 2 × 3 inches and has words printed on it like 'you are going to dream about spaceships.'" A boy thought he was walking through a Chinese market and down a Chinese street. He saw Bruce Lee and fought with him. A girl thought she was in a garden sunbathing. "The trees came to life and came over to me. The grass wrapped itself around me." Stories of graves in cemeteries opening up and things coming out were frequent. Several saw demons and werewolves appearing out of the graves. Tales of spaceships and little green men were also heard often.

A glue user thought he was in Paris at the Eiffel Tower. "I climbed up and fell off. There was a fellow with an umbrella to catch me. The umbrella opened or closed depending on whether or not I opened or closed the glue bag." Once his bedroom was on a turntable. "It went round slowly, and went into another time when there was slavery. There were Roman men in leather armour trying to make us slaves. We escaped by climbing through the ceiling and through the drawers and back into the bedroom". He also said "Ernie told us he had just been to China. We all tried to go but couldn't. Then four of us got to China at the same time. Then six of us got to China at the same time. Our spirits came out of our bodies and we watched our bodies being chased, by Samurai warriers." Several children reported seeing space invaders. A boy said "I used to fire at them with lasers coming out of my eyes. They would then disappear". Others had lasers coming from their heads or bullets coming out of their minds.

Time perceptions

Slowing of time was commoner in the butane group but time passed more quickly in the toluene group. A boy using glue felt that he could control time as on a video machine by saying "Stop, forward, rewind, and fast forward". Another boy said that time went so slowly during 15 minutes sniffing that when he stopped he thought it was the next day.

Colour perception

Colour perception was distorted in over a third of the two groups and often a darkening of colours was described. "An orange colour became light brown", "brown wood went black". It was more often in the toluene group that everything became lighter and brighter. Individuals reported that "silver paint went nearly white", "grey graves went orange," "trees went silvery". "Everything's brighter and lighter - fresh like first thing on a morning when you have been for a run."

Perception of body form

There were many reports of the body feeling lighter. "I feel half my size, as though I'm floating". Less common were reports of "feel heavier". Other more complicated experiences were "I go big and small in time with the glue bag. Once I dropped my glue bag in thorns and I couldn't get it out when I was big, but I could when I was small".

Perception of size

Approximately a quarter of the subjects said that things around them became larger and rather fewer said that things became smaller. Descriptions included, "everything seems bigger - on top of flats looks a long drop down", or "people get bigger, about twice the size of me", or "my mates get smaller; I am always having to look down at them".

Delusions

Delusional ideas were experienced most commonly among the toluene users. Two were convinced they were dead during sessions. A boy thought he had changed into his best friend. Another boy thought that the devil was operating everything from an underground city where UFOs were buried. One boy was convinced that he had the power to change the weather around. A girl thought that her boyfriend had been killed by her best friend and she tried to strangle her best friend. Another believed that her grandfather had died, another that she could walk through things and a boy said he could get rid of people by "glazing" them, that is he looked through them and they disappeared.

Delusions of control were experienced with glue but not with gas. One boy felt there was a force making him do the things he did when intoxicated, and another felt that he was being controlled by the devil and by God. "I knew that if I didn't jump over cracks in the pavement I would go to hell. I wanted to go to heaven – there were points for the devil and points for God. Sometimes I looked into the mirror and was trying to change into the devil. I knew that if I couldn't do it, the devil got points and I went to him."

Eight children had had the delusion of being able to fly and one girl had thought she was a fish and could swim, (she couldn't). "Once in a cemetery I knew I was Batman. I had to jump out of a tree. I was laughing on the ground". Another boy jumped out of a first floor window and fell on a mattress; he had thought his friend was flying and fell when he tried to join him. Others said "I thought I was superman and I tried to fly out of a tree but my friend stopped me", and "I thought I could fly so I jumped off the top floor of a three storey block of flats; I broke my ankle".

None of the youngsters who thought they could fly suffered any serious injury though all had minor injuries and indeed potentially fatal outcomes. Similarly, the girl who thought she could swim came close to death. "I thought I was a fish and could swim very well. I jumped in the lake and nearly drowned". The subjects had a total of eight friends who had also thought they could fly and one friend who had thought he could walk on water.

Withdrawal phenomena and tolerance

There was clear evidence that tolerance had developed in 71% of the toluene cases and 100% of the butane cases. A history of withdrawal syndrome similar to that of central nervous system depressants was elicited, in 62% of the toluene cases and 50% of butane gases. The prominent features were sleep disturbance, irritability, shakiness, sweating, fleeting illusions and nausea. Stomach cramps, lower chest pain and facial tics were also consistently reported. There was a correlation between frequency of symptoms reported and frequency of solvent use (toluene $r=0.58$, $P<0.05$, butane $r=0.57$, $P<0.001$).

Withdrawal symptoms usually took 24–48 h to emerge after solvent use had been discontinued, and they lasted for between 2 and 5 days.

Discussion

Intoxication with toluene-based glue or butane gas presents with a clinical picture of a toxic confusional state. There is general depression of the central nervous system, disorientation and perceptual changes from which secondary mood states and delusional systems may arise. Florid symptoms subside within minutes for butane and half an hour for toluene. The characteristic odour of solvents, circumstances of use and subsequent course will rarely leave doubt about the diagnosis. The picture may be complicated by concurrent drug use or other psychiatric disorder; these problems were not encountered in this study. In the acute phase when elevation of mood and overactivity are prominent features, intoxication may be mistaken for manic-depressive disorder and when auditory hallucinations and paranoid ideas dominate, the mental state may be misdiagnosed as indicating schizophrenia.

Both solvents studied here are highly plastic substances and it was apparent that users are able to create illusions and hallucinations almost at will. 'Sniffing' was associated with very positive expectancies by users and initial elation of mood and pleasant perceptual change was almost invariable. The continued use of a solvent was often associated with a release of internal conflicts and, in particular, unresolved feelings about death about their parents. Compared to intoxication with cannabis and LSD, persecutory thoughts were uncommon though one young girl did try to strangle a friend. Fearful mood was most usually a response to frightening hallucinations. The content of abnormal experiences was strongly influenced by the dominant TV programmes and films to which the young people were exposed.

There were interesting differences between reported effects of toluene and butane though these will need confirming using matched comparison groups. In this study the toluene group most often reported thoughts being slowed but time passing quickly while the opposite was the case for the butane group. The toluene group were more likely to experience paranoid ideas that were not directly related to perceptual change but nonetheless emerged from the intoxicated state. Taken in overview the toluene group experienced more phenomena than the butane group, but then they experienced longer periods of intoxication.

The forensic investigation of death in young people does not include routine testing for the presence of solvents and the authors believe that there may be many more solvent related fatalities than officially recorded. This raises the argument of counselling towards safe use of solvents in young people who are judged to be unwilling to stop their habit.

In this study neuroadaptive change, notably withdrawal symptomatology, was only assessed retrospectively and indeed it would be difficult to do otherwise given the nature of solvent use. The symptoms were essentially similar to withdrawal from central nervous system depressant drugs with the notable addition of stomach and chest pains, and facial tics. The implication is that admission to hospital for detoxification may be indicated more frequently than previously thought.

In conclusion this study has highlighted several aspects of solvent misuse which have not previously been considered in depth and which we believe have important implications for management of these cases.

References

ACKERLY, W. C. & GIBSON, G. (1964) Lighter fuel "sniffing". *American Journal of Psychiatry*, **120**, 1056–1061.

ANDERSON, H. R., MACNAIR, R. S. & RAMSEY, J. D. (1985) Deaths from abuse of volatile substances: a national epidemiological study. *British Medical Journal*, **290**, 304–307.

BARKER, G. H. & ADAMS, W. T. (1963) Glue sniffers. *Sociology and Social Research*, **47**, 298–310.

FRANCIS, J., MURRAY, V. S. G., RUPRAH, M., FLANAGAN, R. J. & RAMSEY, J. D. (1982) Suspected solvent abuse in cases referred to the poisons unit, Guy's Hospital, July 1980–June 1981. *Human Toxicology*, **1**, 271–280.

LAWTON, J. J. & MALMQUIST, C. P. (1961) Gasoline addiction in children. *Psychiatric Quarterly*, **35**, 555–561.

LINDSTROM, F. (1960) Delirium tremens som abstinssymptom vid thinner-sniffning. *Svensk Lakartidn*, **57**, 2214–2219.

MERRY, J. & ZACCARIADIS, N. (1962) Addiction to glue sniffing. *British Medical Journal*, **ii**, 1448.

NATIONAL OPINION POLL. (1982) *Daily Mail*, January.

NYLANDER, I. (1962) "Thinner" addiction in children and adolescents. *Acta Paedopsychiatrica*, **29**, 273–283.

PLANT, M. A., PECK, D. F. & STUART, R. (1984) The correlates of serious alcohol-related consequences and illicit drug use among a cohort of Scottish teenagers. *British Journal of Addiction*, **79**, 197–200.

PRESS, E. & DONE, A. K. (1967) Solvent sniffing – physiological effects and community control measures for intoxification from the intentional inhalation of organic solvents. Parts I and II. *Pediatrics*, **39**, 451–461; 611–622.

SKUSE, D. & BURREL, S. (1982) A review of solvent abusers and their management by a child psychiatric out-patient service. *Human Toxicology*, **1**, 321–329.

SOURINDHRIN, I. & BAIRD, J. A. (1984) Management of solvent abuse: a Glasgow community approach. *British Journal of Addiction*, **79**, 227–232.

TOLAN, E. J. & LINGL, F. A. (1964) "Model psychosis" produced by inhalation of gasoline fumes. *American Journal of Psychiatry*, **120**, 757–761.

WATSON, J. M. (1979) Glue sniffing: two case reports. *Practitioner*, **222**, 845–847.

—— (1982) Solvent abuse: presentation and clinical diagnosis. *Human Toxicology*, **1**, 249–256.

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Patterns of Use and Related Harm with Toluene-based Adhesives and Butane Gas

A. C. EVANS and D. RAISTRICK

The patterns of use and related harm of solvent inhalation were investigated in a group of young people who misused toluene ($n = 31$) and a group misusing butane ($n = 12$). The two groups were similar in terms of patterns of use and solvent related harm. The toluene users were more likely to sniff only in a group setting, probably because of the long duration of intoxication. The toluene group were more approving in their attitudes towards taking other drugs.

In the UK, 3–5% of 15-year-olds are said to have misused volatile substances at some time (National Opinion Poll, 1982; Plant *et al*, 1984). It has been reported that the activity predominates in boys in the 13–15 age-group and that it is usually a group activity (Masterton & Sclare, 1978; Watson, 1982; Sourindhrin & Baird, 1984). Some studies show a high incidence in children from single-parent families with paternal unemployment (Masterton & Sclare, 1978; Sourindhrin & Baird, 1984); others refute this (Press & Done, 1967; Kaufman, 1973).

A number of papers have studied the adverse physical consequences of volatile substances misuse. Antisocial and self-destructive acts occur (Press & Done, 1967; Skuse & Burrel, 1982), and there is often associated misuse of alcohol and multiple illicit drugs (Skuse & Burrel, 1982). The progression of addiction careers in young adult solvent misusers has been commented upon and research is under way to identify the characteristics of the minority who go on from volatile substances to the misuse of alcohol and illicit drugs (Davies *et al*, 1985). Deaths from misuse of volatile substances in the UK have been surveyed (Anderson *et al*, 1985). A common cause of death is accident or trauma while intoxicated, but all volatile substances can cause fatality – probably by direct cardiotoxicity. The aim of this study was to investigate patterns of use. We also assessed related harm. Information was gathered during a study on the phenomenology of solvent abuse. The paper is not intended as a comprehensive study of patterns of use and related harm.

Method

The sample consists of 43 subjects, of whom 18 were the total referrals for volatile substance misuse to the Leeds Addiction Unit between January and August 1985. The remainder were drawn from the three CHE establishments (Community Homes with Education on the premises) administered by the DHSS in Leeds. These 25 were young people identified by the staff of the CHEs as having particular problems with volatile substance misuse.

All subjects were interviewed by one researcher (A.E.). Semi-structured sheets were used to collect data about home circumstances, background and pattern of substances misuse. Subjects also completed an attitude questionnaire. The attitudes to various drugs were assessed using a 10 cm line method to tap the constructs 'safe - dangerous' and 'enjoyable not enjoyable'.

Results

Social characteristics

Thirty-one subjects used toluene-based adhesives as their preferred substance and 12 used butane gas. The mean age of the toluene group was 16 (range 13–19) and male:female sex ratio was 4:1, while the mean age of the butane group was 15 (range 10–19) and sex ratio was 1:1.

The social characteristics of the cohort are undoubtedly distorted by the sampling method, and in particular both groups lived mainly in institutions or hostels (toluene 77%, butane 67%). There is a high incidence of family trauma, with separation of true parents, single parent families and periods of care from an early age. This was more marked for toluene users than the butane users. It is noteworthy that a number of children, particularly in the glue group, had found the transition from middle school to high school at 12–13 years a difficult one, often on account of the fact that they found themselves separated from friends they had made at middle school.

Pattern of misuse

The majority of both the toluene (74%) and butane (67%) groups had been misusing for more than a year, putting them into the chronic class of volatile substance abuser. Nearly 50% of both groups had misused on a daily basis. All subjects had experimented with several solvents before deciding on the preferred one. Thus, aerosol sprays, petrol, nail polish, nail polish remover, paint thinner, correcting fluid, and boot polish had been tried and rejected.

As the survey progressed it became clear that the toluene-using cohort represented a hard core of users. Many of the users knew each other before being institutionalised. They often lived in quite separate parts of the city but would meet

TABLE I
Patterns and consequences of solvent use

	Toluene *n=31*	*Butane* *n=12*
Frequency of use in previous 6 months		
Daily	48%	50%
2–6 days weekly	26%	25%
1 day or less weekly	26%	25%
Duration of use		
<6 months	13%	25%
6–12 months	13%	8%
>12 months	74%	67%
Consumption on heaviest day		
range	0.25–3.5 pints	1–15 cans
mean	1.3 pints	3.9 cans
Circumstances of use		
solitary use	10%	50%* $\chi^2=8.49$
solitary and group	16%	33%
group only	74%	17%** $\chi^2=11.7$
Solvent-related accidents	36%	33%
Solvent-related offending	100%	83%
Time off school or work	71%	50%
Regular cigarette smoking	100%	83%
Regular drinking	26%	25%
Use of other illicit drugs	48%	33%

*$P<0.005$; **$P<0.001$.

TABLE II
Attitudes of toluene and butane groups to various substances

	Toluene (n=31)		*Butane (n=12)*	
	% Safe	*% Enjoyable*	*% Safe*	*% Enjoyable*
Alcohol	54	89	40	60
Cigarettes	39	92	40	60
Cannabis	19	65	10	20
Amphetamine	15	62	10	20
LSD	15	62	10	20
Heroin	4	39	0	20
Solvents	8	92	20	50

in a group to take solvents. Details of the pattern of use are presented in Table I.

A criminal record, usually of theft to support the habit, was almost universal among the misusers. The number having accidents was the same in both groups, despite the very different times taken for the effects of the substances to wear off. Intoxication with toluene takes at least 30 mins for the effects to subside, in contrast to only a few minutes with butane. It is noteworthy that 74% of the glue sniffers only indulged in a group setting. Many said that they were afraid of the consequences if they did it alone. On the other hand, 83% of gas sniffers sniffed alone at least some of the time, again indicating the essentially different natures of the practices.

More people in the toluene group than the butane group had experimented with other groups of drugs. These were most likely to be cannabis, amphetamines and magic mushrooms. None could afford heroin. Smoking was almost universal among the group. Drinking regularly was more common among the toluene group than the gas group, as was regularly taking time off school or work in order to misuse their substance.

Attitudes

The results of attitudes to other drugs are presented in Table II. It is interesting that the toluene group in particular regard their habit as unsafe (92%) but enjoyable (92%) and that this group favours alcohol as being safe (54%) and enjoyable (89%). This finding supports the view that most 'glue sniffers' will mature out of the habit and move on to drinking alcohol. Both groups had powerfully negative views about heroin and, to a lesser extent, other 'hard drugs'. All subjects were also asked to complete a section on their attitudes to work. Every individual in the survey thought that work was a good thing and 77% of the toluene and 90% of the butane group thought work would be enjoyable. A majority of both groups thought that their lives would be shortened on account of their solvent abuse and that their happiness in the future would be diminished because of it.

Discussion

The characteristics of the cohort studied are unlikely to be those of solvent misusers generally. Selecting young people referred to the Addiction Unit or resident in Local Authority institutions might be expected to bias the sample in favour of the most problematic users and this impression of a 'hard core' of users was supported by the fact that many of the young people, particularly in the toluene group, knew each other and had become acquainted only through their connection with solvent misuse gangs in Leeds. It was not surprising to find chaotic family backgrounds and social deprivation although this was less marked in the butane group. The most striking demographic difference between the two groups is the male:female ratio which was 4:1 for toluene and 1:1 for butane. It may be that the mess and persistent smell of toluene adhesives is unattractive to females compared to the quick, clean and short-lived odour of butane gas.

After some experimentation all users settled for a preferred solvent, but contrary to perceived patterns of use elsewhere in the country, none used aerosols as first choice and less than one-fifth had ever tried them. The cost and dangers of aerosols were given as reasons.

There was a significant difference in group – only use for toluene users and this seemed related to the prolonged period of intoxication associated with these solvents and the recognition by the users of the dangers of accidents or untoward behaviour while intoxicated. In spite of the different elimination rates, the number of users involved in solvent-related accidents was the same for both groups, namely one-third; accidents were both the result of behaviour while intoxicated and acting on delusional beliefs. The frequency and potential seriousness of solvent-related accidents has not generally been properly recognised. These results suggest that much more strenuous efforts should be made to prevent solvent inhalation rather than accepting, as most workers do now, that the youngsters will grow out of the habit unharmed.

There has been speculation that use of solvents might lead to use of hard drugs and in order to contribute evidence to this debate some measures of attitudes of this 'hard core' group were taken. The views of youth workers and involved professionals are that solvent misuse is something that the great majority of young people mature out of. Almost all subjects believed heroin to be dangerous and it was the drug fewest (toluene 39%, butane 20%) thought would be enjoyable. The drugs thought to be safest and most enjoyable by both groups were alcohol and nicotine, though two-thirds of the toluene group also expected enjoyable effects from illicit drugs other than heroin. The results suggest that these young people will turn to alcohol and cigarettes as their recreational drugs when they can afford them but both groups are, nevertheless, clearly vulnerable to an increase in availability of illicit substances, particularly if alcohol and cigarettes are found wanting in terms of psychoactive properties. Predicting the future behaviour of these young people was not the purpose of this paper. However, the fact that all of the toluene group and 83% of the butane group reported that they were already regular cigarette smokers and a quarter of both groups reported regular drinking is a matter for concern. This, taken with the very high rates of offending, some use of illicit drugs and time missed from school, should alert professionals to a group of socially and possibly physically damaged young people whose difficulties will not be resolved simply as a result of maturing out of solvent misuse. This applies particularly to toluene misuse. In our view, chronic toluene misuse requires careful investigation and follow-through by health and social workers.

References

ANDERSON, H. R., MACNAIR, R. S. & RAMSEY, J. D. (1985) Deaths from abuse of volatile substances: a national epidemiological study. *British Medical Journal*, **290**, 304–307.

DAVIES, B., THORLEY, A. & O'CONNOR, D. (1985) Progression of addiction careers in young solvent abusers. *British Medical Journal*, **290**, 109–110.

KAUFMAN, A. (1973) Gasolene sniffing among children in a Pueblo Indian village. *Pediatrics*, **50**, 1060–1064.

MASTERTON, G. & SCLARE, A. B. (1978) Solvent abuse. *Health Bulletin (Edinburgh)*, **2**, 305–309.

NATIONAL OPINION POLL. (1982) *Daily Mail*, January.

PLANT, M. A., PECK, D. F. & STUART, R. (1984) The correlates of serious alcohol-related consequences and illicit drug use among a cohort of Scottish teenagers. *British Journal of Addiction*, **79**, 197–200.

PRESS, E. & DONE, A. K. (1967) Solvent sniffing – physiological effects and community control measures for intoxification from the intentional inhalation of organic solvents. Parts I and II. *Pediatrics*, **39**, 451–461, 611–622.

SKUSE, D. & BURREL, S. (1982) A review of solvent abusers and their management by a child psychiatric out-patient service. *Human Toxicology*, **1**, 321–329.

SOURINDHRIN, I. & BAIRD, J. A. (1984) Management of solvent abuse: a Glasgow community approach. *British Journal of Addiction*, **79**, 227–232.

WATSON, J. M. (1982) Solvent abuse: presentation and clinical diagnosis. *Human Toxicology*, **1**, 249–256.

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Bulimia Nervosa

The Impact of Pregnancy on Mother and Baby

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This study examines the impact of pregnancy on the reported eating behaviour of 20 untreated normal body weight bulimia nervosa women; it also reports foetal and obstetric abnormalities and indicates the initial eating habits of the infants. The prevalence of binge-eating and self-induced vomiting reduced sequentially during each trimester of pregnancy. By the third trimester 15 women (75%) had stopped all bulimic behaviour and in the remainder the disturbed eating was less severe. Symptoms tended to return in the puerperium and in nearly half the sample abnormal eating was more disturbed after delivery than before conception. However, the improvement associated with the pregnancy described by seven patients was maintained and for five it appears to have been curative. The common fear among pregnant bulimics that their abnormal eating behaviour may damage their unborn child cannot be dispelled by this study; the incidence of foetal abnormality (including cleft palate and cleft lip), multiple pregnancies and obstetric complications (including breech presentation and surgical intervention) was high. The nutrition and development of the infants was good although three mothers (15%) reported slimming their babies down within the first year.

Early morning sickness, nausea and faddism are often reported concomitants of pregnancy. However, the incidence of these behaviours in pregnant women with bulimia is unknown. Certain aspects of bulimia, particularly the self-induced vomiting (Russell, 1979), the major fluctuations of calorific intake (Lacey & Gibson, 1985), the concern about shape and weight (Birtchnell *et al*, 1985) and the often associated fluctuating dysphoria, lead to the premise that the normal body weight bulimic could be liable to major difficulties during pregnancy and lactation. Further, because of the high co-prevalence of eating disorders in the families of bulimics (Hudson *et al*, 1983) the impact of the patient's eating disorder on her child's initial feeding warrants examination.

The aim of this study is to examine the impact of pregnancy on the dietary difficulties of the normal body weight bulimic woman and to report the eating habits of her child during the first year of life. The relevance of such a study is heightened by two further points. First, the finding of the first author (Lacey *et al*, 1986*a*) that 9% of the patients presented at his clinic do so, inter alia, because they desire to have a child but believe that binge-eating, if left untreated, could damage the unborn infant. Second, and perhaps consequentially, there appears to be a tendency for such women to become pregnant at the end of treatment (Lacey, 1983; Lacey & Gibson, 1985).

Method

All patients were attending the first author's Eating Disorders Clinic. The sample consisted of 20 consecutive bulimic women, of normal body weight, who reported having given birth within 2 years of the initial assessment but prior to receiving any treatment. All patients fulfilled the criteria for bulimia as laid down by the St George's Clinic (Lacey *et al*, 1986*a*, 1986*b*) and DSM-III (American Psychiatric Association, 1980).

A full psychiatric, obstetric and gynaecological history was taken. The patient then completed a questionnaire and any uncertainties were checked on face-to-face interviews. The questionnaire consisted of five sections, each of which in sequential order, dealt with a stage of pregnancy or the puerperium. The first stage – which we called 'pre-conception' – referred to the 3 months prior to known conception; the second, third and fourth sections dealt with the three trimesters of pregnancy, and the fifth section dealt with lactation, weaning and the infant's subsequent eating habits. As far as possible questions were asked such that the replies were either numerical or 'yes/no'. Space was provided after each question for the patient to amplify her answer. If the patient had had more than one child, she was asked to base her answers on her last pregnancy.

Results

Age of delivery ranged from 23–34 years, with an average of 28 years. Sixteen patients were primagravida; of the remainder, only one had more than one previous child. Seventeen patients were married, one was separated (her husband being the father of the child) and two were unmarried. Including the separated woman, three were living apart from the father of their child. The median social class was Social Class III.

Clinical state

All patients described current episodes of distressful binge-eating (mean = 2.5/day s.d. = 1.1). All had a well-established binge-eating pattern (duration from 3–22 years).

The patients reported the following behaviours currently associated with their binge-eating. All 20 engaged in self-induced vomiting and four also abused laxatives. All were attempting to lose small amounts of weight, on average, desired weight being half a stone (3.2 kg) less than presenting weight. Eleven patients had experienced fluctuations in body weight which distressed them. No patient felt an irrational fear (phobia) of normal body weight; their mean body weight was 106% of their matched population mean weight (MPMW), the range being 94–115% MPMW. All realised that their eating pattern was out of control and 16 had at some point felt a lack of control which had extended beyond food abuse into other areas of their life, such as interpersonal relationships. All felt self-depreciatory thoughts and 17 had felt depression in association with eating binges.

Fourteen patients had Type I bulimia (Lacey, 1984*a*) i.e. they did not describe a history of previous anorexia nervosa, weight phobia or massive weight loss. Five patients described Type II bulimia, i.e. they had a previous history of anorexia nervosa but had 'recovered' to normal body weight and no longer had a weight problem. One patient had Type III bulimia, i.e. she gave a history of massive obesity being some 50% above MPMW. Her weight had been within a normal range prior to conception and during the 18 months since delivery. The number of Type II bulimics in the sample (25%) was somewhat less than the number (34%) who regularly present at our clinic.

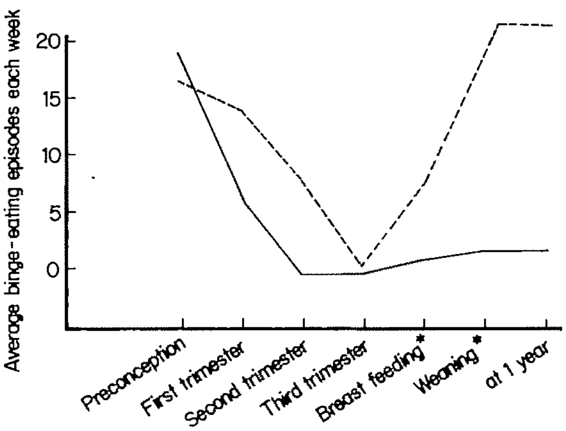
The eating disorder and pregnancy

The number of women who were binge-eating and vomiting reduced sequentially during each trimester of pregnancy but increased again in the puerperium (Table I). It can be seen that only five patients were binge-eating during the final trimester and in four of these the frequency of bingeing was less than in the first 3 months of pregnancy. In fact, 19 of the 20 subjects reduced the frequency of binge-eating over the course of their pregnancy, the exception increased the frequency of her binge-eating, but decreased the frequency of self-induced vomiting and stopped abusing laxatives.

TABLE I
Number of patients binge-eating in each 3-month period

Before conception	20
First trimester	16
Second trimester	14
Third trimester	5
Puerperium	12

All 20 patients reported that they were determined to improve their eating during pregnancy. Three patterns of response were found. Nine patients showed a reduction in the frequency of binge-eating during pregnancy with an increase afterwards to a level in excess of that occurring in the pre-conception period (Fig. 1). Four more patients showed a similar pattern but the frequency during lactation and weaning was not in excess of the frequency before conception. Most interestingly, however, seven of the patients who had stopped binge-eating during pregnancy maintained this improvement not only during lactation but also during weaning, and, in the case of five of them, this improvement had continued for over a year after confinement. These five patients were either pregnant when first assessed or became pregnant shortly afterwards: none have needed formal treatment.



*Sample size = 19 patients (one baby died)

FIG. 1 The changing pattern of binge-eating in seven women (-) who maintained their improvement and 13 who returned to bulimia (—).

The pattern and frequency of self-induced vomiting is similar to the binge-eating data above with the exception of the one patient already described. Four patients abused laxatives. All stopped using them as soon as they discovered they were pregnant but all had returned to laxative abuse either during weaning or bottle feeding. In two cases the amount of laxative abuse was higher during the puerperium than prior to conception. Alcohol abuse was described by three patients. Only one continued to drink during the first trimester of pregnancy and she returned to alcohol abuse after delivery.

Prior to conception the four most preferred binge foods were in order: starchy foods, sugars, 'junk' food and cheese. Little change occurred during pregnancy apart from cheese being more abused among those women who bottle-fed.

Other findings

Prior to conception nine of the patients described an irregular menstrual cycle, although none were amenorrhoeic.

Five described difficulties in conceiving. Fifteen patients had planned their pregnancies.

During the pregnancy all patients reported being 'worried' about what they were eating, ten because they thought their binge-eating might affect their baby and six because of weight gain. Seven patients reported being 'faddy'. Thirteen patients became constipated and all of these took fruit, bran or other fibre rather than proprietary laxatives. All patients took iron or vitamin supplements and one patient took an unprescribed dietary supplement (Complan). Seventeen patients (85%) reported 'morning sickness' but all were able to distinguish it from self-induced vomiting.

Five patients (25%) found the change in body shape disturbing ("I felt my shape would be permanently changed"; "I felt ugly and huge"; "I felt sexually unattractive"), while six patients (30%) liked the change ("I felt proud and pleased").

After having their baby, 17 patients (85%) felt that having a child had altered the need to have a binge. However, only six of these felt that the pregnancy had been a positive help ("My need to be a good mother helped me to stop it"; "I have a horror of my baby witnessing my bingeing"). Eleven believed that the stresses of pregnancy and looking after a child had caused a deterioration ("It is more of a problem now because I cannot throw away the baby's food"; "It has created more problems in our marriage"; "I binge at night when I am feeding the baby"; "I feel so tired and I eat when I cry"). Eleven (55%) of the patients believed that the presence of their babies inhibited the binge-eating, either because they felt that the child was noticing or they were frightened that the child would copy them, or simply that looking after a baby left insufficient time to binge-eat. Six patients reported having their children in the same room as themselves when binge-eating and seven (35%) reported ignoring their child because they were pre-occupied with vomiting. Only six had felt that they had regained their figure after the delivery, but half the sample had felt they had lost the extra weight gained, six by pathological means.

The eating habits of the child

All babies were breast-fed either wholly or in combination with bottle-feeding. All the patients felt that the damage to the figure caused by breastfeeding was more than compensated for by "giving him the best", "not having to concern myself about sterilisation or food preparation" and "in any case I enjoyed it!" However, the majority (70%) of the women had problems with breastfeeding and reported sore nipples, not enough milk, engorgement, inverted nipples, or slow feeding. This resulted in 13 patients using partial bottle feeding – although all regretted the necessity. Weaning took place between 3 and 6 months and the first solid food given to the child showed, in our view, excellent judgement. All the women had informed themselves about childhood nutrition either from their Health Visitor or, more usually, from a popular but responsible women's magazine. All the children – save one (see later) – have grown satisfactorily.

Seven mothers reported concern that their children might be overweight. Three (15%) had slimmed their babies down. Their GPs clinical records showed the babies to have been plump, but well within a normal range of weight. Discussion with the GPs concluded that the mother's behaviour was not justified and in our judgement was related to their psychopathology. In fact, 12 patients reported being concerned that their children would become overweight "like me". Seven babies had had a period of vomiting but in only one case was this behaviourally induced "It happens when I play too much with him". Sixteen children had periods of constipation but none were given laxatives. No mother gave an emetic. Half the babies had periods of diarrhoea due to infection. Four mothers reported trying to avoid giving their children carbohydrate foods and five had prevented him/her having sweets. Three mothers thought their babies were 'faddy' eaters and seven reported uncooperative behaviour at mealtimes. Half the sample had given their children vitamin drops and two mothers had established their babies on a vegetarian diet.

Five mothers (25%) had used food to punish their children, usually by the withdrawal of sweets, and nine had reported giving food for comfort: "it cheers him up". Ten mothers believe that they had used food as a means of expressing the love they felt for their babies.

Abnormalities in pregnancy and delivery

Of 22 babies born, 13 were girls and nine boys. One was born prematurely and died shortly after birth at 29 weeks. The child weighed 950 g and was normal; his mother had a long history of alcohol and soft drug abuse and fulfilled our criteria for multi-impulsive bulimia (Lacey *et al*, 1986*a*). All other children were born at full term and weighed between 2500 and 3700 g (mean = 3200 g). There were two sets of twins. One baby had a cleft palate and one a cleft lip; neither mother was taking benzodiazepines. A number of women reported abnormalities in their pregnancy or in delivery. Nine reported hypertension, including two with pre-exclampsia. Eight deliveries were breech. Four were delivered by forceps and three by Caesarian section. These findings were confirmed by the patient's GP or Obstetrician.

Discussion

The chaotic eating pattern of the bulimic is not made worse by pregnancy. In fact, the reverse is true: pregnancy is associated with an improvement in the condition. Of the 20 patients in our study, 19 reduced the frequency of their binge-eating and all of them reduced the frequency of self-induced vomiting. Further, the rate of binge-eating fell with each trimester as did alcohol abuse in those patients who reported it. Although in most cases, symptoms returned after delivery, (and in some the symptoms were worse than prior to conception), in a quarter of the sample pregnancy appears to have been curative.

It is not possible on the basis of this study alone to determine the reasons for this pattern of response. However, the clinical evidence is strongly suggestive that there was a positive and strong attempt on the part of these mothers to control their eating disorder because they feared harming their babies. Normal body weight bulimics report a sense of being out of control which we believe to be essential for diagnosis (Lacey *et al*, 1986*a*). These findings suggest that this feeling can be overcome by the motivated patient and that pregnancy provides such motivation. Interestingly, it has been noted (Gayford, 1984) that pregnant alcoholic women similarly control their abuse – and probably for the same reason.

Certain other factors – physical, metabolic, perceptual and psychological – also appear to be involved. Physical restraint certainly operates in some patients. As the fundus of the enlarging uterus pushes out of the pelvis, the stomach is restricted and the volume of food it could contain thereby limited. The vast food intake reported by some of our patients (detailed calorific intakes are published in Lacey & Gibson, 1985) would not be possible. Thus the classical cycle of bulimia – binge-eating, vomiting and then bingeing again – is broken and the patient has the opportunity to re-learn a normal eating pattern. Although pregnancy is a time of increased and increasing energy demand this cannot explain our findings. If this were the case the effect would persist during lactation: a time when calorific requirements are higher still. Certainly, some patients maintain their improvement during breast feeding, but the majority show a deterioration.

A change in weight and shape is a normal part of pregnancy, and sensitivity about both are prevalent in bulimia. It is thus noteworthy that not only did the patients in our sample accept that body shape must change, doing nothing to minimise the effects of the pregnancy, but 75% did not even find the change disturbing. Bulimia is associated with a disturbance of body perception. This over-perception becomes significantly less following short-term behavioural and psychodynamic treatment (Birtchnell *et al*, 1985). It could be that pregnancy provides, at least for some bulimics, a similar forum for change. It is an acceptable, if not venerated 'largeness' which provides a break from the pressure of conforming to a cultural ideal of thinness.

It is not possible in any academic sense to differentiate those patients who return to binge-eating after pregnancy from those who maintain their improvement. It is noteworthy that the latter stopped their binge-eating completely when they discovered they were pregnant and that they tended to have stable and meaningful relationships. On the other hand, those who returned to bulimia tended to be women with inter-personal problems and a sense of failure as women – and they used pregnancy as an attempt to combat this, by achieving in an obvious feminine role – that of procreation. We would not be so crass as to suggest that pregnancy is an answer to bulimia. It does, however, provide a period of dietary stability, similar to that attempted in certain treatment programmes (Garner & Garfinkel, 1984). Both pregnancy and treatment reduce the drive to 'diet', both provide external restraint to prevent gross binge-eating, both encourage the acceptance of body shape and weight, both provide motivation to change and both are time-limited and provide an opportunity for self-reflection (Lacey, 1983).

Our findings have implications for treatment: counselling should be given on first interview particularly as a large number of the women (9%) intend shortly to have a child and a number become pregnant before formal treatment can begin. Our own treatment programme (Lacey, 1983, 1984*b*) is composed of short-term weekly sessions of focused behavioural and brief psychodynamic therapy, followed by 3-monthly appointments which continue the counselling and insight-directed therapy and which form an integral part of the treatment (Lacey *et al*, 1986*a*). We believe that treatment for the pregnant woman should be concentrated during the weeks of the third trimester when the eating behaviour is quiescent and deeper issues not thereby obscured. The 3-monthly follow-up sessions take place during the puerperium fitting well with the schedule of the busy young mother.

We do not wish to imply that binge-eating in the first trimester is implicated in foetal abnormality. The sample is too small and other factors – heredity, drugs and alcohol – cannot be discounted. However, the common fear among these patients that their abnormal eating behaviour could damage their unborn child cannot be dispelled by this study. The incidence of foetal abnormality, multiple pregnancies and obstetric complications was high. National figures (OPCS Monitor, 1983) sampled the same year as this project and in a similarly aged maternal population suggests a prevalence for multiple births of 0.82% of pregnancies. The prevalencies of cleft lip and cleft palate were respectively 9.9 and 4.3 per 10 000 total births. In a further case a patient gave birth to a boy with pyloric stenosis, which we have reported elsewhere (Lacey, 1983). This patient was outside this study because she became pregnant *after* treatment. Although the frequency of binge-eating was reduced, it nonetheless continued throughout the first trimester. These findings, even if considered within the context of all pregnant women seen in our clinic, are disturbing and warrant further investigation probably in collaboration with other research centres.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders, 3rd ed.* Washington DC: APA.

Birtchnell, S. A., Lacey, J. H., Harte, A. (1985) Body image distortion in bulimia nervosa. *British Journal of Psychiatry*, **147**, 408–412.

Bruch, H. (1962) Perceptual and conceptual aspects in anorexia nervosa. *Psychosomatic Medicine*, **24**, 187–194.

Garner, D. & Garfinkel, P. (eds) (1984) *Handbook of Psychotherapy for Anorexia Nervosa and Bulimia*, New York: Guilford Press.

Gayford, J. (1984) private communication: Regional Alcoholic Unit, Warlingham Park.

Hudson, J. J., Pope, H. G., Jonas, J. M. & Yurgelen-Todd, O. L. (1983) Family history study of anorexia nervosa and bulimia. *British Journal Psychiatry*, **142**, 133–137.

Lacey, J. H. (1983) Bulimia nervosa, binge-eating and psychogenic vomiting: a controlled treatment study and long-term outcome. *British Medical Journal*, **286**, 1609–1613.

—— (1984*a*) The bulimia syndrome. In *Advanced Medicine*, Vol. 20, (ed. A. Ferguson). London: Royal College of Physicians/Pitman.

—— (1984*b*) Time limited and group treatment of bulimia in *Handbook of Psychotherapy for Anorexia Nervosa and Bulimia* (eds D. M. Garner & P. E. Garfinkel). New York: Guilford Press.

—— & Gibson, E. (1985) Does laxative abuse control body weight? A comparative study of purging and vomiting bulimics. *Human Nutrition: Applied Nutrition*, **39A**, 36–42.

——, Harte, A., Birtchnell, S. A. (1986*a*) Bulimia – towards a rational approach to diagnosis and treatment. *Eating Disorders – Effective Care and Treatment* Vol. 1 (ed. F. E. F. Lacrocca). Isiyaku Euro-America Inc.

——, Coker, S., Birtchnell, S. A. (1986*b*) Bulimia: Factors associated with its etiology and maintenance. *International Journal of Eating Disorders*, **5**(3), 475–487.

OPCS Monitor (1983) FM1 No. 10 and NB3 8415.

Russell, G. (1979) Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychological Medicine*, **9**, 429–448.

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Detection of Postnatal Depression

Development of the 10-item Edinburgh Postnatal Depression Scale

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The development of a 10-item self-report scale (EPDS) to screen for Postnatal Depression in the community is described. After extensive pilot interviews a validation study was carried out on 84 mothers using the Research Diagnostic Criteria for depressive illness obtained from Goldberg's Standardised Psychiatric Interview. The EPDS was found to have satisfactory sensitivity and specficity, and was also sensitive to change in the severity of depression over time. The scale can be completed in about 5 minutes and has a simple method of scoring. The use of the EPDS in the secondary prevention of Postnatal Depression is discussed.

In the last decade several studies (Paykel *et al*, 1980; Cox *et al*, 1982; Kumar & Robson, 1984; O'Hara *et al*, 1984; Watson *et al*, 1984) have provided substantial confirmation for the earlier finding of Pitt (1968) that the months following childbirth are frequently characterised by psychiatric disorder, and that at least 10–15% of mothers experience a marked depressive illness at this time.

Furthermore, the results of a follow-up study showed that postnatal depression was usually accurately recalled by the mothers 3 years later; and that at least half of the depressed mothers had not recovered by the end of the first postpartum year (Cox *et al*, 1984). The finding that the children of such depressed mothers may show behaviour disturbance at 3 years (Wrate *et al*, 1985) or cognitive defects at 4 years (Cogill *et al*, 1986) suggests that postnatal depression may have a long-term negative impact on the family.

In our earlier study (Cox *et al*, 1982), we found that 13 of the 101 women interviewed had a marked post-natal depressive illness and yet the majority of these depressed mothers had not received any sustained treatment from their primary care workers nor had they been referred to a psychiatrist; the three women who had been referred were not those who were depressed. This failure to identify depression in the puerperium, especially when such mothers were usually known to their GP, Community Midwife or Health Visitor, was obviously a cause for much clinical concern.

A further difficulty in identifying depressed mothers is that screening scales for depression appear to have a number of limitations when used on childbearing women. In our earlier study from Edinburgh, for example, the Anxiety and Depression Scale (SAD) of Bedford & Foulds (1978) was found to have uncertain validity; of the 13 pregnant women who scored 6+ (Foulds' threshold for personal illness) only three had any form of psychiatric disorder, while four had minor symptoms only and six had no psychiatric illness whatsoever (Cox *et al*, 1983). The 30-item General Health Questionnaire of Goldberg *et al* (1970) has been assessed by Nott & Cutts (1982) for possible use in the puerperium and was also found to require some slight modifications. These authors reported that 89 (45%) of 200 puerperal women scored highly on the 30-item GHQ, but only 37 (18%) were found to be psychiatric cases. Similar difficulties with the Beck Depression Inventory (Beck *et al*, 1961) for use in identifying depression in the puerperium were reported by O'Hara *et al* (1983). In this study only 11 of the 19 women who scored above the cut-off score fulfilled Research Diagnostic Criteria (RDC) for Depression, and of the 23 who scored below the cut off there were four false negatives. Furthermore, the finding that measures of self control predicted postnatal depression as measured by the Beck Scale were not confirmed when a clinical syndrome diagnosis of depression was made using Research Diagnostic Criteria suggests that studies using this scale as the only measure of depression following childbirth may give misleading results (O'Hara *et al*, 1984).

The possible explanation for these apparent limitations of well established scales when used on childbearing women include their emphasis on the somatic symptoms of psychiatric disorder which may be caused by normal physiological changes associated with childbearing, as well as the reluctance of community workers to use questionnaires which may be regarded as time-consuming or which appear to lack face validity. These limitations may also be relevant to a consideration of the Zung Depression Scale (Zung, 1965) for use in the puerperium.

To be useful as a screening test for depression following childbirth, therefore, a self-report scale must be fully acceptable to women who may not regard themselves as unwell, or as in need of medical help. The scale needs also to be simple to complete, and not require the health worker to have any specialist knowledge of psychiatry. It must have satisfactory reliability and validity.

Furthermore, Williams *et al* (1980) have appropriately emphasised that rating scales which had been validated on hospital samples must be revalidated if they are used in community populations where the differences between psychiatric illness and normality is often less distinct. The earlier work of Snaith (1981, 1983) in this regard was also important, as he clearly recognised the need to modify existing scales of depression for use in new specific clinical situations and in particular was aware of the need to develop a screening questionnaire to detect postnatal depression. Others, such as Kumar (1982) and Cox *et al* (1983), had also emphasised that this task was an important current research priority.

Spurred on by these observations of our colleagues as well as by the pressing need for primary care workers to have practical help in identifying postnatal depression, we therefore decided to develop a self-report scale to detect mothers who were depressed following childbirth.

Method

A detailed analysis of the suitability of the Irritability, Depression and Anxiety Scale (IDA) (Snaith *et al*, 1978), the Hospital Anxiety and Depression Scale (HAD) (Zigmond & Snaith, 1983), and the Anxiety and Depression Scale of Bedford & Foulds (1978) was carried out and we eventually selected 21 items, including several of our own construction, which we thought to be appropriate for the detection of postnatal depression. These items were then tested during extensive pilot interviews with mothers of young babies. The detailed wording of items, their acceptability to mothers and health workers, as well as their likelihood of detecting postnatal depression was then carefully evaluated. Thirteen items were eventually selected as being those most likely to detect postnatal depression; seven of these were items constructed by ourselves and the other six were adapted from the IDA and the HAD.

The validity of this 13-item scale was then established on a sample of 63 purperal women who attended a health centre in Livingston (see Cox, 1986 for details). This study showed that these 13 items distinguished clearly between depressed and non-depressed women, although a rotated factor analysis revealed that the two items from the irritability subscale of the IDA, together with an item concerning the enjoyment of motherhood, formed a separate 'non-depression' factor; this latter finding providing confirmation of Snaith's earlier observation that irritability was often identified as a separate mood from depression and anxiety. As this analysis of our data had suggested that the specificity of the scale might be increased by omitting these three items, we decided to carry out a further validation study using only the 10 items which were more clearly related to depression. This 10-item scale would also have the advantage of taking less time to complete.

The validation study of the 10-item EPDS to be reported in this paper was carried out on 84 mothers living in Edinburgh or at Livingston new town. Most of the mothers, who were taking part in a study to determine the effectiveness of counselling by health visitors in the treatment of postnatal depression, had been identified by their health visitors at about 6 weeks following delivery as being potentially depressed. The health visitors had been asked to indicate whether, in their opinion, these mothers were 'normal', 'depressed', or were considered as having 'problems'. As we envisaged that a useful function of the scale would be to confirm the diagnosis of depression in women already suspected by their primary care worker as being possibly depressed, this sample was particularly appropriate. We also considered it important to determine whether the scale would satisfactorily identify postnatal depression when it was administered in a domestic environment. The mother's home was, therefore, an optimum setting in which to validate the 10-item scale; home visits by health visitors being regarded as an important link between the assessment of puerperal mothers and other members of the primary care team. Mothers in our sample were interviewed by R.S. using Goldberg's Standardised Psychiatric Interview (SPI) (Goldberg *et al*, 1970) and the majority of such interviews took place in the mothers own home (SPI-1). At this home visit the EPDS was first completed by the mother and was then placed in a sealed envelope so that the interviewer remained blind to the score while subsequently administering the SPI. To prevent any possible bias effect caused by the interviewer knowing that the subject may have been regarded by the health visitor as being 'depressed' or as having problems, 12 normal women were also included in the sample. The criteria used for the diagnosis of a depressive illness were the Research Diagnostic Criteria of Spitzer *et al* (1975). Mothers who were observed to have a depressed mood but who did not meet full RDC criteria for depression were, however, also separately identified. As recruitment of subjects into the study was slower than expected, a further 12 women were interviewed by J.C. at a local health clinic. Both interviewers had been trained in the use of the SPI and difficult ratings were jointly discussed. The validation of the 10-item EPDS was determined for the total sample by comparing the EPDS scores with the RDC clinical diagnosis of depression.

Results

Validation sample

The mean age of women was 26 years, and that of their babies was 3 months. Seventy-five percent had had normal deliveries, 15% Caesarian sections and a further 10% had forceps delivery. The majority (81%) were married, whilst 13% had a permanent partner. Only 6% were single parents. Social class distribution (according to husband's, or partner's occupation where one was present, or according to the mother's previous occupation in the case of single parents) was as follows: Social Class II:7%, III:35%, IV:31%, V:27%.

Validation of the 10-item EPDS

The results of the validation of the 10-item EPDS are shown in Fig. 1.

A threshold score of 12/13 was found to identify all of the 21 women with an RDC diagnosis of Definite Major Depressive Illness and two of the three women with Probable Major Depressive Illness. Four of the 11 women with Definite Minor Depression were false negatives, i.e. they scored 12 or below, and there were 11 'false positives', although six of these 11 women had depressive symptoms but did not meet full Research Diagnostic Criteria for depression. The subject with the highest false positive score (21) had a marked personality disorder; while the three women with a psychiatric diagnoses other than depression all scored below the cut-off.

The sensitivity of the EPDS, the proportion of RDC depressed women ($n = 35$) who were true positives ($n = 30$), was 86%; the specificity, proportion of non-depressed women ($n = 49$) who were true negatives ($n = 38$), was 78%. The positive predictive value, the proportion of women above threshold on the EPDS ($n = 41$) who met RDC criteria for depression ($n = 30$), was 73%. As it is important in some clinical or research settings for actual cases of depression not be missed, our data suggest that the failed detection of cases can be reduced to under 10% with a cut-off score of 9/10.

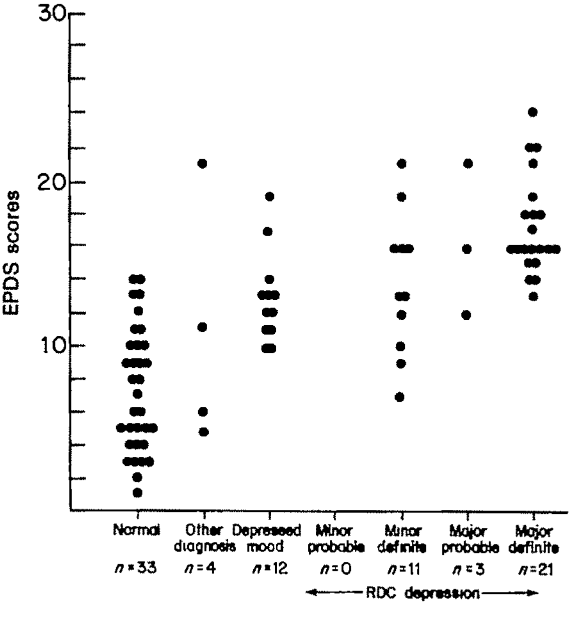

FIG. 1 Validation of Edinburgh Postnatal Depression Scale.

When analysis of our data was carried out on only those women ($n = 60$) interviewed by R.S. (excluding the 12 women with no previously identified problems, as well as the 12 subjects interviewed by J.C. at the Health Centre) the optimum threshold score was almost the same as in the larger sample, sensitivity 85%, specificity 77%, the positive predictive value having increased to 83%. The split-half reliability of the scale was found to be 0.88, and the standardised α-coefficient 0.87.

Sensitivity to change in the severity of depression over time was also established on a subsample by comparing the EPDS score at the first interview (EPDS-1), when mothers were taken into the counselling intervention study, with that obtained at the 11-week follow-up interview. At this second home interview the EPDS was completed for a second time (EPDS-2), and a repeat SPI (SPI-2) was carried out, the interviewer again remaining blind to both the EPDS-1 and the EPDS-2 scores.

Analysis of this data showed that those mothers who were depressed according to RDC criteria at both interviews ($n = 15$), showed no significant difference between their EPDS-1 (16.5) and EPDS-2 (15.38) mean scores on these two occasions, whereas mothers who were depressed at Interview 1 but not at Interview 2 ($n = 16$) had a reduction of score between EPDS-1 and EPDS-2 which was highly significant. (EPDS-1 mean score = 15.8, EPDS-2 mean score = 9.8, $t = 3.72$, $P = 0.002$). The EPDS-2 score in all but one subject fell to below the threshold of 12/13; the mother whose EPDS-2 score increased, but who was not depressed, had a probable cancer of the cervix and was diagnosed as having an anxiety neurosis.

Analysis of the possible influence on the EPDS score of another family member being present when the scale was completed suggested that under these circumstances women tended either to exaggerate, or to minimise, their psychiatric problems. Thus three subjects who had the highest 'false positive' score, and three of the four 'false negatives', had not been alone when they were interviewed.

Discussion

Our study has shown that the 10-item Postnatal Depression Scale, which was derived from the earlier work of Snaith, had satisfactory validity, split-half reliability and was also sensitive to changes in the severity of depression over time. Furthermore, we found that the scale was fully acceptable to the child-bearing women and was usually completed within 5 minutes. The simple method of scoring was an advantage and the health visitors recognised that the scale would greatly assist them in the detection of mothers who were depressed postpartum.

We believe it to be a substantial advantage that this validation study of the 10-item scale was carried out in a community setting and on women who were as close as possible to mothers regarded by their primary care workers as having problems. Our data nevertheless suggested that sensitivity and specificity of the scale may be increased if it is completed when other family members are not present.

It seems likely that the scale will be useful in the routine work of community health workers (e.g. health visitors, community psychiatric nurses and General Practitioners) and assist to identify postnatal depression in mothers thought by their health worker to be at risk. It may also be of use in treatment studies of postnatal depression when carried out on mothers living in the community.

Our data suggested that women who scored above a threshold of 12/13 were most likely to be suffering from a depressive illness of varying severity, and should therefore be further assessed by the primary care worker to confirm whether or not clinical depression is present. The EPDS is not a substitute for this clinical assessment, and a score just below the cut-off should not be taken to indicate the absence of depression, especially if the health professional has other reasons to consider this diagnosis. Our data also suggest that a threshold of 9/10 might be appropriate if the scale was considered for routine use by primary care workers.

We now plan to validate the scale for possible use during pregnancy and also to determine its usefulness in other populations. The scale could, for example, be administered by a computer at a mother's visit to an antenatal or postnatal clinic, and it may be useful as a more general screening scale for depressive illness. The revalidation of the scale for use in these other clinical settings must be carried out, however, before this wider use is recommended.

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References

Bedford, A. & Foulds, G. (1978) *Delusions-Symptoms-States. State of Anxiety and Depression (Manual)* Windsor: National Foundation for Educational Research.

Beck, A. T., Ward, C. H. & Mendelson, M. *et al* (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–571.

Cogill, S. R., Caplan, H. L., Alexandra, H., Robson, K. M. & Kumar, R. (1986) Impact of maternal postnatal depression on cognitive development of young children. *British Medical Journal*, **292**, 1165–1167.

Cox, J. L. (1986) *Postnatal Depression – A Guide for Health Professionals*. Edinburgh: Churchill Livingstone.

——, Connor, Y. & Kendell, R. E. (1982) Prospective study of the psychiatric disorders of childbirth. *British Journal of Psychiatry*, **140**, 111–117.

——, ——, Henderson, I., McGuire, R. J. & Kendell, R. E. (1983) Prospective study of the psychiatric disorders of childbirth by self report questionnaire. *Journal of Affective Disorders*, **5**, 1–7.

——, Rooney, A., Thomas, P. F. & Wrate, R. W. (1984) How accurately do mothers recall postnatal depression? Further data from a 3 year follow-up study. *Journal of Psychosomatic Obstetrics and Gynaecology*, **3**, 185–189.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. Maudsley Monograph, 21, Oxford: Oxford University Press.

——, Cooper, B., Eastwood, M. R., Kedward, H. B. & Shepherd, M. (1970) A standardised psychiatric interview for use in community surveys. *British Journal of Preventive and Social Medicine*, **24**, 18023.

Kumar, R. (1982) Neurotic disorders in childbearing women. In *Motherhood and Mental Illness*, eds I. Brockington & R. Kumar. London: Academic Press.

—— & Robson, K. M. (1984) A prospective study of emotional disorders in childbearing women. *British Journal of Psychiatry*, **144**, 35–47.

Nott, P. N. & Cutts, S. (1982) Validation of the 30-item General Health Questionnaire in postpartum women. *Psychological Medicine*, **12**, 409–413.

O'Hara, M. W., Rehm, L. P. & Campbell, S. B. (1983) Postpartum depression: a role for social network and life stress variables. *Journal of Nervous and Mental Disease*, **171**, 336–341.

——, Neunaber, D. J. & Zekoski, E. M. (1984) Prospective study of postpartum depression: prevalence, course and predictive factors. *Journal of Abnormal Psychology*, **93**, 158–171.

——, Emms, E. M., Fletcher, J. & Rassaby, E. S. (1980) Life events and social support in puerperal depression. *British Journal of Psychiatry*, **136**, 339–346.

Pitt, B. (1968) 'Atypical' depression following childbirth. *British Journal of Psychiatry*, **114**, 1325–1335.

Snaith, R. P. (1981) Rating scales. *British Journal of Psychiatry*, **138**, 512–514.

—— (1983) Pregnancy-related psychiatric disorder. *British Journal of Hospital Medicine*, **29**, 450–456.

——, Constantopoulos, A. A., Jardine, M. Y. & McGuffin, P. (1978) A clinical scale for the self-assessment of irritability. *British Journal of Psychiatry*, **132**, 164–171.

Spitzer, R., Endicott, J. & Robins, E. (1975) *Research Diagnostic Criteria*. Instrument No. 58. New York: New York State Psychiatric Institute.

Watson, J. P., Elliott, S. A., Rugg, A. J. & Brough, D. I. (1984) Psychiatric disorder in pregnancy and the first postnatal year. *British Journal of Psychiatry*, **144**, 453–462.

Williams, P., Tarnopolsky, A. & Hand, D. (1980) Case definition and case identification in psychiatric epidemiology: review and assessment. *Psychological Medicine*, **10**, 101–114.

Wrate, R. M., Rooney, A. C., Thomas, P. F. & Cox, J. L. (1985) Postnatal depression and child development: a three year follow-up study. *British Journal of Psychiatry*, **146**, 622–627.

Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

Zung, W. W. K. (1965) A self-rating depression scale. *Archives of General Psychiatry*, **12**, 63.

Appendix
Edinburgh Postnatal Depression Scale (EPDS)

The Edinburgh Postnatal Depression Scale (EPDS) has been developed to assist primary care health professionals to detect mothers suffering from postnatal depression; a distressing disorder more prolonged than the 'blues' (which occur in the first week after delivery) but less severe than puerperal psychosis.

Previous studies have shown that postnatal depression affects at least 10% of women and that many depressed mothers remain untreated. These mothers may cope with their baby and with household tasks, but their enjoyment of life is seriously affected and it is possible that there are long-term effects on the family.

The EPDS was developed at health centres in Livingston and Edinburgh. It consists of ten short statements. The mother underlines which of the four possible responses is closest to how she has been feeling during the past week. Most mothers complete the scale without difficulty in less than 5 minutes.

The validation study showed that mothers who scored above a threshold 12/13 were likely to be suffering from a depressive illness of varying severity. Nevertheless the EPDS score should *not* override clinical judgement. A careful clinical assessment should be carried out to confirm the diagnosis. The scale indicates how the mother has felt *during the previous week*, and in doubtful cases it may be usefully repeated after 2 weeks. The scale will not detect mothers with anxiety neuroses, phobias or personality disorders.

Instructions for users

1. The mother is asked to underline the response which comes closest to how she has been feeling in the previous 7 days.
2. All ten items must be completed.
3. Care should be taken to avoid the possibility of the mother discussing her answers with others.
4. The mother should complete the scale herself, unless she has limited English or has difficulty with reading.
5. The EPDS may be used at 6–8 weeks to screen postnatal women. The child health clinic, postnatal check-up or a home visit may provide suitable opportunities for its completion.

EDINBURGH POSTNATAL DEPRESSION SCALE (EPDS)

J. L. Cox, J. M. Holden, R. Sagovsky

Department of Psychiatry, University of Edinburgh

Name:
Address:
Baby's age:

As you have recently had a baby, we would like to know how you are feeling. Please UNDERLINE the answer which comes closest to how you have felt IN THE PAST 7 DAYS, not just how you feel today.

Here is an example, already completed.
I have felt happy:
Yes, all the time
Yes, most of the time
No, not very often
No, not at all

This would mean: "I have felt happy most of the time" during the past week. Please complete the other questions in the same way.

In the past 7 days:

1. I have been able to laugh and see the funny side of things
   As much as I always could
   Not quite so much now
   Definitely not so much now
   Not at all
2. I have looked forward with enjoyment to things
   As much as I ever did
   Rather less than I used to
   Definitely less than I used to
   Hardly at all

* 3. I have blamed myself unnecessarily when things went wrong
   Yes, most of the time
   Yes, some of the time
   Not very often
   No, never

4. I have been anxious or worried for no good reason
   No, not at all
   Hardly ever
   Yes, sometimes
   Yes, very often

* 5. I have felt scared or panicky for no very good reason
   Yes, quite a lot
   Yes, sometimes
   No, not much
   No, not at all

* 6. Things have been getting on top of me
   Yes, most of the time I haven't been able to cope at all
   Yes, sometimes I haven't been coping as well as usual
   No, most of the time I have coped quite well
   No, I have been coping as well as ever

* 7. I have been so unhappy that I have had difficulty sleeping
   Yes, most of the time
   Yes, sometimes
   Not very often
   No, not at all

* 8. I have felt sad or miserable
   Yes, most of the time
   Yes, quite often
   Not very often
   No, not at all

* 9. I have been so unhappy that I have been crying
   Yes, most of the time
   Yes, quite often
   Only occasionally
   No, never

*10. The thought of harming myself has occurred to me
   Yes, quite often
   Sometimes
   Hardly ever
   Never

Response categories are scored 0, 1, 2, and 3 according to increased severity of the symptom.
Items marked with an asterisk are reverse scored (i.e. 3, 2, 1 and 0). The total score is calculated by adding together the scores for each of the ten items.
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The Prognosis of Primary Depressive Illness

Its Relationship to the Dexamethasone Suppression Test

P. C. W. BOWIE, A. Y. BEAINI and L. J. BOWIE

We followed up 30 recovered depressives for an average of 2 years. At 18 months, despite prophylactic medication, 30% had relapsed. Persistent DST suppression during the index illness was found to be a predictor of good prognosis.

In an earlier study (Bowie & Beaini, 1985) three categories of dexamethasone suppression test (DST) response were defined on the basis of serial DST results. These three categories – normalising non-suppressors, fluctuating non-suppressors, and persistent suppressors – did not appear to differ demographically, nor in their response to biological treatments. However, it was postulated that the fluctuating non-suppressor category remained at risk of relapse despite symptomatic improvement, i.e. an underlying disease process remained unresolved. This postulate was supported by evidence from studies employing a repeat DST just prior to discharge from hospital (Greden *et al*, 1980; Goldberg, 1980*a*,*b*) which have shown that patients with abnormal (non-suppressed) DST response at that time were at significally higher risk of relapse. As persistent DST non-suppression is a rare finding and perhaps an indicator of poor treatment response (Albala *et al*, 1981), then it seemed likely that these 'at risk' patients were fluctuating non-suppressors.

Further supportive evidence was produced when patients with recurrent affective disorder taking lithium prophylaxis were re-tested (Coppen *et al*, 1983) and DST fluctuation was noted.

The present report is a follow-up of patients approximately 2 years after they were studied and categorised on the basis of serial DST response. The aim was to investigate the longer-term prognostic implications of the three serial DST categories.

Method

An attempt was made to trace and contact all 36 patients who had completed the previous research protocol.

Those patients who were traced and still lived locally were contacted by letter and asked if they would agree to a short semi-structured interview, and a DST.

At interview the following points were covered:

(a) length of time that antidepressant medication had been continued after the index illness
(b) evidence of any relapse
(c) the current medication, if any, that was being taken
(d) current depressive symptomatology was assessed using the Montgomery and Asberg Depression rating scale (Montgomery & Asberg, 1979).

A DST was arranged for patients in the week following the interview. This consisted of 1 mg of oral dexamethasone being taken at 21:30–22:00 and a plasma sample being collected at 16:00 the following day.

Plasma samples were stored at $-20°C$ until required for the estimation of cortisol concentration using the Amerlex direct radioimmunoassay method.

As in the previous study, a plasma cortisol level of above 140 mmol/litre was taken as the criterion for 'non-suppression'.

In addition the hospital/out-patient notes and GP records of all the patients were examined and the following points noted:

(a) length of time that antidepressant medication had been continued after the index illness
(b) evidence of any relapse
(c) the current medication, if any, that was being taken.

For the purposes of the study, relapsed was defined as 'any depressive episode requiring a change of treatment'.

Data was not normally distributed and was therefore analysed using non-parametric statistics (Mann-Whitney *U* test, χ^2 test with Yates' correction, and the Fisher exact probability test, and Kendall's rank correlation coefficient). Relapse/survival rates were extended beyond 18 months using the method of Kaplan & Meier (1958).

Results

Little or no information was available on six patients, but they did not differ from the remainder on the original variables (e.g. categorisation). Twenty-four patients were interviewed, rated for depressive symptoms and had a DST. Five patients were unavailable for interview and one had committed suicide; these patients have been included in the final analysis because sufficient information was available from hospital notes, GP records and the coroner's report on the suicide case.

Two of these patients were originally categorised as persistent suppressors and the remaining four as fluctuating non-suppressors, otherwise they did not differ on the original variables.

TABLE I

Characteristics of patients with non-suppressor DST response at follow-up

Patient	*Serial DST category*	*Follow-up MADSR score*	*No. of relapses*
K.L.	NN	31	2
D.M.	NN	0	0
D.C.	NN	21	1
S.K.	NN	14	2
R.S.	FN	14	3

NN: Normalising Non-Suppressor
FN: Fluctuating Non-Suppressor

Of the 24 patients interviewed six had an MADRS score greater than 10 (range 14–31), and five had non-suppressor DST response; their characteristics are shown in Table I.

As shown in Fig. 1, all three groups were followed up for a similar period and received antidepressant continuation therapy for a similar period.

While there was no significant difference in relapse between the normalising and fluctuating non-suppressors, the persistent suppressors had a trend towards a better prognosis (Fig. 2), which was significant at 27 months (Fisher exact probability, 1-tailed test, $P=0.045$).

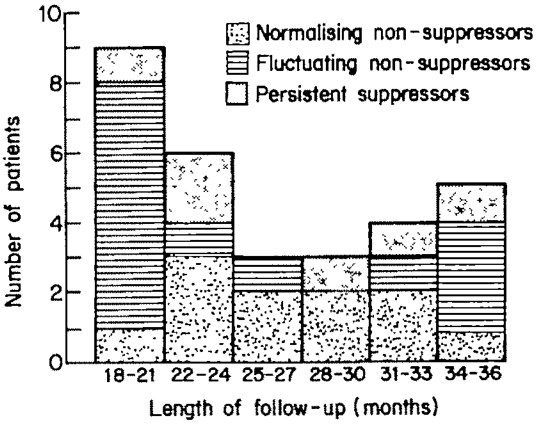

FIG. 1 Distribution of follow-up length.

Other factors which might have predicted long-term outcome were also examined. These findings are shown in Table II. These factors did not prove to be significant prognostic indicators.

However, when the proportion of abnormal DSTs (during the index episode) was examined, a significant correlation was found with the rate of relapse ($r=0.81$, $P<0.05$).

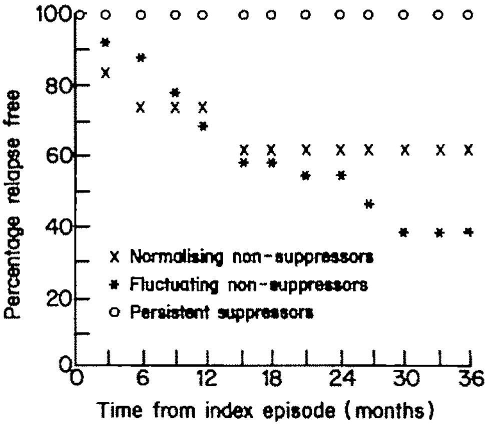

FIG. 2 Relapse rates for the three categories

Discussion

The results do not support the postulate that fluctuating non-suppressors are more likely to relapse than normalising non-suppressors. There are several possible explanations for this finding.

Firstly, the results do suggest a trend supporting the postulate. The non-significance of this trend may be due to the prolonged use of continuation medication in the patients studied. Prophylaxis was continued for an average of 21 months, the three categories receiving prophylaxis for similar periods. The original postulate implied a need for continuation medication for the fluctuating non-suppressors, but not for the normalising non-suppressors. This retrospective study has not adequately tested the postulate.

Alternatively the category of fluctuating non-suppressors may be spurious, and may not reflect an underlying suppressed disease process, but may have been generated by intra-patient changes in dexamethasone pharmacokinetics in the original study. Carroll *et al* (1980) suggested that DST non-

TABLE II

Factors examined for prognostic significance

	Sex		*Family history*		*Previous episode(s)*		*Pre-treatment DST*	
	M	*F*	*Yes*	*No*	*Yes*	*No*	*Non-suppressor*	*Suppressor*
No. of patients relapsing: *n*	3(8)	8(22)	1(8)	10(22)	6(12)	5(18)	9(19)	2(11)

suppression in depressives was not due to increased dexamethasone metabolism, but a more recent report from Berger *et al* (1984) found that a substantial proportion of tested patients fail to absorb dexamethasone adequately enough to produce serum levels that would suppress the hypothalamic-pituitary-adrenal axis.

While we can see no reason why dexamethasone pharmacokinetics should change from one week to the next, the test/re-test pharmacokinetics of dexamethasone have yet to be studied in this population, and therefore changes in dexamethasone pharmacokinetics may be an explanation.

Thirdly, perhaps the original method of categorisation has masked the results. The normalising non-suppressors were categorised on the basis of seven consecutive DST results. At one extreme there could have been one non-suppressor response followed by six suppressor responses or at the other extreme six non-suppressor responses followed by one suppressor response.

Certainly patients with serial DST profiles approaching the latter extreme could have been miscategorised fluctuating non-suppressors since fluctuating non-suppressors would automatically change categories to normalising non-suppressors at their first fluctuation.

The DST profiles of the four normalising non-suppressors who relapsed are shown in Table III. Patients 1 and 3 could certainly have been miscategorised, and this would explain their non-suppressor DST response at follow-up. Patient 2 is unlikely to have been miscategorised, and therefore implies that normalising non-suppressors do relapse, patient 4 not only implies this, but also shows DST fluctuation at follow-up which suggests that distinguishing between normalising and fluctuating non-suppressors is not valid.

TABLE III

DST profiles of the four relapsing normalising non-suppressors

	DST Profile non-suppressor: suppressor	*Follow-up post-dexamethasone cortisol (mmol/litre)*
Patient 1	6:1	244
Patient 2	1:6	97
Patient 3	4:3	304
Patient 4	1:6	391

However, distinguishing between non-suppressors and persistent suppressors does seem worthwhile as the results do show that persistent suppressors have a significant trend towards a better long-term prognosis when compared with all of the non-suppressors. This result suggests that this serial DST category is a more useful predictive tool than the category of DST suppression on the basis of a single test. It is also possible that persistent DST suppression is a more valid category when reflecting normal hypothalamic-pituitary-adrenal activity.

Two questions remain unanswered about this category. Firstly, does persistent DST suppression indicate a good long-term prognosis for primary depression, or does persistent DST suppression reflect a distinct entity within the broad heterogeneous concept of depressive illness?

Secondly, is the prognosis of persistent DST suppression dependant on medication or not?

Another prognostic indicator appears to be the proportion of abnormal DSTs during the index illness, the higher the proportion of abnormal DSTs indicating a worse prognosis. However, this finding has undoubtedly been influenced by the persistent suppressors, and when this influence is removed there is not a significant correlation ($r=0.73$, $P>0.05$). Nevertheless the correlation is high and probably warrants further investigation in a larger scale study.

Finally, we would like to comment on the prognosis of primary depressive illness, comparing our findings with other follow-up studies.

There is little doubt about the efficacy of prophylactic medication for depressive illness (Mindham *et al*, 1973; Glen *et al*, 1984) and the prognosis of our depressives has therefore been modified from its natural course by the use of such prophylaxis.

However, while prophylaxis may postpone relapse it certainly does not abolish it altogether – Mindham *et al* (1973) found that 22% of those on active medication relapsed within 6 months, Glen *et al* (1984) in a 3 year follow-up study found that 66% of those on active medication had relapsed.

Our results are somewhat similar – after 18 months (the shortest follow-up period) nine (30%) of our depressives had relapsed, only one of these not having continued with prophylaxis. Using the method of Kaplan & Meier (1958) the estimated relapse rate at 3 years is 40%.

The question of when is the risk of relapse highest is also worth considering. Our findings are similar to those of Glen *et al* (1984) with half of the relapses occurring after only 6 months (relapse rates of 20% and 33% respectively). Therefore both studies suggest that the risk of relapse is highest in the first 6 months after the index episode. This may be as a result of the re-appearance of symptoms associated with the index illness in some patients. Such as explanation has been discussed by Fleiss *et al* (1978). The difference in actual relapse rates probably reflects the difference in original patient selection.

The index episode was the first illness for the majority of our depressives, but Glen *et al* studied depressives with at least one episode prior to the index episode. Other follow-up studies beyond 6 months (Prien *et al*, 1984) have also involved depressives with episodes prior to the index episode. However, our relapse rate at 6 months compares well with the 22% relapse rate found by Mindham *et al* (1973) studying a demographically similar population.

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References

ALBALA, A. A., GREDEN, J. F., TARIKA, J. & CARROLL, B. J. (1981) Changes in serial dexamethasone suppression tests among unipolar depressives receiving electroconvulsive treatment. *Biological Psychiatry*, **16**, 551–560.

BERGER, M., PIRKE, K. M., DOERR, P., KREIG, J. C. & VON ZERSSEN, D. (1984) The limited utility of the dexamethasone suppression test for the diagnostic process in psychiatry. *British Journal of Psychiatry*, **145**, 372–382.

BOWIE, P. C. W. & BEAINI, A. Y. (1985) Normalisation of the dexamethasone suppression test: a correlate of clinical improvement in primary depressives. *British Journal of Psychiatry*, **147**, 30–35.

CARROLL, B. J., SCHROEDER, K., MUKHOPADHYAY, S., GREDEN, J. F. & TARIKA, J. (1980) Plasma dexamethasone concentrations and cortisol suppression response in patients with endogenous depression. *Journal of Clinical Endocrinology and Metabolism*, **51**, 433–437.

COPPEN, A., ABOU-SALEH, M., MILLN, P., METCALFE, M., HARWOOD, J. & BAILEY, J. (1983) Dexamethasone suppression test in depression and other psychiatric illness. *British Journal of Psychiatry*, **142**, 498–504.

FLEISS, J. L., PRIEN, R. F., DUNNER, D. L. & FIEVE, R. R. (1978) Actuarial studies of the course of manic-depressive illness. *Comprehensive Psychiatry*, **19**, 355–362.

GLEN, A. I. M., JOHNSON, A. L., SHEPHERD, M. (1984) Continuation therapy with lithium and amitriptyline in unipolar depressive illness: a randomized, double-blind, controlled trial. *Psychological Medicine*, **14**, 37–50.

GOLDBERG, I. K. (1980*a*) Dexamethasone suppression test as indicator of safe withdrawal of antidepressant therapy. *Lancet*, **i**, 376.

—— (1980*b*) Dexamethasone suppression tests in depression and response to treatment. *Lancet*, **ii**, 92.

GREDEN, J. F., ALBALA, A. A., HASKETT, R. F., JAMES, N. M., GOODMAN, L., STEINER, M. & CARROLL, B. J. (1980) Normalization of dexamethasone suppression test: a laboratory index of recovery from endogenous depression. *Biological Psychiatry*, **15**, 449–458.

KAPLAN, E. L. & MEIER, P. (1958) Non-parametric estimation from incomplete observations. *Journal of the American Statistical Association*, **53**, 457–481.

MINDHAM, R. H. S., HOWLAND, C. & SHEPHERD, M. (1973) An evaluation of continuation therapy with tricyclic antidepressants in depressive illness. *Psychological Medicine*, **3**, 5–17.

MONTGOMERY, S. A. & ASBERG, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, **134**, 382–389.

PRIEN, R. F., KUPFER, D. J., MANSKY, P. A., SMALL, J. G., TUASON, V. B., VOSS, C. B. & JOHNSON, W. E. (1984) Drug therapy in the prevention of recurrences in unipolar and bipolar affective disorders. *Archives of General Psychiatry*, **41**, 1096–1104.

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A Combined Study of Cortisol, ACTH and Dexamethasone Concentrations in Major Depression

Multiple Time-Point Sampling

B. G. CHARLTON, A. LEAKE, C. WRIGHT, H. W. GRIFFITHS and I. N. FERRIER

The pathophysiology of hypercortisolaemia in major depression was examined. ACTH was measured using a novel immunoradiometric assay of high specificity and sensitivity. Twenty-eight patients with major depression and 18 control subjects were studied. Blood samples for basal hormone concentration were taken at 09:00, 16:00 and 23:00 on day 1, followed by administration of 1 mg dexamethasone at 23:00. Further samples were taken at 09:00 and 16:00 on day 2. Dexamethasone concentration was measured in day 2 samples and no significant difference was found between the depressed group and control subjects. In the depressed group cortisol concentration was elevated at 23:00 on day 1, and ACTH concentrations were elevated in post-dexamethasone samples. ACTH and cortisol concentrations were not directly correlated in individual patients. The elevated plasma cortisol associated with major depression is not solely mediated by changes in ACTH.

Elevated secretion of corticosteroids is probably the best-validated biochemical abnormality in depression (Arana *et al*, 1985; Rubin, 1985) and is characterised by reduced diurnal rhythmicity and resistance to the suppressive effects of dexamethasone: DST non-suppression (Carroll *et al*, 1981). DST non-suppression has been related to endogenous type depression (Holden, 1983), increased severity of depression (Klein *et al*, 1984) and enhanced treatment responses to antidepressants (Nelson *et al*, 1982) and ECT (Coryell, 1982). The abnormality is usually reported to normalise upon clinical recovery (Bowie & Beaini, 1985); if the DST does not normalise there is an increased risk of relapse (Targum, 1984). DST non-suppression is not specific to major depression (Berger *et al*, 1984), but occurs in other conditions such as mania (Christie *et al*, 1986) and dementia (McKeith, 1984) and in normal subjects (Hallstrom *et al*, 1983). Some previous studies are unreliable as normal control groups were not used and the studies have indiscriminately applied the cut-off value for elevated cortisol suggested by Carroll (1982). In spite of this research, little is known of the pathophysiology underlying hypercortisolaemia in depression.

Cortisol is generally considered to be under stimulatory control by ACTH. Studies of plasma ACTH concentrations in depression have yielded contradictory results, some showing normal concentrations (Fang *et al*, 1981; Yerevanian & Woolf, 1983; Linkowski *et al*, 1985) and others showing elevated concentrations (Kalin *et al*, 1982; Demisch *et al*, 1983; Reus *et al*, 1983). These contradictory results may reflect the lack of suitable control groups (i.e. those studies which compare DST suppressors and non-suppressors), infrequent blood sampling, or failure to measure ACTH both before and after dexamethasone administration. Furthermore, serious problems arise when measuring ACTH using radio-immunoassay (RIA) systems. Most RIA methods for ACTH tend to be non-specific (Schoneshofer *et al*, 1981); insensitive, requiring large volumes of plasma (Rees *et al*, 1971); or prone to plasma interference, requiring prior extraction (Krieger, 1979). In this study we have overcome these difficulties by using a recently developed two-site recognition immuno-radiometric assay (IRMA) for the measurement of ACTH (Hodgkinson *et al*, 1984). This uses antibodies directed at both terminals of the ACTH molecule, so cross-reactivity with other species is minimal. Furthermore, the assay is very sensitive and uses only small volumes of unextracted plasma.

Dexamethasone causes suppression of ACTH and cortisol secretion. Variability in the absorption and metabolism of orally administered dexamethasone may be expected to alter the plasma concentration, and therefore its effectiveness at causing suppression. Recent studies have demonstrated lower plasma dexamethasone concentrations in depressed DST non-suppressors when compared with DST suppressors

(Berger *et al*, 1984; Johnson *et al*, 1984; Morris *et al*, 1986). This suggests that altered disposition of dexamethasone may help to explain abnormal DST results. In contrast, Carroll *et al* (1980) showed no significant differences in dexamethasone concentrations in a similar study. These contradictory findings seem to require further clarification, and accordingly plasma dexamethasone concentrations were measured on the day following oral administration.

This study is the first to measure cortisol, ACTH and dexamethasone plasma concentrations in a single group of patients and control subjects.

Patients and methods

Ethical permission for the study was obtained from the Newcastle NHS ethical committee. Patients were drawn from the psychiatric wards of two general hospitals. The criteria for DSM-III Major Depressive episode were employed (American Psychiatric Association, 1980), and patients were further subclassified according to presence or absence of melancholia. Severity of depression was measured using the 17-item Hamilton Rating Scale (HRS) (Hamilton, 1960) and 'endogenicity' was measured using the Newcastle Endogenous Depression Diagnostic Inventory (NEDDI) (Carney *et al*, 1965). Eight patients were drug-free and the remainder were taking a variety of medications, principally low dose benzodiazepines ($n=15$), tricyclic antidepressants ($n=5$) and lithium carbonate ($n=4$). We were careful to exclude any subjects taking drugs known to influence the DST (Carroll, 1982). Control subjects were healthy volunteers.

The study involved 28 depressed patients (9 male, 19 female, age range 36–63) and 18 control subjects (8 male, 10 female, age range 19–57). Among the depressed patients, 59% were classified as having major depression with melancholia; and 61% scored $\geqslant 6$ on the NEDDI ('endogenous' depression). The mean HRS score was 25, (s.d. = 5).

Venous samples were collected in lithium heparin coated tubes at 09:00, 16:00 and 23:00 on day 1, after which 1 mg of dexamethasone was administered orally, and further samples were collected at 09:00 and 16:00 on day 2. Blood was maintained at 4°C during collection and centrifugation, and enzyme inhibitors (Aprotinin 200 KIU per ml blood and phenylmethanesulphonyl fluoride, *p*-hydroxymercuribenzoate, *N*-ethylmaleimide and EDTA to a final concentration of 1 mmol/litre were added to prevent degradation of peptides (Hodgkinson *et al*, 1984), before storage at −30°C.

Cortisol was measured using a modification of the competitive protein binding (CPB) assay (Murphy, 1967). Cortisol is the most abundant of the glucocorticoids present in plasma. We will use 'cortisol' to refer to the whole range of corticosteroids which are measured by the assay system (Meltzer & Fang, 1983). This convention, although not explicitly stated, is used in most previous research and we have adopted it to avoid confusion. Cross-reactivities are as follows: cortisone 17.5; corticosterone 124.5; 11-deoxycorticosterone 83.4; 11-deoxy-, 17-hydroxycorticosterone 87.5; 17-hydroxy progesterone 89.3%. Dexamethasone has negligible cross-reactivity at 0.001%. Mean intra-assay variation was 8% and inter-assay variation was 6%. The minimum detectable concentration was typically 55 nmol/litre. ACTH was measured using an IRMA. Mean intra-assay variation was 6% and inter-assay variation was 17%. The minimum detectable concentration was 10 pg/ml. Dexamethasone concentration was measured in the samples collected on day 2 using an RIA (Kream *et al*, 1983). Cross-reactivity with endogenous corticosteroids was negligible. Mean intra-assay variation was 8% and inter-assay variation was 14%. The minimum detectable concentration was 2 nmol/litre.

Statistics involved comparisons of depressed patients with normal subjects at each time-point. Student's unpaired *t* test was employed, and differences were considered significant for values of $P<0.05$.

Results

Cortisol

Cortisol results are shown in Table I and Fig. 1. The only time-point at which there was a significant difference between patients and control subjects was at 23:00 on day 1, when cortisol was elevated in major depression. Cortisol values in post-dexamethasone samples were not significantly different in the two groups. Cortisol concentration was not significantly correlated with HRS scores or different between those patients with and without melancholia or 'endogenicity'.

TABLE I
Cortisol concentrations at different time-points

	Cortisol (nmol/litre); mean ± s.d.				
	Day 1			*Day 2*	
	09:00	*16:00*	*23:00*	*09:00*	*16:00*
Control subjects	480 ± 169	285 ± 114	145 ± 57	91 ± 57	93 ± 29
Major depression	499 ± 168	354 ± 120	264 ± 188*	120 ± 69	146 ± 124

*Significantly greater than control subjects, $P<0.05$.

TABLE II
ACTH concentrations at different time-points

	ACTH (pg/ml); mean ± s.d.				
	Day 1			*Day 2*	
	09:00	*16:00*	*23:00*	*09:00*	*16:00*
Control subjects	43 ± 13	35 ± 10	29 ± 9	25 ± 8	24 ± 6
Major depression	47 ± 24	41 ± 19	36 ± 13	34 ± 12*	34 ± 10**

*Significantly greater than control subjects, $P < 0.05$.
**Significantly greater than control subjects, $P < 0.01$.

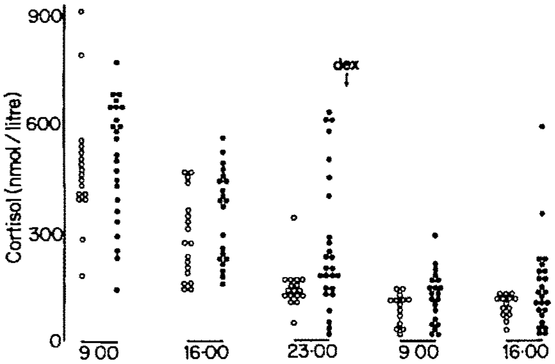

FIG. 1 Plasma cortisol concentration at different time-points; 1 mg dexamethasone (dex) administered orally following 23:00 sample; open circles = control subjects; closed circles = depressed patients.

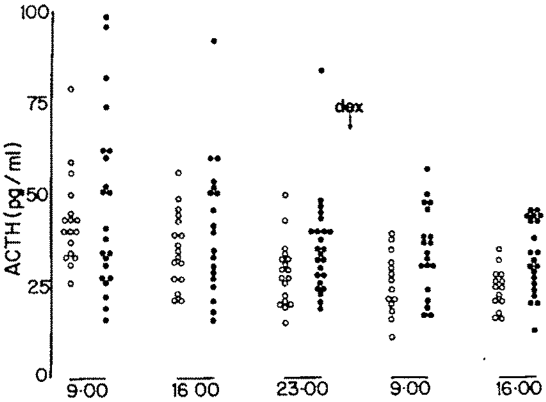

FIG. 2 Plasma ACTH concentrations at different time-points: 1 mg dexamethasone (dex) administered orally following 23:00 sample; open circles = control subjects; closed circles = depressed patients.

ACTH

ACTH results are shown in Table II and Fig. 2. Plasma ACTH concentration was elevated in the depressed group in samples taken on day 2 (i.e. following dexamethasone administration). Day 1 samples showed no significant differences. ACTH concentrations were not significantly correlated with cortisol at any time-point and were not related to HRS score or the presence of melancholia and/or 'endogenicity'.

Dexamethasone

Dexamethasone results are shown in Table III. All subjects had detectable dexamethasone concentrations at 09:00, showing that they had complied with medication. There was no significant difference between control subjects and depressed patients at either time-point, and no significant correlation between dexamethasone concentration and either cortisol or ACTH concentration.

Analysis using 'cut-off' values

The DST cut-off value of 138 nmol/litre (5 μg/dl) of cortisol in the post-dexamethasone samples was employed (Carroll, 1982): 58% of depressed patients and 7% of control subjects were defined as DST non-suppressors.

Hypersecretion was defined as a concentration of hormone greater than one standard deviation above the control subjects' mean. Thirteen depressed patients (46%) showed hypersecretion of cortisol at 23:00, and 13 depressed patients (46%) showed ACTH hypersecretion following dexamethasone. However, only seven patients (25%) exhibited both abnormalities. This group was not distinguishable from the other patients on the basis of HRS score, 'endogenicity' or melancholia, nor on dexamethasone concentration.

TABLE III
Dexamethasone concentrations on day 2

	Dexamethasone (nmol/litre); mean ± s.d.	
	09:00	*16:00*
Control subjects	7.9 ± 3.4	5.1 ± 3.0
Major depression	8.2 ± 3.7	6.0 ± 3.3

Discussion

The group of depressed patients contained 58% DST non-suppressors using the cut-off value of 138 nmol/litre. This figure is broadly comparable with previous studies on similar groups of patients. However, we did not show elevation in post-dexamethasone cortisol concentrations, and the cortisol data did not fall into a bimodal distribution. Many groups have employed the 138 nmol/litre cut-off value determined by Carroll (1982), despite the recommendation by Carroll's group (Ritchie *et al*, 1985) and others (Meltzer & Fang, 1983) that each laboratory should define its own values using normal subjects. We contend that the use of cut-off values is often misleading and suggest that data is more informative if presented in full, with comparisons between depressed and normal groups being performed before the use of cut-off values. In contrast to the DST data, a significant elevation in the cortisol concentration at 23:00 was noted in depressed patients, which reflects a blunting of diurnal rhythmicity.

The ACTH results in this study are more extensive than in most previous studies, demonstrating both diurnal rhythm and the effect of dexamethasone. The highly specific and sensitive assay was able to detect ACTH even after dexamethasone administration. Basal concentrations of ACTH were not altered in depression, but ACTH was resistant to dexamethasone suppression in this group. ACTH concentrations did not correlate closely with cortisol and there was only a partial overlap between the group with elevated cortisol and the group with elevated ACTH. Dissociation between ACTH and cortisol has been noted in several previous studies; for example, following haemorrhage in dogs (Gann, 1979), following pain in cats (Bereiter *et al*, 1982), with increased intracranial pressure in humans (Feibel *et al*, 1983), and in depression (Fang *et al*, 1981; Kalin *et al*, 1982; Reus *et al*, 1983; Yerevanian & Woolf, 1983). It is probable that factors additional to ACTH are responsible for modulating cortisol secretion (Lowry, 1984; Fehm *et al*, 1984). Depressed patients have been found to secrete more cortisol in response to synthetic ACTH 1–24 than control subjects (Amsterdam *et al*, 1983). It would therefore seem that some other factor is potentiating the effect of ACTH on the adrenal. A possible candidate is *N*-terminal pro-opiocortin (*N*-POC) which is secreted from the same precursor as ACTH (Lowry, 1984) and has been shown to potentiate the action of ACTH on cortisol secretion *in vitro* (Pederson *et al*, 1980) and in animal studies (Al Dujaili *et al*, 1981). *N*-POC has been measured in elevated concentrations following cardiac arrest, when cortisol was elevated but ACTH remained at normal concentrations (Wortsman *et al*, 1985). Taken together, the above observations suggest that increased *N*-POC concentration could contribute to the hypercortisolaemia seen in depression.

Plasma dexamethasone concentrations were similar in depressed patients and normal subjects, implying that the end result of dexamethasone absorption and metabolism is not altered in depression, and therefore the reduced suppressive effect of dexamethasone on cortisol secretion may be mediated at the receptor level. To avoid these uncertainties, the 23:00 basal cortisol concentration seems to be a better index of hypercortisolaemia in depression.

This study showed no variation in cortisol or ACTH concentrations which were related to melancholia, 'endogenicity' or to HRS score. Disagreement in the literature could be due to differences in populations studied (for example, this population consists predominantly of patients with 'endogenous' depression of moderate severity) and to differences between hormone assay systems.

It has been suggested by Gold (1985) that CRF hyperactivity underlies the hypercortisolaemia associated with depression. Gold proposes a model of sequential changes in the hypothalamo–pituitary–adrenal axis whereby tonically elevated CRF causes initial ACTH hypersecretion, resulting in adrenal hypertrophy. Following this, cortisol feedback causes normalisation of ACTH secretion, which then produces elevated cortisol from enlarged adrenals. No time-scale is suggested for these changes but it may be anticipated that patients with a short history of depression would have higher ACTH concentrations than those with a long history. We tested this hypothesis using a retrospective analysis of casenotes to determine the duration of depressive illness at the time of endocrine testing. There were 18 sets of casenotes suitable for this analysis. Length of illness varied from 3 weeks to 10 years. We analysed the data to determine whether length of illness was correlated with ACTH at any time-point, and we found no such relationship. Therefore this finding does not provide support for the hypothesis of Gold (1985).

This study defines a group of depressed patients with reduced diurnal rhythmicity of cortisol and another, overlapping, group with ACTH resistance to dexamethasone suppression. The hypercortisolaemia associated with depression is not mediated by ACTH alone, and the nature of its pathophysiology remains to be elucidated.

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References

Al-Dujaili, E. A. S., Hope, J., Estivariz, F. E., Lowry, P. J. & Edwards, C. R. W. (1981) Circulating human pituitary pro-γ-melanotropin enhances the adrenal response to ACTH. *Nature*, **291**, 156–158.

American, Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed. Washington, DC: APA.

Amsterdam, J. D., Winokur, A., Abelman, E., Lucki, I. & Rickel, K. (1983) Cosynotropin (ACTH 1-24) stimulation test in depressed patients and healthy subjects. *American Journal of Psychiatry*, **140**, 907–909.

Arana, G. W., Baldessarini, R. J. & Ornsteen, M. (1985) The dexamethasone suppression test for diagnosis and prognosis in psychiatry. *Archives of General Psychiatry*, **42**, 1193–1204.

Bereiter, D. A., Plotsky, P. M. & Gann, D. S. (1982) Tooth pulp stimulation potentiates the adrenocroticotropin response to haemorrhage in cats. *Endocrinology*, **111**, 1127–1132.

Berger, M., Pirke, K. M., Doerr, P., Krieg, J. C. & Zerssen, D. V. (1984) The limited utility of the dexamethasone suppression test for the diagnostic process in psychiatry. *British Journal of Psychiatry*, **145**, 372–382.

Bowie, P. C. & Beaini, A. Y. (1985) Normalisation of the dexamethasone suppression test. *British Journal of Psychiatry*, **147**, 30–35.

Carney, M. W. P., Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

Carroll, B. J. (1982) The dexamethasone suppression test for melancholia. *British Journal of Psychiatry*, **140**, 292–304.

——, Schroeder, K., Mukhopadhyay, S., Greden, J. F., Feinberg, M., Ritchie, J. & Tarika, J. (1980) Plasma dexamethasone concentrations and cortisol suppression response in patients with endogenous depression. *Journal of Clinical Endocrinology and Metabolism*, **51**, 433–437.

——, Feinberg, M., Greden, J. F., Tarika, J., Albala, A. A., Haskett, R. F., James, N. M., Kronfol, Z., Lohr, N., Steiner, M., Vigne, J. P. D. & Young, E. (1981) A specific laboratory test for the diagnosis of melancholia. *Archives of General Psychiatry*, **38**, 15–22.

Christie, J. E., Whalley, L. J., Dick, H., Blackwood, D. M. R., Blackburn, I. M. & Fink, G. (1986) Raised plasma cortisol concentrations a feature of drug-free psychotics and not specific for depression. *British Journal of Psychiatry*, **148**, 58–65.

Coryell, W. (1982) Hypothalamic–pituitary–adrenal axis abnormality and ECT response. *Psychiatry Research*, **6**, 283–291.

Demisch, K., Demisch, L., Bochnik, H. J. & Schulz, F. (1983) Comparison of the ACTH suppression test and dexamethasone suppression test in depressed patients. *American Journal of Psychiatry*, **140**, 1511–1512.

Fang, V. S., Tricou, B. J., Robertson, A. & Meltzer, H. Y. (1981) Plasma ACTH and cortisol levels in depressed patients. *Life Sciences*, **29**, 931–938.

Fehm, H. L., Klein, E., Holl, R. & Voigt, K. H. (1984) Evidence for extrapituitary mechanisms mediating the morning peak of plasma cortisol in man. *Journal of Clinical Endocrinology and Metabolism*, **58**, 410–414.

Feibel, J., Kelly, M., Lee, L. & Woolf, P. (1983) Loss of adrenocortical suppression after acute brain injury. *Journal of Clinical Endocrinology and Metabolism*, **57**, 1245–1250.

Gann, D. S. (1979) Cortisol secretion after haemorrhage. *Nephron*, **23**, 119–124.

Gold, P. W. (1985) Corticotropin-releasing factor stimulation in hypercortisolaemic psychiatric states. In Clinical applications of corticotropin-releasing factor (moderator Chrousos, G. P.). *Annals of Internal Medicine*, **102**, 352–355.

Hallstrom, T. J., Samuelsson, S., Balldin, J., Walinder, J., Bengtsson, C., Nystrom, E., Andersch, B., Lindstedt, G. & Lundberg, P. A. (1983) Abnormal dexamethasone test in normal females. *British Journal of Psychiatry*, **142**, 489–497.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

Hodgkinson, S. C., Allolio, B., Landon, J. & Lowry, P. J. (1984) Development of a non-extracted two-site immunoradiometric assay for corticotropin utilising extreme amino and carboxyterminally directed antibodies. *Biochemistry Journal*, **218**, 703–711.

Holden, N. L. (1983) Depression and the Newcastle scale. *British Journal of Psychiatry*, **142**, 505–507.

Johnson, G., Hunt, G., Kerr, K. & Caterson, I. (1984) Dexamethasone suppression test and plasma dexamethasone levels in depressed patients. *Psychiatry Research*, **13**, 305–313.

Kalin, N. H., Weiler, S. J. & Sheldon, S. E. (1982) Plasma ACTH and cortisol concentration before and after dexamethasone. *Psychiatry Research*, **7**, 87–92.

Klein, H. E., Bender, W., Mayr, H., Niederschweiberer, A. & Schmauss, M. (1984) The DST and its relationship to psychiatric diagnosis, symptoms and treatment outcome. *British Journal of Psychiatry*, **145**, 591–599.

Kream, J., Mulay, S., Fukushima, D. K. & Solomon, S. (1983) Determination of plasma dexamethasone in the mother and the newborn after administration of the hormone in a clinical trial. *Journal of Clinical Endocrinology and Metabolism*, **57**, 127–133.

Krieger, D. T. (1979) Plasma ACTH and corticosteroids. In *Endocrinology* (ed. L. J. Degroot). New York: Grune and Stratton.

Linkowski, P., Mendlewicz, J., Leclerq, R., Brasseur, M., Mubain, P., Golstein, J., Copinschi, G. & Cauter, E. V. (1985) The 24 hour profile of adrenocorticotropin and cortisol in major depressive illness. *Journal of Clinical Endocrinology and Metabolism*, **61**, 429–438.

Lowry, P. J. (1984) Pro-opiocortin. *Bioscience Reports*, **4**, 467–482.

McKeith, I. G. (1984) Clinical use of the DST in psychogeriatric population. *British Journal of Psychiatry*, **145**, 389–393.

Meltzer, H. Y. & Fang, V. S. (1983) Cortisol determination and the dexamethasone suppression test. *Archives of General Psychiatry*, **40**, 501–505.

Morris, M., Carr, V., Gilliland, J. & Hooper, M. (1986) Dexamethasone concentrations and the dexamethasone suppression test in psychiatric disorders. *British Journal of Psychiatry*, **148**, 66–69.

Murphy, B. E. A. (1967) Some studies of the protein-binding of steroids and their application to the routine micro and ultramicro measurement of various steroids in body fluids by competitive protein-binding radioassay. *Journal of Clinical Endocrinology and Metabolism*, **27**, 973–990.

Nelson, W. H., Orr, W. W., Stevenson, J. M. & Shane, S. R. (1982) Hypothalamic pituitary–adrenal axis activity and tricyclic response in major depression. *Archives of General Psychiatry*, **39**, 1033–1036.

PEDERSON, R. C., BROWNIE, A. C. & LING, N. (1980) Pro-adrenocorticotropin/endorphin-derived peptides. *Science*, **208**, 1044–1045.

REES, L. H., COOK, D. M., KENDALL, J. W., ALLEN, C. F., KRAMER, R. M., RATCLIFFE, J. G. & KNIGHT, R. A. (1971) A radioimmunoassay for rat plasma ACTH. *Endocrinology*, **89**, 254–261.

REUS, V. I., MARSHALL, J. & DALLMAN, M. (1983) Regulation of ACTH and cortisol in depression. *Peptides*, **4**, 786–788.

RITCHIE, J. C., CARROLL, B. J., OLTON, P. R., SHIVELY, V. & FEINBER, M. (1985) Plasma cortisol determination for the dexamethasone suppression test. *Archives of General Psychiatry*, **42**, 493–497.

RUBIN, R. T. (1985) The prospects for clinical psychoneuroendocrinology. *Psychological Medicine*, **15**, 451–454.

SCHONESHOFER, M., FEUNER, A. & MOLNAR, I. (1981) Heterogeneity of corticotrophin-immunoreactive compounds in human body fluids. *Clinical Chemistry*, **27**, 1875–1877.

TARGUM, S. D. (1984) Persistant neuroendocrine dysregulation in major depressive disorder. *Biological Psychiatry*, **19**, 305–318.

WORTSMAN, J., FRANK, S., WEHRENBERG, W. B., PETRA, P. H. & MURPHY, J. E. (1985) γ_3 melanocyte-stimulating hormone immuno-reactivity is a component of the neuroendocrine response to maximal stress (cardiac arrest). *Journal of Clinical Endocrinology and Metabolism*, **61**, 355–360.

YEREVANIAN, B. G. & WOOLF, P. D. (1983) Plasma ACTH levels in primary depression. *Psychiatry Research*, **9**, 45–51.

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Depression Assessment in Brazil
The First Application of the Montgomery-Åsberg Depression Rating Scale

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Cross-cultural investigation in psychiatry is revealing the need for standardised instruments in diagnosing and assessing depression. Recently, a new instrument was developed to evaluate depressed patients, namely the Montgomery-Åsberg Depression Rating Scale (MADRS). The present study introduced the MADRS in Brazil, comparing it to the Hamilton Depression Rating Scale, the Visual Analogue Mood Scale (a self-rating scale), and with the global clinical assessment of independent Brazilian psychiatrists. The results show correlation between MADRS and the three other assessments, indicating that it is a useful and operational instrument to evaluate depressed patients. They also support the application of the MADRS in cross-cultural studies of depression in Brazil and other countries. These results are critically discussed.

Adequate epidemiologic data are necessary for planning mental health care programs (Sartorius, 1976). Thus, standardised diagnostic criteria and rating scales seem to be operational instruments, as they allow a common language between researchers and practitioners, as well as comparative studies of mental diseases in different cultural settings (Jablensky *et al*, 1981; Sartorius *et al*, 1983).

Cross-cultural studies have revealed that depressive disorders constitute a public health problem in most societies. However, there is still little coordination among the various institutions and workers in this field (Sartorius, 1974; Jablensky *et al*, 1981). Thus, the introduction of cross-culturally applicable methods, such as depression rating scales (Hamilton, 1960, 1967; Beck *et al*, 1961; Zung, 1965; Carney & Sheffield, 1972; Folstein & Luria, 1973) would allow the conjugation of efforts in several countries. Since its introduction, the Hamilton Depression Rating Scale (HDRS) has had a widespread use and has been considered a standard instrument to assess depressive symptoms, and even to compare with other rating scales (Carroll *et al*, 1973). A few cross-cultural studies have analysed rating scales performance in different societies (Zung, 1969; Åsberg *et al*, 1973).

The Comprehensive Psychopathological Rating Scale (CPRS) is a rating scale, recently elaborated, to be sensitive to changes in symptoms induced by several psychiatric treatments (Åsberg *et al*, 1978). It was applied to English and Swedish patients (Montgomery *et al*, 1978), the most frequent symptoms of primary depressive disorders were identified, and finally rearranged as the Montgomery-Åsberg Depression Rating Scale (MADRS) (Montgomery & Åsberg, 1979). Therefore, the MADRS cross-cultural English and Swedish roots suggest its adequacy for studies on depression in different societies. Thus, it was applied to a Brazilian depressed population, and its performance compared with those of the HDRS, the Visual Analogue Mood Scale (VAMS), and the global clinical assessment (Dratcu *et al*, 1985).

The present paper reports on further data from the first application of the MADRS in Brazilian depressed patients, comparing its performance with those from the HDRS and VAMS. Furthermore, the rating scales evaluation will be compared with the independent clinical assessment of depression prevalent in a group of Brazilian psychiatrists.

Method

The study was carried out at the Department of Psychiatry and Medical Psychology, Hospital do Servidor Público Estadual "Francisco Morato de Oliveira", São Paulo, where there are three care modalities: in-patient, day-hospital and out-patient facilities.

Subjects were patients consecutively admitted by the staff-psychiatrists to the three care facilities, during a 6-month period, and diagnosed as depressed according to several clinical criteria. Their treatment and prescription were the staff-psychiatrists' responsibility. Those who, after being informed, consented to participate in the study were re-evaluated, within 1 week of admission, by two trained and independent psychiatrists. A total of 40 patients (60–70% of the interviewed depressed patients) met the criteria for Major Depressive Disorder (MDD) of the Research Diagnostic Criteria (RDC; Spitzer *et al*, 1980).

The re-evaluation interview, lasting an average of 75 min, included the application of three depression rating scales

Table I

Mean scores ± s.d. and variation range (in parens) of MADRS, HDRS, and VAMS of depressed patients

	MADRS	*HDRS*	*VAMS*
Evaluation			
First	38.5 ± 8.1	31.2 ± 6.1	20.7 ± 22.6
($n = 40$)	(23–55)	(12–41)	(88–0)
Second	12.9 ± 16.1*	12.0 ± 12.9*	61.5 ± 31.4**
($n = 10$)	(02–51)	(03–43)	(91–00)
Possible variation	0–60	0–52	100–0

The first evaluation was carried out within 1 week from beginning of treatment ($n = 40$), whereas the second ($n = 10$) occurred after week 4 of antidepressant therapy or at the end of an ECT series. Paired t-test, one-tailed of first and second evaluations: (*$P \leqslant 0.001$; **$P = 0.005$).

Table II

MADRS, HDRS and VAMS mean scores ± s.d. of depressed patients (n = 40) admitted, according to clinical assessment, to the hospital, day-hospital or out-patient clinic

	Hospital *(n = 18)*	*Day-hospital* *(n = 5)*	*Out-patient clinic* *(n = 17)*
MADRS	45.2 ± 5.6	36.0 ± 4.9*	32.2 ± 4.3*
HDRS	36.2 ± 3.3	27.4 ± 3.4*	27.1 ± 5.0*
VAMS	13.9 ± 19.1	17.8 ± 21.3	28.6 ± 23.8

Duncan's multiple range test showed difference between day-hospital and out-patient clinic groups compared with the hospital group (*$P < 0.01$)

to the patients who met the RDC criteria for depression: Montgomery-Åsberg Depression Rating Scale (MADRS), Hamilton Depression Rating Scale (HDRS) and a self-rating scale, the Visual Analogue Mood Scale (VAMS). A second re-evaluation (clinical global assessment and the three rating scales application), of ten of the 40 patients, was carried out 4 weeks from the beginning of antidepressant treatment or at the end of an electroconvulsive therapy (ECT) series.

The scores of the three rating scales were analysed taking the group mean and the standard-deviation. Correlation between the scales scores was obtained with the Pearson's correlation coefficient. MADRS, HDRS and VAMS mean scores from in-patient, day-hospital and out-patient groups were compared with the Duncan's Multiple Range Test (Steel & Torrie, 1960).

Results

Of the total 40 patients with the RDC diagnosis of Major Depressive Disorder (MDD), 30 were women and 10 were men. Their mean age was 53 ± 12.9 years (± standard deviation, s.d.), and ranged from 23 to 77 years. The subtypes of MDD were: primary ($n = 37$), secondary ($n = 3$), recurrent ($n = 17$), psychotic ($n = 8$), incapacitating ($n = 29$), endogenous ($n = 39$), agitated ($n = 11$), retarded ($n = 23$), situational ($n = 10$) and simple ($n = 37$). Eighteen were in-patients, five were being treated at the day-hospital facility, and seventeen were attending the out-patient clinic.

The mean scores of the rating scales at the first re-evaluation ($n = 40$) as well as the possible variation range, are shown in Table I. The highest MADRS and HDRS scores point towards a more severe depressive symptomatology, whereas the VAMS scores go the opposite way: lowest scores rate indicates worst mood. The MADRS mean score was 38.5 ± 8.1 (s.d.), with a variation from a minimal score of 23 to a maximal score of 55. The HDRS mean score was

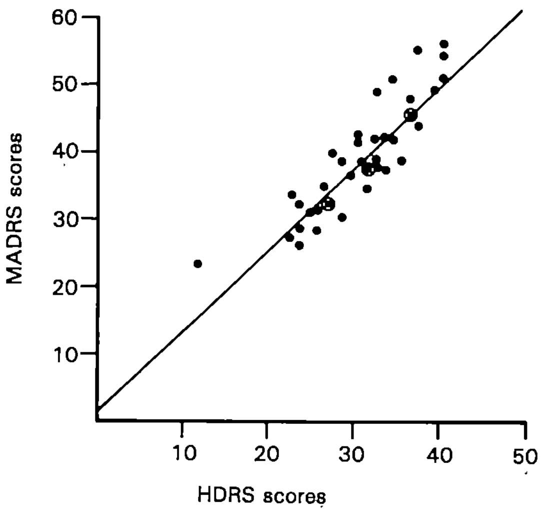

Fig. 1 Correlation ($r = 0.89$, $P < 0.01$) between the total scores of 40 patients assessed with the Hamilton Depression Rating Scale (HDRS), and the Montgomery-Åsberg Depression Rating Scale (MADRS).

31.1 ± 6.1 (s.d.); the lowest score was 12, and the highest 41. The VAMS mean score was 20.7 ± 22.6 (s.d.), with scores ranging from 88 through 0 mm. The correlation between MADRS and HDRS scores was positive and significant ($r = 0.89$, $P < 0.01$) and is shown in Fig. 1. The correlations between MADRS and VAMS, and HDRS and VAMS were, as expected, negative and significant ($r = -0.41$, $P < 0.02$; $r = -0.50$, $P < 0.01$, respectively).

The MADRS, HDRS and VAMS mean scores from the second re-evaluation ($n = 10$) are also shown in Table I. The scores obtained with the three scales were different from those of the first re-evaluation (paired t-test, one-tailed, MADRS: $t = 5.13$, HDRS: $t = 5.00$; VAMS: $t = 3.20$; $P \leq 0.005$) although the highest scores of the variation range remained almost the same. There was a significant and high correlation between the three scales scores: MADRS × HDRS ($r = 0.996$, $P < 0.01$) MADRS × VAMS ($r = 0.919$, $P < 0.01$) and HDRS × VAMS ($r = -0.925$, $P < 0.01$).

The MADRS and HDRS mean scores of the in-patient group were significantly higher than those obtained from the day-hospital and out-patient groups (Duncan's Multiple Range Test, $P < 0.01$) (Table II). There were no significant differences between MADRS and HDRS mean scores of day-hospital and out-patient groups, although the day-hospital group mean scores were higher than those of the out-patient group. The VAMS mean score of the in-patient group was lower than those of the day-hospital and out-patient groups, but these differences also did not reach statistical significance.

Discussion

The sex and age characteristics of the depressed patient sample studied correspond to those reported in the classical psychiatric literature.

All the patients, except one, met the RDC criteria for the endogenous subtype of Major Depressive Disorder. In fact, a careful analysis and the practical application of these diagnostic criteria led to the observation of an overlap of them for the diagnosis of Major Depressive Disorder and its endogenous subtype. Both the RDC and the Diagnostic and Statistical Manual of Mental Disorders, (3rd edition) (DSM-III, American Psychiatric Association, 1980) still constitute matter of discussion and controversy, and should not be used as a 'gold standard' for diagnosis (Klerman *et al*, 1984). It is feasible that the staff-psychiatrists have emphasised the features of endogenicity in diagnosing depression, as they are closer to the classical description of melancholia. Thus, non-endogenous depressed patients might have been previously excluded from this study. In addition, almost half of these patients had been admitted to the hospital, probably because they were considered by the staff-psychiatrists as severely depressed. Therefore, this study have included patients with more severe depression, perhaps due to the endogenous features of their depressive disorders. Anyway, the experimental design (double diagnostic selection), and the results found (sex and age distribution, predominance of endogenous depression, and high HDRS mean score) point towards the reliability of the diagnosis of depression.

The high correlation between MADRS and HDRS scores indicates that both scales are equally consistent instruments to evaluate the intensity of depressive symptoms. However, some differences between them seem important. The HDRS has 17 items while the MADRS only has 10, and is apparently simpler. Every MADRS item rates on a 6-point scale, and there are intermediate points between them. Thus, the rater has more flexibility to assess and decide on the rating of those symptoms which do not correspond exactly to those described. Furthermore, the MADRS discriminates between observed and reported symptoms avoiding doubts which could mislead the evaluation. In addition, the MADRS does not emphasise somatic symptoms as much as the HDRS does, therefore minimising the interference of organic disfunctions or treatment side-effects. Finally, the MADRS does not present contradictory items, such as those found in the HDRS (e.g. inhibition and agitation), which make virtually impossible the maximal score (52 points). The HDRS highest score in this study was 41 (79% of the maximal possible score), whereas the MADRS highest score was 55 (92% of the maximal possible). Some of these HDRS limitations have been already observed, discussed, and modifications made to improve it (Bech *et al*, 1981; Miller *et al*, 1985). Thus, the MADRS would be more sensitive to subtle and earlier changes in symptoms. In fact, it might contribute to its capacity to evaluate treatment responses, specially of severely depressed patients, with a higher precision and precedence. It confirms its sensitivity to treatment-induced changes in symptoms, the main purpose of the scale developers (Montgomery & Åsberg, 1979).

The negative correlation between the VAMS and the Montgomery-Åsberg and Hamilton Scales, applied by the raters, indicates that both follow the patients self-rating. However, the high self-rating standard deviation value reveals the individual variability, as the way of filling in the VAMS differs from one patient to the other.

There was a decrease in intensity of symptoms, as might have been expected, in the mean scores of the three scales applied to ten of the 40 patients during the post-treatment re-evaluation. Eight patients improved with tricyclic antidepressants or electroconvulsive therapy, whereas the remaining two (one received an antipsychotic, and the other nomifensine) did not. Consequently, their MADRS and HDRS scores have not changed. It explains the practically unchanged high value of the variation range as well as the high standard deviation.

The higher correlation between the scales found at the re-evaluation was obviously due to the decrease of number and intensity of the remaining symptoms. Therefore, the three scales were sensitive enough to evaluate treatment-induced changes. Nevertheless, the MADRS and the HDRS seem to offer advantages, as they allow to a better quantification of these changes.

The global clinical assessment of depression is reflected on the staff-psychiatrists' choice of the treatment care program. The patients considered as severely depressed were admitted to the hospital, those with a moderate depression were admitted to the day-hospital, and those with a mild depression were followed at the out-patient clinic. The MADRS, HDRS and VAMS scores accompanied the staff-psychiatrists clinical assessment, as the rated symptoms intensity was indeed in the same order of patients grouping. The mean scores differences did not reach statistical significance for all the groups probably because of the limited sample size. Thus, if the sample size were increased, the found correlation between the global clinical assessment and the three rating scales scores would become more evident.

Finally, the Montgomery-Åsberg Depression Rating Scale, as adequate as the Hamilton Depression Rating Scale, was a useful instrument to assess depression in a Brazilian depressed population sample, in spite of having been developed in an European context. Furthermore, it has some practical advantages, such as higher simplicity, specificity and sensibility than the HDRS. Therefore, it might be considered a valid instrument for research information exchange, as well as for cross-cultural studies on depression in Brazil and other countries.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual*, 3rd ed. Washington DC: APA.

Åsberg, M., Kragh-Sorensen, P., Mindham, R. H. S. & Tuck, J. R. (1973) International reliability and communicability of a rating scale for depression. *Psychological Medicine*, 3, 458–465.

——, Montgomery, S., Perris, C., Schalling, D. & Sedwall, G. (1978) The CPRS: development and application of a psychiatric rating scale. *Acta Psychiatrica Scandinavica* (suppl), 271, 5–27.

Bech, P., Allerup, P., Gram, L. F., Reisby, N., Rosenberg, R., Jacobsen, O. & Nagy, A. (1981) The Hamilton Depression Scale. Evaluation of objectivity using logistic models. *Acta Psychiatrica Scandinavica*, 63, 290–299.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. & Erbaugh, J. (1961) An Inventory for Measuring Depression. *Archives of General Psychiatry*, 4, 561–571.

Carney, M. W. P. & Sheffield, B. F. (1972) Depression and the New Castle Scale: their relationship to Hamilton's Scale. *British Journal of Psychiatry*, 121, 35–40.

Carroll, B. J., Fielding, J. M. & Blashki, T. G. (1973) Depression rating scales. A critical review. *Archives of General Psychiatry*, 28, 361–366.

Dratcu, L., Costa Ribeiro, L. & Calil, H. M. (1985) Escalas de avaliação da depressão e sua utilidade clínica: Hamilton, Montgomery-Åsberg e Visual Análoga do Humor. *Revista da Associação Brasileira de Psiquiatria*, 7, 59–65.

Folstein, M. F. & Luria, R. (1973) Reliability, validity, and clinical application of the visual analogue mood scale. *Psychological Medicine*, 3, 479–486.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, 23, 56–62.

—— (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, 6, 278–296.

Jablensky, A., Sartorius, N., Gulbinat, W. & Ernberg, G. (1981) Characteristics of depressive patients contacting psychiatric services in four cultures. *Acta Psychiatrica Scandinavica*, 63, 367–383.

Klerman, G. L., Vaillant, G. E., Spitzer, R. L. & Michels, R. L. & Michels, R. (1984) A Debate on DSM III. *American Journal of Psychiatry*, 141, 539–553.

Miller I. W., Bishop, S., Norman, W. H. & Maddever, H. (1985) The Modified Hamilton Rating Scale for Depression: reliability and validity. *Psychiatry Research*, 14, 131–142.

Montgomery, S. & Åsberg, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, 134, 382–389.

——, ——, Traskman, L. & Montgomery, D. (1978). Cross-cultural studies on the use of the CPRS in English and Swedish patients. *Acta Psychiatrica Scandinavica* (suppl), 272, 33–39.

Sartorius, N. (1974) Depressive illness as a worldwide problem. In *Depression in Everyday Practice*, (ed. P. Kielholz). Bern: Hans Huber Publishers.

—— (1976) Classification: an international perspective. *Psychiatric Annals*, 6, 24–35.

——, Davidian, H., Ernberg, G., Fenton, F. R., Fujii, I. *et al* (1983) Depressive Disorders in different cultures. Report on the WHO Collaborative study on "standardized assessment of depressive disorders" (SADD). Geneva: World Health Organization.

Spitzer, R. L., Endicott, J. & Robins, E. (1980) Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders, 3rd ed. New York: Research Assessment and Training Unit, New York State Psychiatric Institute.

Steel, R. G. D. & Torrie, J. H. (1960) *Principles and Procedures of Statistics with special reference to the biological sciences*. New York: McGraw Hill.

Zung, W. W. K. (1965) A self-rating depression scale. *Archives of General Psychiatry*, 12, 63–70.

—— (1969) A cross-cultural survey of symptoms in depression. *American Journal of Psychiatry*, 126, 116–121.

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Mental Health and Psychological Well-being Among the Old and the Very Old Living at Home

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Using four brief assessment scales, profiles of mental health and psychological well-being were obtained from 507 old (aged 65-74 years) and 535 very old (aged 75+) individuals randomly sampled from the community. Assessments of dementia and depression were subsequently validated against diagnostic ratings made by experienced psychogeriatricians. Levels of agreement between psychometric and clinical ratings of dementia ($\kappa=0.83$) and depression ($\kappa=0.66$) were satisfactory. The old and very old groups reported similar levels of anxiety and personal disturbance, and showed a similar prevalence of depression. However, those aged 75+ showed higher levels of dementia and significantly lower levels of social involvement and morale.

Epidemiological studies of mental disorders among the elderly are becoming increasingly important in guiding policy making and service provision (Kay & Bergmann, 1980; Arie & Jolley, 1982; Henderson & Macfadyen, 1985). Instruments for assessing mental health in the aged vary greatly in content, detail, and administration time, but it is generally agreed that brief screening tests are clinically useful in case identification and in monitoring change over time in known cases (Hinton & Withers, 1971; Gurland, 1980; Blessed, 1985). In the present study extensive use has been made of brief rating scales to assess cognitive and affective status and social functioning in a large representative sample of elderly individuals living in the community.

While there is broad agreement concerning the value of screening for mental disorders among the elderly, there appears to be less agreement concerning the appropriate age structure of target populations for such assessments. It is widely acknowledged that the group traditionally defined as elderly (i.e. that section of the community at or above statutory retirement age) is neither clinically nor socially homogeneous. When such a group is further divided into older and younger subgroups, dementing illness (e.g. Kay & Bergmann, 1980), social deprivation (e.g. Abrams, 1978) and dependency (e.g. Hunt, 1978) are more common among the 'older' elderly. Nevertheless, in only a small minority of epidemiological studies in geriatric psychiatry are the old, and the very old considered separately (see Kay & Bergmann, 1980; Henderson, 1986). Rather, descriptive statistics are frequently reported only for a single group comprising all those above a criterion age, usually 60, 65 or 70 years. In Britain, data from the 1981 census show 4.93 million individuals aged 65-74 years compared with 3.05 million aged 75 years and over - an old to very old ratio of 1.62:1 (OPCS, 1983). Simple random selection of those over the age of 65 years ensures that the oldest and most dependent individuals are numerically under-represented. Selection of the present sample, therefore, was such as to allow those aged 65-74 years, and those aged 75 years and over to be equally represented.

In addition to the assessment of mental health the present survey also focused upon morale and social functioning, with the intention of constructing a multidimensional profile of 'psychological well-being' for each respondent. This research forms part of a longitudinal project the principal aim of which is to evaluate relationships between levels of customary physical activity and well-being in old age. Assessments of mental competence, depression, life-satisfaction, and social functioning were accomplished using rating scales administered in the course of a structured interview. Standardised instruments were used wherever possible, and psychometric ratings of mental competence and mood were subsequently validated against diagnostic ratings made by experienced psychogeriatricians. In this paper, we present evidence for the reliability and validity of the brief assessment scales and describe (a) the prevalence of psychiatric disorders, and (b) variations in psychological wellbeing and social functioning within representative samples of 'young' old and very old elderly individuals living at home.

Method

Instruments

Mental competence was assessed using the 12-item Information/Orientation scale from the Clifton Assessment Procedures for the Elderly (CAPE: Pattie & Gilleard, 1979). Although originally developed for use in institutional settings (Pattie & Gilleard, 1975, 1976) this scale has been successfully employed in surveys of the elderly at home (Clarke *et al*, 1984). To improve social acceptability in the present study, items 1-6 of the scale (e.g. name, address, date of birth) were presented at the beginning of the interview, while the remaining items (e.g. name of the British Prime Minister, name of the US President) were presented later, when a rapport between interviewer and respondent had developed. If any of the first six items were incorrectly answered, however, the interviewer was instructed to continue immediately with items 7-12. The scale yields a maximum score of 12. Scores of 7 and below are associated with dementia or acute organic brain syndrome (Pattie & Gilleard, 1979). If, after appropriate prompting, the respondent failed to achieve a minimum Information/Orientation score of 8 and, in the opinion of the interviewer, was unlikely to respond reliably to the remaining questionnaire items, the interview was discontinued.

The Symptoms of Anxiety and Depression scale (SAD: Bedford *et al*, 1976) was used to assess functional mental disorders. Derived from the Delusions, Symptoms and States Inventory (Foulds & Bedford, 1979), the SAD scale focuses exclusively on recent symptomatology and comprises two subscales of seven items relating to depression and anxiety respectively. Items are scored 0, 1, 2 and 3 according to the degree of personal disturbance reported for each (i.e. none, a little, a lot, and unbearable respectively). In non-elderly populations, Bedford *et al* (1976) suggest that an overall score of 6 or more indicates psychiatric illness, while scores of 4 or more on either the depression or anxiety subscales indicate membership of that set. The SAD scale has previously been used as a screening instrument for functional disorders among the very elderly (McNab & Philip, 1980).

Measurements of morale were provided by Wood *et al*'s (1969) 13-item version of Neugarten *et al*'s (1961) Life Satisfaction Index (the LSIZ). To improve comprehension of this American scale in a British sample, the word form of some of the items was, in the present study, slightly modified (for example item 2 "I have gotten more of the breaks in life than most of the people I know" was changed to "I have had more chances in life than most of the people I know"). Scores of 2 for a 'right' answer, 0 for a 'wrong' answer, and 1 for a 'don't know' were assigned to each item (see Wood *et al*, 1969).

Of particular interest in the present study was the degree to which elderly individuals engage actively (e.g. voting, going on holiday) or symbolically (e.g. watching television, listening to the radio) in the social milieu. In the absence of a standardised instrument for assessing social functioning a Social Engagement Scale was developed as follows. Items judged relevant to the assessment of social involvement were included in the structured interview. Of these items 13 were derived from a prototype scale the validity and reliability of which is described elsewhere (Morgan *et al*, 1985). All items were dichotomously scored, 1 for a positive response and 0 for a negative response. Items were then removed from the scale (a) if positive answers were obtained from less than 2% of the survey population (i.e. if that item showed too little variance) and (b) if it correlated less than 0.2 with the sum of the remaining items, and its removal substantially increased the value of the reliability coefficient alpha (Cronbach, 1951). This procedure resulted in the 20-item scale shown in Table I.

TABLE I
Social Engagement Scale items (abbreviated)

1. Has access to a telephone
2. Has made a personal telephone call in the last week
3. Reads a newspaper or magazine
4. Attends religious services, gatherings, or meetings
5. Voted in the last general/local/European election
6. Has been away on holiday in the last year
7. Is planning to go on holiday within the next year
8. Uses public library
9. Attends meetings of clubs, societies etc.
10. Has senior citizen's rail card
11. Has access to a car
12. Has television and radio
13. Is employed or engaged in voluntary work
14. Has sufficient contact with family and friends
15. Shares accommodation
16. Is mobile (ambulant/non-ambulant)
17. Gets out and about as much as desired
18. Has one or more friends in district
19. Is infrequently lonely
20. Knows at least one person to approach for help

The sample

The survey was conducted in three geographically separate districts of Nottingham each within the psychogeriatric catchment area of the University Department of Health Care of the Elderly. Prior demographic analyses had established that the age, sex, and social class structure, and the proportion of the elderly individuals living alone within the survey area as a whole showed no significant departure from the national pattern as described by the 1981 census (OPCS, 1983). With the consent and cooperation of general practitioners a register of elderly persons living within the survey area was compiled from patient records maintained by the Nottinghamshire Family Practitioner Committee. All general practitioners with surgeries in the three survey areas cooperated. In this way a total population of 8409 individuals aged 65 years and over (4856 aged 65-74 years; 3553 aged 75+ years) was registered, representing the combined patients of 25 general practitioners in nine practices. While practice and census ward boundaries do not necessarily coincide, the number of elderly individuals identified in the survey area closely approximated the number provided by the 1981 census. Equal probability

TABLE II
Survey sample by age and sex

	65–74	*75+*	*Males*	*Females*	*Total*
Target sample	747	852	586	1013	1599
Losses					
deaths	29	68	44	53	97
change of address etc.	85	118	73	130	203
Effective sample	633	666	469	830	1299
refusals	126	131	63	194	257
Interviewed sample	507	535	406	636	1042

target samples were then selected from the old (i.e. those aged 65–74 years) and the very old (i.e. those aged 75 years and over) subpopulations. Those in institutional care were excluded from the target sample. As the intention was to interview approximately 500 individuals from both subpopulations, the samples drawn were large enough to accommodate losses due to deaths, migration, etc.

Procedure

Interviews were conducted between 7 May and 24 September 1985 by ten women aged 27–56 years. Each interviewer received 2 weeks of intensive classroom based training, and a further 2 weeks of close supervision in the field. Before being interviewed respondents were first contacted by letter and invited to participate in the survey. After a minimum of 3 clear days, each was then visited by an interviewer. The structured questionnaire contained a total of 318 items covering aspects of health and lifestyle and with the consent of the respondent, was followed by measurements of right and left handgrip strength, weight, shoulder flexibility, and stature. Previous experience had shown that interviews could be completed in 30–90 minutes.

Validation of dementia and depression ratings

In order specifically to assess the accuracy of psychometric classification in the present study, and generally to assess the value of the Information/Orientation and SAD scales as screening instruments, selected ratings of dementia and depression were subsequently validated against clinical diagnoses. On completion of the survey respondents were classified as (a) Cognitively Impaired, (b) Borderline Impaired, (c) Depressed, or (d) Normal. Classification was as follows:

(a) Using the cut-off point recommended by Pattie & Gilleard (1979) for identifying cases of dementia, respondents with an Information/Orientation score of $\leqslant 7$ were classified as Cognitively Impaired.

(b) Respondents with Information/Orientation scores of 8 or 9, and who were not rated as depressed (see below) were classified as Borderline Impaired. These Information/Orientation scores fall between the thresholds for impairment ($\leqslant 7$) and non-impairment (>10) suggested by Pattie & Gilleard (1979).

(c) Using the cut-off points suggested by Bedford *et al* (1976), respondents with a total SAD score of $\geqslant 6$, and a depression subscale score of $\geqslant 4$, and who were not rated as Borderline Impaired, were classified as Depressed. Effectively, this procedure identified those who showed significant personal disturbance characterised by symptoms of depression, but who were cognitively intact (fourteen respondents whose scores indicated both Depression *and* Borderline Impairment were unclassified. These individuals will be considered in a separate report.)

(d) Those with Information/Orientation scores of 10, 11 or 12, and who were not rated as depressed were classified as Normal.

Groups were then randomly selected to represent the Cognitively Impaired, Borderline Impaired, and Depressed classes. For the Normal (control) group, individuals were matched for age (+ or − 5 years) and sex with the Impaired group. Respondents thus selected were visited at home and clinically assessed by an experienced psychogeriatrician who was 'blind' to the survey classifications. Evidence for dementia or depression respectively was rated by each clinician on a 4-part scale as follows: 0 – No Evidence; 1 – A Little Evidence; 2 – A Lot of Evidence; 3 – Outstanding Evidence. If appropriate, ratings of severity were then completed and scored: 1 – Mild (dementia/depression); 2 – Moderate; 3 – Severe. Wherever possible clinical diagnostic assessments were carried out within 12 weeks of the original survey interview.

Results

The sample

Losses from the original target sample of 1599, mostly due to deaths and changes of address totalled 300, resulting in an effective target sample of 1299 individuals (see Table II). Of these, 1042 (507 aged 65–74 years; and 535 aged 75 years and over) were interviewed – a response rate of 80.3%. The age and sex structure of the interviewed sample is also shown in Table II.

Internal consistency of the assessment scales

All four brief assessment scales showed acceptable levels of internal consistency. α-Coefficients (Cronbach, 1951), and the number of respondents completing each scale were as follows: 12-item Information/Orientation Scale, $n=1022$, $\alpha=0.84$; 14 item SAD scale, $n=979$, $\alpha=0.85$; 13-item LSIZ, $n=986$, $\alpha=0.74$; 20-item Social Engagement Scale, $n=971$, $\alpha=0.70$.

Criterion validity of dementia and depression ratings

Clinical diagnostic ratings were obtained for 20 respondents classified as Cognitively Impaired, 14 classified as Borderline Impaired, 25 classified as Depressed, and 20 classified as Normal. The smallest group – Borderline Impaired – included all the respondents thus classified. The correspondence between survey classifications of Cognitive Impairment and Depression, and clinical ratings of

Table III

Agreement between clinicians' and survey ratings of dementia[1] and depression[1] among 65 selected respondents

Survey Scale Ratings	*Clinicians' Ratings* *Dementia* *No. %*	 *Depression* *No. %*	 *Normal* *No. %*
Cognitively Impaired (demented)	18 (0.28)	0	2 (0.03)
Depressed	3 (0.05)	16 (0.25)	6 (0.09)
Normal	0	1 (0.02)	19 (0.29)

Total $n = 65$

Overall agreement = 81.5%; $\kappa = 0.73$, $P < 0.001$

1. Mild, moderate, or severe

dementia (mild, moderate or severe) and depression (mild, moderate or severe) was assessed using the κ statistic (Cohen, 1960). Table III shows an overall level of agreement of 81.5% with a κ-coefficient of 0.73 ($P < 0.001$). Ratings of dementia and depression were then considered individually by collapsing these data into fourfold tables comparing the dichotomous categories of dementia/not dementia and depression/not depression. For dementia ratings, the analysis showed an overall agreement of 92.3% with a κ-coefficient of 0.83 ($P < 0.001$). Preliminary analyses of the survey and clinical ratings of depression showed that the cut-off scores suggested by Bedford *et al* (1976) possibly overestimate psychiatric morbidity in the elderly (see Table III). A higher cut-off score was therefore selected to reduce total misclassifications, namely, a total SAD score of $\geqslant 8$ with a depression subscale score of $\geqslant 6$. Using this adjusted threshold, clinical and survey ratings showed an overall agreement of 84.6% with a κ-coefficient of 0.66 ($P < 0.001$).

Rank order correlation coefficients showed a highly significant association between Information/Orientation scores in the combined Cognitively Impaired and Borderline Impaired groups, and clinical ratings of evidence for ($n = 34$, $\rho = 0.60$, $P < 0.001$) and severity of ($n = 34$, $\rho = 0.61$, $P < 0.001$) dementia. Within the Depressed group, survey scale scores and clinical ratings were less consistently associated. While a weak correlation obtained between overall SAD scores and clinical ratings of evidence for depression ($n = 25$, $\rho = 0.43$, $P < 0.05$), the correlation between SAD scores and ratings of the severity of depression failed to reach significance ($n = 25$, $\rho = 0.32$, $P = 0.08$).

Prevalence of dementia and depression

When, for the purposes of statistical description, the old and the very old subgroups were combined, the older subgroup was appropriately weighted so that the age structure of the combined subgroups approximated that of a true (i.e. non-stratified) random sample. The weighted distribution of Information/Orientation scores is shown in Table IV. Evidence of significant cognitive impairment, as defined by a scale score of 7 or less was present in 3.2% of the weighted sample. The distribution of Information/Orientation scores above and below this cut-off point according to age and sex is shown in Table V.

The distributions of SAD anxiety and depression subscale scores are shown in Table VI and VII respectively. Combining both age groups, 9.8% of the weighted sample met the survey criteria for a psychometric classification of depression (a total SAD score of $\geqslant 6$, and a depression subscale score of $\geqslant 4$). Within the older and younger subgroups 10.0% of those aged 65–74 years, and 9.5% of those aged 75+ were classified as depressed. Using the more conservative criteria which had maximised agreement between clinical and survey ratings (namely a total SAD score of $\geqslant 8$, with a depression subscale score of $\geqslant 6$), 4.9% of the weighted sample were classified as depressed – 4.5% of those aged 65–74 years, and 5.5% of those aged 75 years and over. Total SAD scores for the older and younger subgroups, compared using the Mann-Whitney statistic (Siegel, 1956), showed no significant difference ($Z = 0.94$, $P = 0.34$).

Table IV

Weighted distribution of Information/Orientation scores for the entire interviewed sample

Scale score	*Weighted frequency*	*Cumulative frequency (%)*
0	1	0.1
1	1	0.2
2	2	0.5
3	2	0.8
4	3	1.1
5	3	1.4
6	4	1.9
7	11	3.2
8	6	4.0
9	8	5.0
10	23	7.9
11	113	21.8
12	637	100.0
	814[1]	

1. Weighted total

Life satisfaction and social engagement

The distribution of raw scores from the Life Satisfaction Index and, to a lesser extent, the Social Engagement Scale were negatively skewed (skew coefficients = −0.65 and

Table V

Prevalence (%) of cognitive impairment (as defined by an information/orientation score of $\leqslant 7$) by age and sex

Age group	*Males*	*Females*	*Overall*
65+	1.6*	4.4*	3.2*
65–74	1.4	2.1	1.8
75+	2.3	7.2	5.6

1. Weighted population estimates

TABLE VI
Distribution of anxiety subscale scores by age-group

	65–74		75+		Overall[1]	
Score	*No*	*Cumulative %*	*No*	*Cumulative %*	*No*	*Cumulative %*
0	241	(49.0)	228	(46.4)	378	(48.0)
1	82	(65.7)	87	(64.2)	134	(65.1)
2	58	(77.4)	69	(78.2)	99	(77.7)
3	34	(84.3)	29	(84.1)	51	(84.3)
4	25	(89.4)	20	(88.2)	37	(89.0)
5	13	(92.1)	27	(93.7)	29	(92.7)
6	10	(94.1)	11	(95.9)	17	(94.8)
7	9	(95.9)	7	(97.4)	13	(96.5)
⩾8	20	(100.0)	13	(100.0)	29	(100.0)

1. Weighted estimates

TABLE VII
Distribution of depression subscale scores by age-group

	65–74		75+		Overall[1]	
Score	*No*	*Cumulative %*	*No*	*Cumulative %*	*No*	*Cumulative %*
0	342	(69.8)	331	(67.7)	541	(69.0)
1	52	(80.4)	54	(78.7)	84	(79.8)
2	30	(86.5)	29	(84.7)	47	(85.8)
3	17	(90.0)	24	(89.6)	31	(89.8)
4	20	(94.1)	14	(92.4)	28	(93.5)
5	6	(95.3)	9	(94.3)	11	(94.9)
6	6	(96.5)	7	(95.7)	10	(96.2)
7	5	(97.6)	5	(96.7)	8	(97.2)
⩾8	12	(100.0)	16	(100.0)	23	(100.0)

1. Weighted estimates

TABLE VIII
Life Satisfaction and Social Engagement scores for the old (65–74) and very old (75+) subgroups

	65–74			75+			Mann-Whitney
	mean	*(s.d.)*	*median*	*mean*	*(s.d.)*	*median*	*(Z)*
Life Satisfaction Index (scale range 0–26)	17.1	(5.6)	18	16.4	(5.5)	17	2.18*
Social Engagement Scale (scale range 0–20)	13.0	(2.8)	13	11.2	(3.2)	11	9.12**

*$P<0.05$; **$P<0.001$

−0.39 respectively). Nevertheless, while scores tended to cluster at the higher end of the distribution (i.e. greater Life Satisfaction and more Social Engagement) the Life Satisfaction Index was active throughout its range, and scores on the Social Engagement Scale ranged from 2 to maximum. Total scale scores for the age subgroups 65–74 and 75 years and over were compared using the Mann-Whitney statistic. For both Life Satisfaction ($Z=2.18$, $P<0.05$) and Social Engagement ($Z=9.12$, $P<0.001$) scores were significantly higher in the younger subgroup (Table VIII). As regards Life Satisfaction, Table VIII also provides normative data for a British sample for this variant of the widely used Life Satisfaction Index.

Relatively strong correlations obtained between Life Satisfaction and Social Engagement scores ($\varrho=0.49$, $P<0.001$), and also between Life Satisfaction and SAD scores ($\varrho=-0.49$, $P<0.001$). While significant, the correlation between engagement and SAD scores was rather modest ($\varrho=-0.27$, $P<0.001$).

Discussion

Satisfactory response rates, calculated as a percentage of the effective sample (see Table II) were achieved for both the old (80.1%) and the very old (80.5%) subgroups. Of the 257 individuals who refused to be interviewed 108 (42%) returned short postal questionnaires which had requested information on their marital status, health status, occupation (or husband's occupation) before retirement, and reasons for refusing. Insufficient time, a desire for privacy, and lack of interest were the most frequently expressed reasons for not wishing to participate in the study. Overall, results from these postal questionnaires provided no evidence that the cognitively or physically impaired were over-represented among the interview refusals.

All four psychometric scales displayed levels of internal consistency consonant with their use as survey instruments. Both the Information/Orientation and SAD scale classifications showed significant levels of agreement with clinical ratings of dementia/not dementia and depression/not depression respectively. Concordance between survey and clinical ratings of Cognitive Impairment-Dementia were particularly high. Only two of the 20 individuals in the Cognitively Impaired class were rated 'not demented' by the clinicians (see Table III). Of these two, one, a man aged 71, was described by the psychogeriatrician as 'educationally subnormal'. The only other misclassification was a woman aged 83 with an Information/Orientation score of 7. Fourteen individuals were classified as Borderline Impaired and of these, nine (64.3%) were subsequently rated as demented (eight as 'mild' and one as 'severe') by the clinicians. A high level of dementia in this class was not unexpected. The Borderline Impaired group included those individuals for whom, on the basis of their Information/Orientation scores, serial re-assessment is recommended in order to "... clarify diagnostic uncertainty" (Pattie & Gilleard, 1979). Our findings support this recommendation. The relationships shown here between survey classifications of Cognitive/Borderline Impairment and clinical ratings of dementia indicate that the 12-item Information/Orientation scale can effectively identify cases and probable cases of dementia among the elderly living at home.

Relatively lower levels of agreement were found between survey classification and clinical ratings of depression. In part, this may have been due to the sometimes lengthy interval between the administration of the SAD scale, with its emphasis on recent symptomatology, and subsequent clinical assessments. While most of the clinical assessments were conducted within 3 months of the original survey interview, a minority were conducted some 4 months later. There was evidence in the present study that concordance between survey and clinical ratings of depression was related to the degree of personal disturbance originally reported, suggesting that the stability of depressive states over time may be related to severity. Overall SAD scores among those for whom survey and clinical ratings of depression were concordant ($n=16$, mean SAD score $=16.8$) were significantly higher than those for whom survey and clinical ratings were discordant ($n=9$, mean SAD score $=12.3$; Mann-Whitney $Z=1.93$, $P=0.05$). Nevertheless, the scale used in the manner described here identified clinically significant depression in this elderly community sample with a probability well above chance.

Table V shows a weighted overall prevalence rate of cognitive impairment/dementia of 3.2%. Given that 64.3% of those with borderline Information/Orientation scores of 8 or 9 were subsequently rated as demented by psychogeriatricians, the 'true' prevalence of dementia falls within the score range 7–9 which, in the present study, includes between 3.2% and 5.0% of the sample (see Table IV). Adding the Borderline Impaired individuals who were clinically rated as demented ($n=9$ to those whose Information/Orientation score was $\leqslant 7$ yields a weighted overall prevalence estimate of 4.3% (2.4% of the men, and 5.5% of the women). This estimate falls between the rates of 'pervasive dementia' reported by Gurland *et al* (1983) for similar samples of elderly people living at home in London (2.3%) and New York (4.9%). In both age subgroups the present data show a higher prevalence of impairment among females. While this may result from selective institutionalisation, future clinical studies will examine the possibility of a genuine sex difference in prevalence.

The overall weighted prevalence of depression (9.8%) found in the present study also approaches the rates of 'pervasive depression' reported by Gurland *et al* (1983) for their London (12.4%) and New York (13.0%) samples. In contrast with the findings of Kay *et al* (1985) for a community sample in Hobart, Tasmania, we found little difference in the prevalence of depression between the younger and older subgroups. However, when the SAD cut-off points were raised to include only those (presumably more severe cases) with a total score of $\geqslant 8$ with a depression subscale score of $\geqslant 6$, a greater prevalence among those aged 75+ did emerge (4.5% versus 5.5%).

While the old and the very old subgroups showed similar levels of depression, anxiety, and overall 'disturbance', life satisfaction was significantly lower among those aged 75+. Nevertheless, the significant correlation between SAD and LSIZ scores does indicate that the scales assess some common aspects of well-being. Levels of social engagement were also significantly lower among the older subgroup. It is interesting to note that the strong correlation between social engagement and LSIZ scores is consistent with an activity-affect relationship in old age. That the correlation between the engagement and total SAD scores was relatively weaker further suggests that the influence of this relationship does not extend equally to all domains of psychological well-being. As expected, increasing levels of psychiatric disturbance (as measured by the total SAD scores) were associated with decreasing levels of morale.

The present study has demonstrated the usefulness of four brief assessment scales in providing a profile of psychological well-being in the old and the very old living at home. In addition the survey, conducted within a defined psychogeriatric catchment area, provided information of immediate value to the clinical department of Health Care of the Elderly. Two cases of severe dementia with no previous contact with primary care services were referred to general practitioners following research clinical assessment by psychogeriatricians. A further two cases of depression were similarly referred. Thus, the present study illustrates the value of screening elderly populations and also emphasises the benefits of combining clinical and research interests in screening programmes.

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References

Abrams, M. (1978) *Beyond Three-Score and Ten*, Mitcham, Surrey: Age Concern Publications.

Arie, T. & Jolley, D. J. (1982) Making services work: Organisation and style of psychogeriatric services. In *Psychiatry of Later Life*, (eds R. Levy & F. Post). Oxford: Blackwell Scientific.

Bedford, A., Foulds, G. A. & Sheffield, B. F. (1976) A new personal disturbance scale. *British Journal of Social and Clinical Psychology*, **15**, 387–394.

Blessed, G. (1985) Measurement in psychogeriatrics. In *Recent Advances in Psychogeriatrics*, (ed. T. Arie). Edinburgh: Churchill Livingstone.

Clarke, M., Clarke, S., Odell, A. & Jagger, C. (1984) The elderly at home: health and social status. *Health Trends*, **16**, 3–7.

Cohen, J. (1960) A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, **20**, 37–46.

Cronbach, L. J. (1951) Coefficient alpha and the internal structure of tests. *Psychometrika*, **16**, 297–334.

Foulds, G. A. & Bedford, A. (1979) *Manual of the Delusions – Symptoms – States Inventory*. Windsor: NFEF Nelson.

Gurland, B. (1980) The assessment of the mental health status of older adults. In *Handbook of Mental Health and Aging*, (eds J. E. Birren & R. B. Sloan). New York: McGraw-Hill.

——, Copeland, J., Kuriansky, J., Kelleher, M., Sharpe, L. & Dean, L. L. (1983) *The Mind and Mood of Ageing*. London: Croom Helm.

Henderson, A. S. (1986) The epidemiology of Alzheimer's disease. *British Medical Bulletin*, **42**, 3–10.

Henderson, J. H. & Macfadyen, D. M. (1985) Psychogeriatrics in the programmes of the World Health Organization. In *Recent Advances in Psychogeriatrics*, (ed. T. Arie). Edinburgh: Churchill Livingstone.

Hinton, J. & Withers, E. (1971) The usefulness of clinical tests of the sensorium. *British Journal of Psychiatry*, **119**, 9–18.

Hunt, A. (1978) *The Elderly at Home*. London: HMSO.

Kay, D. W. K. & Bergman, K. (1980) Epidemiology of mental disorders among the aged in the community. In *Handbook of Mental Health and Aging*, (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

——, Henderson, A. S., Scott, R., Wilson, J., Rickwood, D. & Grayson, D. A. (1985) Dementia and depression among the elderly living in the Hobart community: the effect of the diagnostic criteria on the prevalence rates. *Psychological Medicine*, **15**, 771–788.

McNab, A. & Philip, A. E. (1980) Screening an elderly population for psychological well-being. *Health Bulletin*, **38**, 160–162.

Morgan, K., Dallosso, H. M. & Ebrahim, S. B. J. (1985) A brief self-report scale for assessing personal engagement in the elderly. In *Ageing: Recent Advances and Creative Responses*, (ed. A. Butler). London: Croom Helm.

Neugarten, B. L., Havighurst, R. J. & Tobin, S. S. (1961) The measurement of life satisfaction. *Journal of Gerontology*, **16**, 134–143.

Office Of Population Censuses And Surveys (1983) *Census 1981: Sex, age and marital status, Great Britain*. London: HMSO.

Pattie, A. H. & Gilleard, C. J. (1975) A brief psychogeriatric assessment schedule – validation against psychiatric diagnosis and discharge from hospital. *British Journal of Psychiatry*, **12**, 489–493.

—— & —— (1976) The Clifton Assessment Schedule – further validation of a psychogeriatric assessment schedule. *British Journal of Psychiatry*, **129**, 68–72.

—— & —— (1979) *Manual of the Clifton Assessment Procedures for the Elderly (CAPE)*. Sevenoaks, Kent: Hodder & Stoughton.

Siegel, S. (1956) *Nonparametric Statistics for the Behavioural Sciences*. New York: McGraw-Hill.

Wood, V., Wylie, M. & Sheafor, B. (1969) An analysis of a short self-report measure of life satisfaction: correlation with rater judgements. *Journal of Gerontology*, **24**, 465–469.

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Comparison of the Sensitivity of Three Instruments for the Detection of Cognitive Decline in Elderly Living at Home

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We followed up 181 elderly living at home over 2 years. The changes shown on a brief dementia rating scale (the Abbreviated Mental Test Score (AMTS)) were monitored. At follow-up, subjects were classified as organic or non-organic by three potential screening tools – a screening questionnaire (the Psychogeriatric Assessment Schedule), a psychometric test (the Inglis' Paired Associate Learning Test) and a dementia scale (the AMTS). The value of these as screening tools for community samples was considered as a function of their sensitivity to cognitive decline. The classifications made by each were significantly related to previous cognitive change, but all were conservative, missing many subjects who had declined. Of the three, the AMTS appeared the most useful as a predictor of previous change on the AMTS. It remains to be seen whether it is equally useful with different samples and with different measures of outcome.

There is a pressing need for an economical and acceptable screening tool to identify elderly living in the community who have senile dementia (Cooper & Bickel, 1984; Henderson & Huppert, 1984). Kay *et al* (1964) established that most demented elderly live outside hospital or residential care, and the alarmingly high prevalence of this disorder among the community elderly has been confirmed by most studies (Kay & Bergmann, 1980; Henderson & Kay, 1984). Unfortunately these cases may be poorly recognised (Williamson *et al* 1964; Parsons, 1965; Williams *et al*, 1972) and often relatives/friends bear the brunt of care with inadequate support from the services (Gilhooly, 1984). Bergmann (1979) suggests that more timely identification and support might enable the community to accommodate the demented person for longer and thus reduce demands on over-stretched services.

Recognition of senile dementia at an early stage is a difficult undertaking. The demented patient shows changes in cognition, affect and behaviour which are hard to differentiate from those observed in normal ageing or other age-related conditions. There appears no obvious boundary between normal and abnormal ageing. Kral (1962) proposed that not all cognitively impaired elderly show 'malignant' (i.e. progressive) change, although his concept of 'benign senescent forgetfulness' has yet to be validated. Furthermore, physical and affective illness and non-progressive organic brain disease can produce changes in the elderly similar to those observed in dementia although very different in outcome. Cases of dementia may be best distinguished by the rate of changes shown (Whitehead, 1976). Thus, within clinical samples, the diagnostic sensitivity of psychometric tests is improved with repeated assessment over brief time intervals (e.g. Walton, 1958).

Serial assessment is an expensive undertaking, so for community screening, cases will have to be identified on the basis of current levels of function, from which previous deterioration is presumed. Most community studies have relied upon clinical interviews (structured or unstructured) to identify cases. This is expensive and may be of dubious validity. Recent community studies following up clinically diagnosed cases suggest that many show outcomes inconsistent with the original diagnosis (Gurland *et al*, 1982, 1984). This is particularly true for early cases (Bergmann *et al*, 1971). Studies have reported problems in applying the diagnostic criteria developed to establish differential diagnosis within clinical samples to case-identification in the community (Cooper & Schwarz, 1982; Gurland *et al*, 1982).

Alternative, less expensive methods of case-identification have been developed and used. These can be employed by non-medical personnel to identify possible cases which require clinical confirmation. These include structured screening interviews assessing several areas of function (e.g. Gurland *et al*, 1977–1978), dementia scales briefly quantifying mental status (e.g. Pattie, 1981) and informant ratings of self-care impairment and behavioural disturbance (e.g. Pattie, 1981). Other studies have employed psychometric tests developed to measure specific cognitive functions in clinical samples (e.g. Williamson *et al*, 1964).

There have been few attempts to establish or compare the sensitivities of these screening methods (cf. Milne *et al*, 1972). Most tools have been validated against clinical diagnosis only, often in situations where direct or indirect contamination could operate (Shapiro *et al*, 1956). This is unacceptable, given the problems of establishing diagnosis in community cases. Some have been validated only in clinical samples, despite important differences between community and hospital cases (Bergmann *et al*, 1979). The ultimate test of these tools is their ability to identify as cases those elderly showing the type and rate of changes characteristic of dementia.

The present study was undertaken to compare the sensitivity of three possible screening tools – a semi-structured screening interview (the Psychogeriatric Assessment Schedule (PAS) (Bergmann *et al*, 1975)), a dementia scale (the Abbreviated Mental Test Score (AMTS) (Hodkinson, 1972)) and a psychometric test of verbal learning and memory impairment (the Inglis' Paired Associate Learning Test (IPALT) (Inglis, 1958, 1959)). The PAS was developed for community screening. The AMTS was developed for screening geriatric in-patient samples but has been used with samples in residential care (e.g. Brocklehurst *et al*, 1978). The IPALT is not a screening tool as such but has been used to quantify cognitive impairment in community studies (e.g. Williamson *et al*, 1964). (No informant-assessment was included because of the difficulties of finding reliable informants in elderly community samples (Kay, 1977)). The sample collected (elderly subjects living at home, identified from their general practitioners' lists) is representative of community populations that researchers or clinicians might wish to screen. The sensitivity and specificity of these tools were examined with regard to the cognitive changes shown over the preceding 2 years by the subjects. Sensitivity was defined as the proportion of subjects showing marked cognitive decline correctly identified as probably organic, specificity as the proportion of those showing no decline (or slight decline) correctly identified as probably non-organic.

Method

Tests used to classify subjects

Three different classifications of organic/non-organic were made: the PAS, the AMTS and the IPALT.

Psychogeriatric Assessment Schedule

The PAS is a semi-structured interview developed for use by non-medically qualified personnel to screen elderly samples. The interview used is a modification of an earlier schedule (Bergmann *et al*, 1975) and further details can be obtained from Dr Bergmann. Subjects are classified as probably normal, organic or functional. The organic classification is made in two stages. Firstly, subjects who rate their ability to care for themselves as poor and/or show impaired comprehension during the interview are given nine questions assessing memory and orientation, and rated from direct observation by the interviewer on three scales measuring behaviour indicative of organic impairment (impaired comprehension, puzzled appearance and lack of expression (Bergmann & Eastham, 1974)). Secondly, subjects who score below a validated cut-off point on these questions and ratings are classified as organic. Subjects classified as normal or functional were considered as non-organic for this study. This interview takes (on average) 15 minutes to complete.

Abbreviated Mental Test Score

The AMTS is a ten-item questionnaire assessing memory for recent and remote events, and orientation, developed by Hodkinson (1972) from a longer questionnaire based on that of Blessed *et al* (1968). Scores on the longer test are related concurrently to clinical diagnosis and prospectively to neuropathological and neurochemical indices of dementia (Blessed *et al*, 1968; Perry *et al*, 1978). Scores on the AMTS agree well with those on the longer questionnaire, and a validated cut-off score has been established by Hodkinson (1972) to classify subjects as organic/non-organic. Three items of the AMTS are identical to those included in the PAS. This test takes (on average) 5 minutes to complete.

Inglis' Paired Associate Learning Test

The IPALT provides a sensitive measure of verbal learning ability which is significantly related to clinical diagnosis in elderly psychiatric and geriatric patients (e.g. Inglis, 1958; Isaacs, 1962), and in clinical samples to outcome over periods of up to 5 years (e.g. Whitehead & Hunt, 1982). Subjects were required to learn the associations between three pairs of words (cup–plate; cat–milk; gold–lead) taken from the mediate pairs of Inglis (1959). Up to ten trials were given, and correct responses scored from 0 to 30 according to Whitehead (1971). There is no validated cut-off score for this test, so one was established from the present sample by taking all subjects with complete data (PAS, AMTS, IPALT) at follow-up ($n = 181$) and estimating the base rate of organic impairment from their PAS and AMTS scores. These tests identified 18% and 14% respectively of the sample as organic. The IPALT score giving the most similar proportion of organics was selected as the cut-off point. A score of 23 or less gave 18% of the sample as organic. This cut-off point was cross-validated by consideration of a second sample ($n = 147$) living in residential care. Using the same score, 28% of subjects were classified as organic, which agrees well with the 31% and 27% given as organic by the PAS and AMTS.

The IPALT takes (on average) 10 minutes to administer.

Assessments

All measures were gathered in the same interview (lasting around 30 minutes in most cases). Tests were always given in the order PAS, AMTS and IPALT. The three different interviewers (two State Registered Nurses and a clinical psychologist) were trained together and achieved perfect agreement at the end of training. No further assessment of inter-rater reliability was made.

Sample

This sample was gathered as part of a larger study examining the incidence and prevalence of cognitive impairment in the elderly living at home, for which all patients aged over 65 registered with a GP practice in South London were approached. There were 893 patients located, of whom 81 (9%) refused to be interviewed and 8 (1%) were too physically ill. The remaining 804 subjects were given the PAS and the AMTS and 746 (93%) finished the interview. A random sample of those classified as normal at interview by the PAS and all those classified as organic or functional were followed up 2 years later. The non-random follow-up was undertaken to ensure sufficient numbers of cases for statistical comparison. At follow-up 21 could not be traced and of the remaining 283, 59 (21%) were dead, 37 (13%) refused to be re-interviewed at all and 6 (2%) refused the AMTS or IPALT. This left 181 surviving subjects with complete data at follow-up.

At baseline these subjects were aged (± one standard deviation) 74.45 (± 6.34), and 69% were female, 31% male. Most were living with others (38% with spouses, 17% with family and 2% with friends). Of those living on their own the majority (74%) were in regular contact with friends or family. They scored (± one standard deviation) 7.76 (± 1.37) on the AMTS. Scores on this test ranged from 3 to 10.

Validation criteria

The classifications made by the three tests at follow-up were validated against the cognitive decline shown over the preceding 2 years. Cognitive decline was established from the change in performance on the AMTS from baseline. Since some cognitive decline might result from non-pathological age-related changes or from chance fluctuation in performance (attributable to imperfect reliability of the measure), an arbitrary cut-off was selected to classify subjects on the basis of change scores. A decline in score of one-fifth or more of the range of the test (scored 0 to 10) was taken as indicating a significant decline in performance. Thus those subjects whose scores had declined by 2 or more points during follow-up were classified as showing decline. All subjects scored over 2 at baseline, so no subject was unable to show decline because of floor effect. A check was made of this arbitrary cut-off by estimating the standard error of measurement of the AMTS using the equation of Payne and Jones (1957); that is, standard error of measurement = standard deviation $\times \sqrt{(1-r)}$, where r is a reliability coefficient of the test. The test–retest reliability coefficient over a 2-week period of 0.92 reported by Quershi & Hodkinson (1974) was substituted in this equation, together with the standard deviation of baseline scores from the present sample. This gave a standard error of measurement of ± 0.38. The cut-off change score selected (−2) falls outside the 99% confidence limits for scores on the test and so it most unlikely to occur by chance (presuming a normal distribution of change scores).

Results

At follow-up, the 181 subjects with complete data scored (± one standard deviation) 7.34 (± 2.27) on the AMTS and 25.07 (± 9.11) on the IPALT. Thirty-two (18%) were classified as organic by the PAS and 149 (82%) as non-organic. The intercorrelations between the three tests used to classify subjects are given in Table I. All correlations were significant, indicating that the three measures share variance.

TABLE I

Intercorrelations (Pearson-R) between the three tests used to classify subjects at follow-up

	Coefficient r^1		
	Entire sample (n = 181)	*Subjects showing no decline on the AMTS (n = 151)*	*Subjects showing decline on the AMTS (n = 28)*
PAS × AMTS	0.66	0.40	0.84
PAS × IPALT	0.54	0.35	0.54
AMTS × IPALT	0.77	0.65	0.70

1. All coefficients significant at $P \leqslant 0.001$.

Only 179 of these subjects had AMTS scores at baseline. The average change shown on the AMTS was (± one standard deviation) a decline of 0.39 (± 1.47); the change ranged from a decline of 6 points to an improvement of 2 points during follow-up. Comparing baseline and follow-up performance on this test, overall the sample showed a significant decline ($T = 3.51$; d.f. 178; $P \leqslant 0.001$). Twenty-eight subjects (16%) showed a decline of 2 or more points, the remaining 151 (84%) showed little change or an improvement in performance.

Classification of subjects as organic non-organic

Similar proportions of subjects were classified as organic by each of the three tests. Despite this, rather different subjects were identified as cases by each (Fig. 1). (Because of the small numbers in some cells the Venn diagram shown in Fig. 1 has not been drawn proportionately.) The three tests agreed in 82% of cases (all three classifying 73% of subjects as non-organic and 9% as organic). In the remaining 18% of cases the classifications made by one test disagreed with those made by the other two.

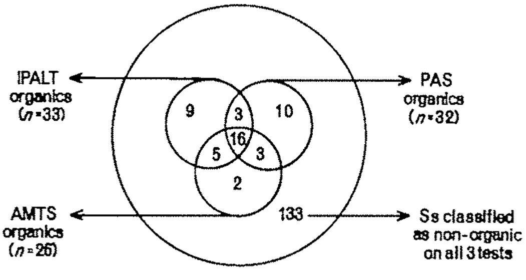

FIG. 1 Overlap of classifications made by the three tests at follow-up (Ss = subjects).

Sensitivity of each of the three tools to cognitive decline

When subjects classified as organic and non-organic at follow-up were compared, substantially more subjects in the organic group had declined over the previous 2 years (Table II). However, whereas only 20% of subjects classified as organic on the AMTS had not declined, about half of the PAS and IPALT organics showed no previous decline.

TABLE II
Relationship between classifications and cognitive change over the preceding 2 years

	Percentage showing decline on AMTS	*χ^2 (d.f. 1)*[1]
PAS organics	48%	27.54*
PAS non-organics	8%	
AMTS organics	80%	85.63*
AMTS non-organics	5%	
IPALT organics	53%	38.10*
IPALT non-organics	8%	

1. χ^2 (corrected for continuity) comparing the outcomes of organics and non-organics.
*$P \leq 0.001$.

The sensitivity and specificity of test classifications were examined with regard to the cognitive changes shown since baseline (Table III). Although specific (correctly identifying 89–97% of those who had not declined as non-organic), the tests missed many subjects who had declined (correctly identifying 54–71% as organic). The AMTS is the most sensitive and specific of the three. When the overall misclassification rates were considered (Goldberg, 1972), only 7% of the sample showed previous cognitive change inconsistent with their AMTS classifications. This figure rises to 15% and 17% for the IPALT and PAS respectively. The hits positive rate (Meehl & Rosen, 1955) represents the probability of previous cognitive decline for subjects classified as organic, taking into account the prevalence of decline in the sample. This is considerably higher for the AMTS (P = 0.82) than for either of the other two tests (P = c.0.50).

TABLE III
Sensitivity and specificity of classifications with regard to cognitive change over the preceding 2 years

	AMTS changes			
Test	*Sensitivity*[1]	*Specificity*[2]	*Overall mis-classification rate*[3]	*Hits positive rate*[4]
PAS	0.54	0.89	0.17	0.48
AMTS	0.71	0.97	0.07	0.82
IPALT	0.61	0.90	0.15	0.54

1. Proportion of subjects showing decline classified as organic.
2. Proportion of subjects showing no decline classified as non-organic.
3. Proportion of 'false positives' (i.e. subjects showing no decline classified as organic) plus proportion of 'false negatives' (i.e. subjects showing decline classified as non-organic).
4. Probability of having declined given a classification of organic.

TABLE IV
Cognitive change over the preceding 2 years shown by subjects classified as organic on all, some or none of the tests

	AMTS	
	Average change (±1 s.d.)	*Percentage showing decline*
Subjects classified as organic on all three tests	−3.73 ± 1.79	80
Subjects classified as organic on some tests	−0.78 ± 1.13	31
Subjects classified as organic on no tests	+0.09 ± 0.86	4
ANOVA[1]	F = 98.57 (d.f. 2, 76)	$P \leq 0.001$
χ^2[2]	χ^2 = 65.31 (d.f. 2)	$P \leq 0.001$

1. ANOVA comparing the average changes shown by the three groups.
2. χ^2 comparing the outcomes of the three groups.

TABLE V
Sensitivity and specificity of the organic classifications in combination to cognitive change over the preceding 2 years

	Sensitivity	*Specificity*
Subjects classified as organic on all three tests	0.43	—
Subjects classified as organic on at least one test	0.79	—
Subjects classified as organic on no tests	—	0.83

Validity of the organic classifications considered in conjunction

Given the considerable disagreements between the classifications made by the different tests (Fig. 1), the classifications made by the three in conjunction were considered. The sample was split into three groups: subjects whom all three tests classified as non-organic, those whom all three classified as organic and those for whom inconsistent classifications were made (i.e. whom only one or two of the tests classified as organic). There were insufficient numbers of subjects classified as organic by only some of the tests ($n = 32$) to break this group down further. The sensitivity and specificity of these combinations of classifications to cognitive decline were examined.

Those classified as organic by all three tests showed substantially more decline on the AMTS than those classified as organic by only some tests. The group classified as non-organic by all three tests showed almost no change during follow-up (Table IV). The group classified as organic by no test accounted for 83% of all subjects who had not declined, whereas those classified as organic by all three tests included only 43% of the subjects who had declined. The sensitivity to previous change was increased to 79% when subjects classified as organic by one or more tests were considered (i.e. when subjects classified as organic by some and by all tests were pooled (Table V)). Considering as cases those classified as organic by one or more tests and as non-cases those classified as non-organic by all three tests gave an overall misclassification rate of 48% and a hits positive rate of 0.47.

Discussion

The value of a screening tool can be considered as a function of its sensitivity (i.e. the proportion of those with a disorder classified as cases) and specificity (i.e. the proportion of those without a disorder classified as non-cases) (Goldberg, 1972). If screening is a two-stage process, with possible cases confirmed by subsequent examination, then sensitivity is of more importance than specificity since it is better to be overinclusive than to miss true cases. In the present study, three tools with established diagnostic validity were examined as possible first-stage screening tools for senile dementia: a screening interview (the PAS), a dementia scale (the AMTS) and a psychometric test (the IPALT). Their value as possible tools for elderly living at home was considered a function of their ability to classify subjects correctly on the basis of the cognitive changes shown on the AMTS over the previous 2 years. A useful first-stage screening tool would be one which identified as possible cases those who had shown a significant decline in cognitive function (i.e. a decline unlikely to have occurred by chance). This would not by itself establish the diagnostic validity of the tool. Some of these possible cases would not be dementing since their decline might be attributable to benign age-related change or to other pathologies (physical or affective illness or non-progressive organic pathology). This can only be established by careful examination, ideally supplemented by follow-up to establish outcome. However, it is unlikely that those with dementia will have shown no decline, unless floor effects were operating (which was not the case here since no subject scored less than 2 at baseline on the measure of change).

As first-stage screening tools all three were quick and easy to administer. They seemed reasonably acceptable, with refusal rates of 9% at baseline and 15% at follow-up. The interviewers all considered the IPALT to be the least acceptable, but since this test was given last this may be confounded with fatigue. Subjects classified as organic by each test were significantly more likely to have declined than were those classified as non-organic (Table II). However, the sensitivity and specificity of the three tools differed (Table III). The overall misclassification rates ranged from 7% (for the AMTS) to 17% (for the PAS). Each was unduly conservative, with acceptable specificity (ranging from 0.89 for the PAS to 0.97 for the AMTS) but poor sensitivity (ranging from 0.54 for the PAS to 0.71 for the AMTS). As first-stage screening tools all might be improved by less conservative cut-offs to classify subjects as organic, given that sensitivity is of more concern than specificity. The AMTS emerges as the most sensitive and specific.

The power of these tools to identify cases has to be considered with regard to the base rate of disorder in the sample. Goldberg (1972) claims that for decisions made on the basis of screening tools to be of use, two conditions should be satisfied: (a) the base rate of disorder < specificity of tool ÷ (specificity + false negative rate); (b) the base rate of non-disorder < sensitivity of tool ÷ (sensitivity + false positive rate).

In the present sample the base rate of 'disorder' (considered here as cognitive decline) was 16%. This is higher than the prevalence of dementia within community samples (Kay & Bergmann, 1980; Henderson & Kay, 1984), but the present follow-up sample was not randomly drawn and was deliberately weighted towards pathology. Furthermore, cognitive decline is not equivalent to dementia. The prevalence of decline was considerably less than the index of test specificity and the false negative rate for each test (which ranged from 0.66 for the PAS to 0.77 for the AMTS). The prevalence of non-decline (84%) was similar to the index of test sensitivity and the false positive rate for the PAS (0.83) and IPALT (0.86) but considerably less than that for the

AMTS (0.96). For each test the probability of subjects classified as organic having declined was substantially higher than the base rate of decline (Table III). Thus some useful decisions can be made on the basis of all three tools, but those made on the basis of the AMTS are clearly the best.

The value of these tests as screening tools should be compared with that of conventional methods of case-identification used within similar samples. Three studies have examined how well clinically identified cases are recognised as dementing by their general practitioners, and report detection rates of 13% (Williamson *et al*, 1964), 38% (Williams *et al*, 1972) and 75% (Parsons, 1965). Even the rather low sensitivities here represent an improvement upon the detection rates reported in the first two studies. Furthermore, the value of clinical interview and diagnosis for detecting community cases has been questioned by Gurland *et al* (1982, 1984) who found that almost half of their 'cases' had outcomes inconsistent with dementia. This casts doubt upon the specificity of clinical case detection; its sensitivity has not been examined.

Given the different content of the three tools it is not surprising that they classified rather different groups of subjects as organic (Fig. 1). Twice as many subjects were classified as organic by some tests (18%) than were classified as organic by all (9%). In spite of the fact that the PAS and AMTS classified different individuals as organic, they gave similar overall prevalence rates (18% and 14% respectively). (The prevalence rate from the IPALT was fixed from those of the other tests.) This suggests caution when examining epidemiological surveys which use different tools to identify cases. It is incorrect to presume that because studies report similar prevalence rates they are considering equivalent groups. Although the tests identified different groups as organic, there is little value in considering the tests in combination (Table V). The sensitivity to decline of combinations of tests was slightly higher than that of the most sensitive individual test (the AMTS), but this gain was at the expense of reduced specificity, and the overall misclassification and hits positive rates were less satisfactory than those of the AMTS. This is surprising, given the improved diagnostic sensitivity gained by combining tests of different functions (e.g. Irving *et al*, 1970; Haglund & Schuckitt, 1976).

From the present results, although all three tests appear economical, acceptable and valid, the AMTS is clearly the most useful screening tool. There is little to choose between the PAS and the IPALT, although the former may be more acceptable. Several studies report that, as diagnostic discriminators, brief dementia scales are as good as (or better than) longer scales or detailed psychometric tests (Hodkinson, 1972; Haglund & Schuckitt, 1976; Kuriansky *et al*, 1976). This may be true for prediction of cognitive change as well. However, it must be noted that all three tests were validated against change on the AMTS. Given that generally reliability coefficients for individual tests are higher than the correlations between different tests, follow-up scores on a test may agree better with changes shown on the same test than do follow-up scores on different tests. A decline of 2 points on the AMTS (the validation criteria) would necessarily bring subjects closer to the organic cut-off score on this test. Possibly with different measures of change the AMTS may be less useful. Furthermore, sensitivity to previous decline is only one of the criteria which screening tools must satisfy. A more stringent test of the tool may be its ability to predict future change. The present sample is one of several samples being followed up to examine the ability of the three tools to predict several measures of outcome.

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References

Bergmann, K. (1979) How to keep the family supportive. *Geriatric Medicine*, **9**, 53–57.

——, Kay, D. W. K. & Foster, E. M. (1971) A follow-up study of randomly selected community residents to assess the effects of chronic brain syndrome and cerebrovascular disease. *Psychiatry* Part II (International Congress Series No. 274). Amsterdam: Excerpta Medica.

—— & Eastham, E. H. (1974) Psychogeriatric ascertainment and assessment for treatment in acute medical ward setting. *Age & Ageing*, **3**, 174–188.

——, Gaber, L. B. & Foster, E. M. (1975) Development of an instrument for early ascertainment of psychiatric disorder in elderly community residents: a pilot study. In *Gerontopsychiatrie*, 4, *Janssen Symposen* (ed. R. von Degkwitz, H. Radebold & P. W. Schulte). Dusseldorf: Janssen.

——, Proctor, S. & Prudham, D. (1979) Symptom profiles in hospital and community resident elderly persons with dementia. In Bayer Symposium VI: *Brain Function in Old Age*. Springer-Verlag.

Blessed, G. B., Tomlinson, B. E. & Roth, M. (1968) The association between quantitative measures of dementia and of degenerative changes in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry*, **114**, 797–811.

Brocklehurst, J. C., Carty, M. H., Leeming, J. T. & Robinson, J. M. (1978) Medical screening of old people accepted for residential care. *Lancet*, **ii**, 141–143.

Cooper, B. & Bickel, H. (1984) Psychological screening and the detection of dementing disorders in old age: a review. *Psychological Medicine*, **14**, 81–95.

—— & Schwarz, R. (1982) Psychiatric case-identification in an elderly urban population. *Social Psychiatry*, 17, 43–52.

Gilhooly, M. L. M. (1984) The social dimensions of senile dementia. In *Psychological Approaches to the Care of the Elderly* (eds I. Hanley & J. Hodge). New York: Croom Helm.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire* (Maudsley Monographs 21). London: Oxford University Press.

Gurland, B., Kuriansky, J., Sharpe, L., Simon, R., Stiller, F. & Birkett, P. (1977–1978). The Comprehensive Assessment and Referral Evaluation (CARE) – rationale, development and reliability. *International Journal of Ageing & Human Development*, 8, 8–42.

——, Dean, L. L., Copeland, J., Gurland, R. & Golden, R. (1982) Criteria for the diagnosis of dementia in the community elderly. *Gerontologist*, 22, 180–186.

——, Golden, R. R., Teresi, J. A. & Challop, J. (1984) The SHORT-CARE: an efficient instrument for the assessment of depression, dementia and disability. *Journal of Gerontology*, 39, 166–169.

Haglund, R. M. J. & Schuckitt, M. A. (1976) A clinical comparison of tests of organicity in elderly patients. *Journal of Gerontology*, 31, 654–659.

Henderson, A. S. & Huppert, F. A. (1984) The problem of mild dementia. *Psychological Medicine*, 14, 5–11.

—— & Kay, D. W. K. (1984) The epidemiology of mental disorders in the aged. In *Handbook of Studies on Psychiatry and Old Age* (eds D. Kay & G. Burrows). Amsterdam: Elsevier.

Hodkinson, H. M. (1972) Evaluation of a mental test score for assessment of mental impairment in the elderly. *Age & Ageing*, 1, 233–238.

Inglis, J. (1958) *Relationships Between Learning and Other Cognitive Functions: an Investigation with Especial Reference to 'Memory Disorders' in Elderly Psychiatric Patients*. PhD thesis, University of London.

—— (1959) Learning, retention and conceptual usage in elderly patients with memory disorder. *Journal of Mental Science*, 105, 210–215.

Irving, G., Robinson, R. A. & McAdam, W. (1970) The validity of some cognitive tests in the diagnosis of dementia. *British Journal of Psychiatry*, 117, 149–156.

Isaacs, B. (1962) A preliminary evaluation of a paired-associate verbal learning test in geriatric practice. *Gerontologica Clinica*, 4, 43–55.

Kay, D. W. K. (1977) The epidemiology and identification of brain deficit in the elderly. In *Cognition and Emotional Disturbance in the Elderly* (eds C. Eisdorfer & R. O. Friedel). Chicago: Year Book Medical Publishers.

——, Beamish, P. & Roth, M. (1964) Old age mental disorders in Newcastle-upon-Tyne – Part I: A study of prevalence. *British Journal of Psychiatry*, 110, 146–158.

—— & Bergmann, K. (1980) The epidemiology of mental disorders in the community. In *Handbook of Mental Health and Ageing* (eds J. Birren & R. Sloane). New Jersey: Prentice Hall.

Kral, V. A. (1962) Senescent forgetfulness: benign and malignant. *Canadian Medical Association Journal*, 86, 257–260.

Kuriansky, J., Gurland, B. & Cowan, D. (1976) The usefulness of a psychological test battery. *International Journal of Ageing & Human Development*, 7, 331–342.

Meehl, H. & Rosen, A. (1955) Antecedent probability and the efficiency of psychometric signs, patterns or cutting score. *Psychological Bulletin*, 52, 194–216.

Milne, J. S., Maule, M. M., Cormack, S. & Williamson, J. (1972) The design and testing of a questionnaire and examination to assess physical and mental health in older people using a staff nurse as the observer. *Journal of Chronic Diseases*, 25, 385–405.

Pattie, A. (1981) A survey version of the Clifton Assessment Procedures for the Elderly. *British Journal of Clinical Psychology*, 20, 173–178.

Payne, R. W. & Jones, H. G. (1957) Statistics for the investigation of individual cases. *Journal of Clinical Psychology*, 13, 115–121.

Perry, E. K., Tomlinson, B. E., Blessed, G., Bergmann, K., Gibson, P. H. & Perry, R. H. (1978) Correlation of chalinergic abnormalities with senile plaques and mental scores in senile dementia. *British Medical Journal*, 11, 1457–1459.

Quershi, K. N. & Hodkinson, H. M. (1974) Evaluation of a ten-question mental test in the institutionalised elderly. *Age & Ageing*, 3, 152–157.

Shapiro, M. B., Post, F., Lofving, B. & Inglis, J. (1956) 'Memory function' in psychiatric patients over 60: some methodological and diagnostic implications. *Journal of Mental Science*, 102, 233–246.

Walton, D. (1958) The diagnostic and predictive accuracy of the Wechsler Memory Scale in psychiatric patients over 65. *Journal of Mental Science*, 104, 1111–1118.

Whitehead, A. (1971) *An Investigation of Learning in Elderly Psychatric Patients*. PhD thesis: University of London.

—— (1976) The prediction of outcome in elderly psychiatric patients. *Psychological Medicine*, 6, 469–479.

—— & Hunt, A. (1982) Elderly psychiatric patients: a five-year prospective study. *Psychological Medicine*, 12, 149–157.

Williams, E. I., Bennett, F., Nixon, J. V., Nicholson, M. R. & Gabert, J. (1972) Sociomedical study of patients over 65 in general practice. *British Medical Journal*, 2, 445–448.

Williamson, J., Stokoe, I. H., Gray, S., Fisher, M., Smith, A., McGhee, E. & Stephenson, E. (1964) Old people at home: their unreported needs. *Lancet*, i, 1117–1120.

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Range of Mental Illness Among the Elderly in the Community

Prevalence in Liverpool Using the GMS-AGECAT Package

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A sample of 1070 elderly persons aged over 65 living in the Liverpool community was interviewed using the community version of the Geriatric Mental State (GMS) and the findings processed to provide a computerised diagnosis by AGECAT. Levels of organic disorder, probably dementia, reached 5.2%, intermediate between those of London and New York derived from previous studies. Levels for depressive illness overall were below those of other studies at 11.3% while levels for neurotic disorder were much the same at 2.4%. The rise in the prevalence of dementia with age was further confirmed. The GMS AGECAT Package provides a method for standardising both the collection of data and the diagnostic process for comparative epidemiological studies and other research.

Reviews by Kay & Bergmann (1980) and Henderson & Kay (1984) of the prevalence of mental illness in community studies of the elderly show that figures for mild and severe mental illness vary considerably between such studies. The use of diverse techniques has made it difficult to interpret the cause of these differences. The importance of standardised methods for collecting data has been stressed and such instruments are now in use. However, to improve accuracy further it is necessary to standardise two more stages, the decision on diagnosis, and the definition of case levels of illness, particularly of minor illness, in a reliable manner.

In the US/UK Cross-National (Diagnostic) Study of the elderly in the cities of New York and London, Gurland *et al* (1983) used the CARE interview developed from the Geriatric Mental State (GMS) (Copeland *et al*, 1976; Gurland *et al*, 1976). They also developed a method for identifying levels of 'pervasive dementia' and 'pervasive depression'. The Diagnostic and Statistical Manual, (DSM-III) of the American Psychiatric Association (1980) also sets forward criteria for the diagnosis of both dementia and depression, and the Research Diagnostic Criteria (Spitzer *et al*, 1978) for depression. Although representing advances in technique, these methods still allow too much individual interpretation of the criteria, (Jorm & Henderson, 1985; Copeland & Gurland, 1985). We consider it necessary for psychiatric epidemiology, to standardise the diagnostic and case-selection processes by using as reliable a method as possible if the comparative studies of populations are to be interpretable. Copeland *et al* (1987*a*), recently re-examined the New York/London community study of the elderly and applied the computerised diagnostic system AGECAT to the data. In two large studies (Copeland *et al*, 1986; Copeland *et al*, 1987*b* AGECAT diagnosis has been shown to approximate reasonably closely to the diagnosis of psychiatrists trained in the British tradition. A computer method of diagnosis has the disadvantage that it must await input of the data to the computer, but it has the advantage of consistency, can take into consideration a wider range of unusual combinations of symptoms, can reliably categorise subcase as well as case levels, and can provide symptom profiles and scale scores.

The study reported here was undertaken as part of a pilot longitudinal study aimed at examining the early detection of dementia in the community elderly. The sample therefore excludes those elderly resident in institutions.

Method

Interviewing techniques: GMS and AGECAT

The development of the GMS has been described in detail elsewhere (Copeland *et al*, 1976; Gurland *et al*, 1976).

GMSA, the shortened version of the GMS for use in the community, was developed specially for this study by selecting those GMS items derived from a series of discriminant function analyses applied to data from the London community sample (Copeland *et al* 1987*a*), aimed at distinguishing between depression and dementia; depression, dementia and other psychiatric diagnoses; all psychiatric diagnoses and non-cases. It covers the important symptom areas for the psychotic and neurotic disorders likely to be encountered in the community and AGECAT can be applied to it. This interview is also a syndrome case finding instrument. A not dissimilar interview (GMS6) was derived independently by Henderson and his colleagues in Canberra by an intuitive selection of items, but unfortunately

AGECAT has only limited application to this version for the identification of subcases.

AGECAT (Copeland *et al*, 1986; Dewey & Copeland, 1986; Copeland *et al*, 1987*b*) consists of a computer program derived from a theoretical model which was subsequently tested against psychiatric diagnosis. It groups the items of the GMS and related interviews into 157 symptom components. These components are gathered under eight diagnostic 'clusters' and allocated to groups according to their importance for establishing the certainty of diagnosis for that diagnostic cluster. For example, in the organic cluster the groups concern 'mild memory disturbance'; 'moderate memory disturbance and time disorientation'; and 'place/person disorientation and organic thought disorder'. While those for depression represent 'depressive mood'; 'non-specific symptoms of depression'; 'symptoms characteristic of neurotic, reactive depression or dysthymic mood'; 'symptoms denoting severity'; 'symptoms characteristic of psychotic, endogenous depression or major affective disorder'; and 'mood congruent hallucinations and delusions'. Each subject is awarded a level from 0–5 for most diagnostic clusters according to the certainty 'of diagnosis' on that cluster. All levels on each cluster are then compared across clusters, one with another, according to a hierarchy of illness starting with organic and proceeding to schizophrenic, manic, depressive (psychosis and neurosis), obsessional, hypochondriacal, phobic and anxiety. The subject emerges with a main diagnosis and a subsidiary or alternative diagnosis if appropriate, the levels on all eight diagnostic clusters, the routes by which these levels are achieved, a symptom profile, a decision on whether or not the condition reaches the 'syndrome case' stage, organic and depressive scale scores and an Organic/Depression Index (Copeland *et al*, 1986*b*, 1987*a*).

A 'syndrome case' is reached when a psychiatrist recognises that a subject's symptoms have formed a recognisable diagnostic pattern. In practice, this usually implies the need for professional intervention. Bereavement reaching syndrome case level on the depressive cluster is an example of an exception. Syndrome cases may therefore be further defined, as requiring or not requiring such intervention. A subject may reach case level on several syndromes. AGECAT will select one of these as the overall diagnosis. We then refer to this as a 'diagnostic syndrome case of ——'. Most studies will require additional case criteria tailored to the aims of the study. We have called cases so selected, 'criterion cases'. In this study we report levels of illness at the 'syndrome case' and 'diagnostic syndrome case' stage.

The agreement between AGECAT and psychiatrists' diagnoses was compared in an initial study (Copeland *et al*, 1986*a*) and in a replication study (Copeland *et al*, 1987*b*). For 396 community-based subjects in the initial study the κ value for overall agreement was 0.74, for cases of depression 0.80, for organic cases 0.88 and for non-cases 0.74. For the replication study, diagnostic concordance was reached on 89% of 647 community subjects, giving a value for Cohen's κ overall of 0.71, for depression 0.73, and for organic disorder 0.77. The last was complicated by the introduction of a third category of 'borderline organic' case. Where the decision was case/non-case, κ reached 0.89 for organic disorders. These studies and others show that psychiatrists trained in different centres appear to agree with AGECAT diagnoses for both institutionally based and community based subjects.

The sample

The 360 General Practitioners forming the Liverpool Family Practitioners' Committee were randomly sampled to provide 58 names. These GPs were approached by a letter explaining the study and requesting permission to interview their patients. Three refused permission at this stage, leaving 55 who were randomly allocated to 11 subsamples of five practitioners each. Each subsample formed a random sample in its own right. Failure to complete one subsample would not have invalidated the random nature of the total sample, and separate subsamples were available for individual studies.

The potential pool of subjects was 12 909. In order to provide approximately 1000 subjects, 2425 or 19% of the available pool were selected from cards held by the Family Practitioner Committee. In the event, only nine of these subsamples were required.

Each subject was sent an individually signed letter describing the project, accompanied by a letter of introduction from the GP. The explanatory letter stressed the random selection of the subjects, the nature of the health survey, that it was not 'an official survey' and that there was no obligation to take part. An address and telephone number for contact were also given. Written or verbal refusals were accepted without question, otherwise subjects were visited up to four times if necessary, whether or not they replied to the letter. To a small number of subjects who objected to being approached in this way a personal letter of explanation was sent.

Interviewers

Six interviewers participated in the project, three full-time and three part-time. Two of the full-time interviewers possessed first degrees in psychology and the third was an ex-ward sister (SRN) with geriatric experience. Two of the part-time interviewers had degrees in psychology and one an HNC in medical subjects.

Training on the GMS was undertaken using videotaped and live interviews in the usual manner. A short pre-pilot study was undertaken in a geriatric day hospital to test the confidence of the interviewers when confronted with patients with mental and physical illness. Inter-rater reliability was assessed using 19 videotaped interviews of community residents. Each of the six non-medical interviewers recorded two or three interviews while the three medically qualified raters who participated in the follow-up study (see below) each recorded two. All raters subsequently viewed and scored each other's interviws. The results are to be reported (Forshaw *et al*, in prep.). Satisfactory agreement between trained non-medical and medical raters for the Present State Examination has been demonstrated (Cooper *et al*, 1977) and there is little reason to suppose that this can not be achieved for older age

TABLE I
Liverpool: Comparison of mean organic scores and subject's mean age for each rater

Rater	*Organic mean score*	*s.d.*	*Mean age*	*s.d.*
1	2.6	3.3	73.3	7.1
2	2.4	3.1	75.1	6.9
3	2.4	3.7	72.6	5.9
4	2.4	2.9	73.5	6.8
5	3.1	3.4	74.5	6.1
6	3.1	4.6	73.2	6.2

Organic score $F=0.44$ (NS); Age $F=1.62$ (NS).

subjects. Meanwhile, Table I shows a comparison of subjects' organic mean scores and age for each rater. The organic score was made up of those items which best discriminated between organic disorders and other mental illness, and between organic disorders and non-cases, using a series of linear discriminant function analyses. The rating of organic symptoms was particularly important for the study as a whole. The variation between raters is not great for the overall organic mean score nor for the mean age of subjects seen. Standard deviations are similar and differences are not statistically significant.

Initial test battery

All subjects, at the initial interview lasting 50–90 minutes, received: a first blood pressure reading; a health questionnaire; a social information questionnaire; a number of psychological tests including the SET Test, the National Adult Reading Test, and the Mill Hill Vocabulary Test; the second blood pressure reading; the GMSA; and the third blood pressure reading. The Stockton Behaviour Rating Scale was abandoned early in the study as unsuitable for a community population of comparatively well subjects.

A subsample of dementing subjects had a physical examination, computed tomographic scan, biochemical investigations and an interview by a psychiatrist using the GMSA.

Results

Table II shows the overall response rate. As expected, the GPs lists were inaccurate. Although places of residence were repeatedly visited where necessary and attempts made to gather additional information, a proportion of subjects was not accounted for. One GP refused access to his patients after the interviewing started. In all, 1070 or 72% of the eligible sample were interviewed. Although the procedure in Liverpool was similar to that used in our London studies the response rate was 9% lower, only a little above that found in New York. We discuss this further below.

Table III shows the distribution of the sample by age divided into half decades. The proportions of male and female subjects are not significantly different from either those found in the 1981 census for Liverpool city or in the study in London. The mean age of 74 years is similar to that of both the London and New York samples. The low proportion of male subjects aged between 80 and 84 is probably a result of the First World War. Both subjects who refused interview and those who were not traced have a similar age distribution to the full sample.

TABLE II
Liverpool: Random sample of subjects aged 65 and over living in the community of Liverpool city: response rate

Total estimated subjects available aged 65 and over: 12 909		
Selected from GPs' list	2425	(19%)
Discovered already dead	114	
Discovered already moved away	270	
Subsamples drawn but not used, not traced or GP refused	555	
Sample eligible for study	1486	
Refused interview	416	(28%)
Sample interviewed	1070	

Response rate 72%

Table IV shows some of the other demographic features of the sample. They differ little from the census statistics where these are available, and apart from 'race', are similar to those found in the previous London and New York studies.

Prevalence

Table V shows the male and female distribution of the major psychiatric syndromes. AGECAT diagnostic confidence levels of 3 or above are used to define 'syndrome cases'. It was our experience based on the London and New York studies that acute organic states are rarely diagnosed in community samples. Therefore, subjects with AGECAT diagnostic organic disorders quoted here are likely to be suffering from dementia (overall 5.2%).

The total proportion of cases of all diagnoses is 19.3%. The proportions of subjects with no AGECAT level on any of the syndrome clusters is 47% for males and 37% for females. Thus over one-third of the population had no important psychiatric symptoms.

Organic disorders

Table VI shows the distribution of AGECAT diagnostic syndrome cases and subcases of organic disorders according to levels of confidence of diagnosis. AGECAT organic levels which are associated with diagnostic cases of other conditions are not included. There are some 14 'routes' by which subjects may be allocated to organic confidence levels. In general, to achieve level 01 there must be a score of at least 2 on symptoms of mild memory disturbance, such as failing to remember the interviewer's name, miscalculating the length of time at the present address, the rater's opinion that the memory defect is a problem for the subject, etc, while for level 02 a higher proportion of such symptoms is required or others of moderate memory disturbance, inability to calculate age, failing to recall the

TABLE III
Liverpool: Distribution of the sample by age and sex (males n = 410, females n = 660)

Age	*Males* *n*	*Males* %	*Females* *n*	*Females* %	*Full sample* *n*	*Full sample* %	*1981 census Liverpool* %	*Not traced etc* %	*Subject refused interview* %
65–69	151	44	192	56	343	32	34	33	30
70–74	115	37	194	63	309	29	29	27	26
75–79	86	38	142	62	228	21	20	19	22
80–84	32	28	84	72	116	11	11	12	13
85–89	20	36	36	64	56	5 }7	}7	6 }9	5 }9
90+	6	33	12	67	18	2		3	4
	410	38	660	62	1070	100	100	100	100

Mean age 74

TABLE IV
Liverpool: Social class, marital state and race

Social class Adapted from Registrar General's classification	*n*	%		*n*	%
I	15	1.4	White collar	301	28.1
II	136	12.7	Blue collar	384	35.9
III	521	48.7	Unskilled/service	224	20.9
IV	143	13.4	Houseperson (personal service)	92	8.6
V	123	11.5	Missing	69	6.5
Houseperson or inadequately described	63	5.9			
Missing	69	6.5			

Marital state	*n*	%		*1981 census Liverpool* %	*Race*	*n*	%
Currently married	474	44.3		44.0	Caucasian	1064	99.4
Widowed	434	40.6			Mongolian	2	0.2
Divorced/ separated	32	3.0	55.7	56.0	Negroid	2	0.2
Never married	125	11.7			Unknown	2	0.2
Unknown	5	0.5					

interviewer's name on the second occasion or misidentifying objects. Case level 03 is reached when a substantial number of such symptoms is recorded often accompanied by time disorientation. Higher levels, 04 and 05 (the community version of the GMS did not provide for these levels at that time) require the presence of severe disorientation in place and person as well as organic thought disorder.

A higher proportion of female subjects is evident at the case level but not at the subcase level ($P < 0.001$). If, as anticipated, subcase levels 01 and 02 mainly represent minor acute confusional states there is no reason to suppose that these would be more common in female patients. They would also be expected to include early cases of dementia but these would be comparatively rare. Subjects achieving any kind of AGECAT organic level represent 9.3% of male and 10.6% of female subjects. The difference is not statistically significant.

Figure 1 shows the distribution of AGECAT 'diagnostic syndrome cases' (03 and above) of organic disorder by age and sex. There is a linear relationship between age and the logit of the proportion of demented subjects ($P < 0.001$). The fall after age 90 is a less certain finding because of the

TABLE V
Liverpool: Comparison by sex using AGECAT diagnostic syndrome cases (males n = 410, females n = 660)

	Males		*Females*		*Total sample*	
	n	*%*	*n*	*%*	*n*	*%*
AGECAT diagnostic syndrome case						
Organic	11	2.7	45	6.8	56	5.2
Depression	31	7.6	90	13.6	121	11.3
Schizophrenia/ paranoid	1	0.2			1	0.1
Neuroses	3	0.7	23	3.5	26	2.4
Other	1	0.2	1	0.2	2	0.2
Total cases	47	11.5	159	24.1	206	19.3
Total sample	410	100	660	100	1070	100

TABLE VI
Liverpool: Distribution of AGECAT diagnostic organic syndromes (case and subcase) by levels of confidence of diagnosis (males n = 38, 9.3%, females n = 70, 10.6%)

	AGECAT organic confidence levels						
	Subcase				*Case*		
	01		*02*		*03*		*Total sample*
	n	*%*	*n*	*%*	*n*	*%*	*n*
Males	16	3.9	11	2.7	11	2.7	410
Females	11	1.6	14	2.1	45	6.8	660
Combined	27	2.5	25	2.3	56	5.2	1070

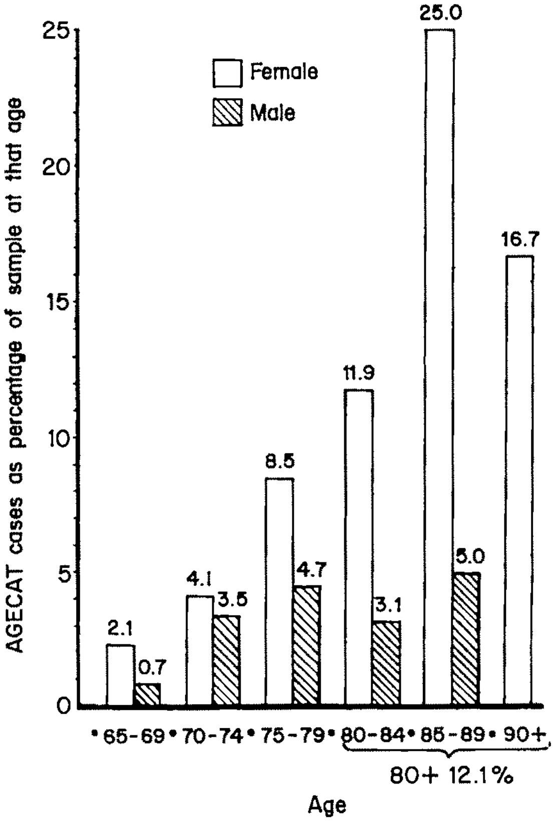

FIG. 1 Distribution of AGECAT diagnostic syndrome cases of organic disorder (mainly dementia) by age and sex. (Full sample: males $n = 410$, females $n = 660$.)

few subjects in this age group. The proportion aged over 80 with organic disorder is only 12.1%, rather below the often quoted figure of 20% (this sample does not include the comparatively small proportion of institutionalised subjects).

Depressive illness

Table VII shows the distribution of AGECAT diagnostic syndrome cases and subcases of depressive disorders by levels of diagnostic confidence. The table excludes those levels of depression associated with diagnostic cases of other conditions. There are over 60 'routes' for achieving levels of confidence on the depressive cluster. As a rough guide, level D1 is achieved by subjects with minor mood symptoms and some symptoms of non-specific depression, such as loss of concentration, interest, lack of enjoyment, lack of energy, etc., D2 requires a higher proportion of such symptoms. Case levels of depressive neurosis (DN3) must have characteristic symptoms in addition, such as, depression worse in the evening, difficulty getting off to sleep, depersonalisation, feelings of loneliness, etc. in the absence of more psychotic symptoms. Level DN4 is achieved if more severe symptoms (group D) are also prominent, such as, the future appearing bleak, wishing to be dead, feeling worthless, having conceived a suicidal plan or attempted to carry it out. Case levels of depressive psychosis (DP) require symptoms such as, depressive mood worse in the morning, being unable to cry, slowness in the morning, early morning wakening, retardation on examination, muddled thinking and a combination of appetite and weight loss. Level 4 of depressive psychosis depends on the prominence of group D symptoms and level 5 on the presence of mood congruent delusions and/or hallucinations.

There is a consistent tendency for there to be a higher proportion of female subjects at each depressive level of diagnostic confidence ($P < 0.05$). In all, 17.3% of males and 24.9% of females are nominated as either diagnostic syndrome depression subcases or cases. For the full sample with the sexes combined, 10.7% of subjects achieve diagnostic syndrome subcase level and 11.3% case level, making a total proportion of 22.0%. The overall prevalence of diagnostic syndrome cases of depressive psychosis is 3.0%.

TABLE VII

Liverpool: Distribution of AGECAT diagnostic depressive syndromes (case and subcase) by levels of confidence of diagnosis (males n = 71, 17.3%, females n = 164, 24.9%)

	AGECAT depressive confidence levels														
	Subcase				*Case*										
					Depressive neurosis				*Depressive psychosis*						
	D1		*D2*		*DN3*		*DN4*		*DP3*		*DP4*		*DP5*		*Total sample*
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*
Males	21	5.1	19	4.6	21	5.1	3	0.7	2	0.5	5	1.2	0	0	410
						5.9%					1.7%				
Females	41	6.2	33	5.0	54	8.2	11	1.7	16	2.4	8	1.2	1	0.2	660
						9.9%					3.6%				
Combined	62	5.8	52	4.9	75	7.0	1.4	1.3	18	1.7	13	1.2	1	0.1	1070
						8.3%					2.9%				

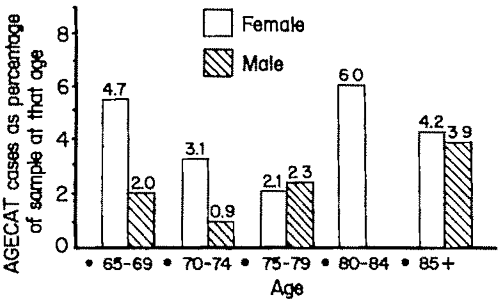

FIG. 2 Distribution of AGECAT diagnostic syndrome cases of depressive psychosis (DP) by age and sex. (Full sample: males n = 410, females n = 660.)

Figure 2 shows the distribution of AGECAT 'diagnostic syndrome cases' of depressive psychosis by age and sex. Subcase levels are excluded from this figure. Again, there appears to be a preponderance of female subjects at all half decades except one.

Figure 3 shows the distribution of 'diagnostic syndrome cases' of depressive neurosis by age and sex. Again, a higher proportion of female patients is evident at each half decade.

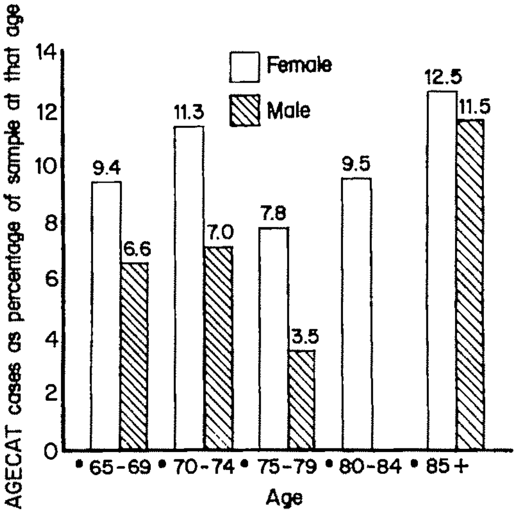

FIG. 3 Distribution of AGECAT diagnostic syndrome cases of depressive neurosis (DN) by age and sex. (Full sample: males n = 410, Females n = 660.)

Neurotic disorders

Table VIII shows the distribution of AGECAT diagnostic syndrome cases and subcases of neurotic disorders by levels of confidence of diagnosis and by sex. The table excludes those neurotic levels associated with diagnostic cases of other conditions. For the three conditions obsessive, phobic and anxiety neurosis there is a tendency for rather higher proportions of subjects to be found at level 2 than at the case levels.

Whether or not a subject achieves syndrome case levels on the hypochondriasis cluster largely depends on whether he or she shows undue preoccupation with physical symptoms for which there is no apparent cause. For subjects on the obsessional cluster to reach syndrome case levels the symptoms must interfere with daily living. Subjects at level OB2 have genuine obsessional symptoms in so far as they have rituals or compulsive thoughts which they attempt to resist. For subjects to achieve syndrome case level on the phobic cluster there must be evidence of active avoidance or in more extreme cases the subject must be housebound. To achieve syndrome case levels on the anxiety cluster subjects must complain of or show evidence of somatic symptoms or symptoms of tension.

TABLE VIII

Liverpool: Distribution of AGECAT diagnostic neurotic syndromes (subcase and case) by levels of confidence of diagnosis (males n = 105, 25.6%, females n = 181, 27.4%)

AGECAT neurotic confidence levels										
	Subcase				*Case*					Total sample
		HC1		*HC2*		*HC3*		*HC4*		
Hypochondriasis	*n*	*%*			*n*	*%*				*n*
Males	1	0.24			2	0.49				410
Females	1	0.15			3	0.46				660
		OB1		*OB2*		*OB3*		*OB4*	*OB5*	
Obsessive			*n*	*%*	*n*	*%*				*n*
Males			10	2.44						410
Females			9	1.36	1	0.15				660
		PH1		*PH2*		*PH3*		*PH4*		
Phobic			*n*	*%*	*n*	*%*				*n*
Males			15	3.66						410
Females			37	5.61	8	1.20				660
		AN1		*AN2*		*AN3*		*AN4*	*AN5*	
Anxiety	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*		*n*
Males	12	2.93	64	15.61			1	0.24		410
Females	9	1.36	102	15.45	10	1.52	1	0.15		660

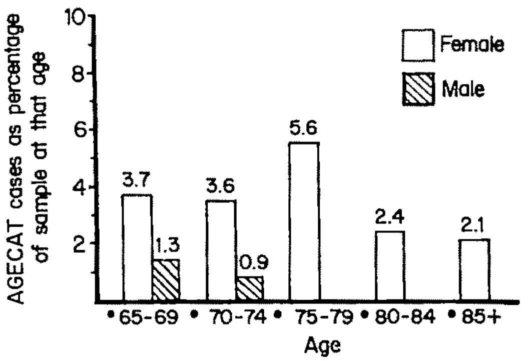

FIG. 4 Distribution of AGECAT diagnostic syndrome cases of neurotic disorders by age and sex. (Full sample: males $n = 410$, females $n = 660$.)

Table VIII shows that although neurotic syndrome 'case' levels as defined here are rare in these age groups, high proportions of neurotic symptoms, particularly anxiety, are often present. On the evidence of previous studies, confidence levels 4 and 5 for anxiety are uncommon, unless associated with diagnostic cases of depression, dementia or schizophrenia. The overall prevalence of diagnostic syndrome cases of neurosis is 2.4% (males 0.3%, females 2.1%). However, if those subjects with diagnostic subcase level 2 are included with the cases on the grounds that this level represents substantial neurotic symptoms (although they may not interfere with daily living), and because case/non-case distinctions are somewhat 'arbitrarily' chosen for neurotic conditions, the overall prevalence rises to 24.6% (males 8.6%, females 16%, NS). If all diagnostic syndrome cases and subcases of neurotic disorders are included, the overall prevalence is 26.7%. Only levels of phobic symptoms are significantly different between the sexes with females having rather more symptoms than males ($P < 0.05$).

Figure 4 shows the distribution of AGECAT diagnostic syndrome cases of neurosis by age and sex. The higher proportion of female cases is evident, ($P < 0.003$). The apparent decline with age is not significant.

Discussion

Response rate

Although the same procedures were used in both Liverpool and London, the response rate was lower in Liverpool. It is similar to the response rate in New York and a little below that achieved by Kay *et al* (1985) when both their younger and older samples of the elderly in Hobart are combined. The reasons why subjects refuse to take part in a study are varied but it is our impression that a substantial proportion of refusals are caused by misinterpretations of the study's aims and a failure to appreciate its importance in terms either of the usefulness of its results or in its financial outlay. Even when attempts are made by letter to explain the importance of interviewing both the well and the ill, this may still not be truly appreciated by the subject. We have found in subsequent studies that a substantial improvement in response rate occurs, if a preliminary visitor calls at the house to explain in person the nature of the project. Such a procedure increases the overall expense of the project but can also save interviewer's time if the preliminary visitor arranges precise appointments.

As there were no significant differences in age distribution between those who were interviewed and those who refused interview, we do not expect to have lost a higher proportion of cases than non-cases, although this always remains a possibility in such studies.

Prevalence

Organic disorders

The level of 5.2% for diagnostic syndrome cases of organic disorder (probably mostly dementia) compares with 4.3% for London and 8.3% for New York. All prevalence figures for the New York and London studies quoted here are those given by Copeland *et al* (1987*a*) derived from the application of AGECAT to the New York and London CARE data. Mental state item differences between the CARE Schedule and the GMSA are small and, in any case, diluted by the pooling of items to form the symptom components. It is unlikely, therefore, that differences in prevalence can be ascribed to differences in the technique used.

The age distribution in the Liverpool sample is similar to that in the London sample. The prevalence of organic disorders in males in Liverpool is similar to that in London, while that in females lies midway between the New York and London figures. The overall proportion of organic syndrome subcases lies between the London and New York figures. The combined proportions of subcases and cases of 10.1% and the proportion for cases of 5.2% are remarkably similar to the findings of the original Newcastle studies (Kay *et al*, 1964) of approximately 10% for both mild and severe organic brain syndromes and 4.9% for severe alone.

Depression

The overall prevalence figure of 11.3% for diagnostic syndrome cases of depressive disorders is somewhat below that for either New York 16.2%, or London 19.4%. It is accounted for by a lower proportion of cases (also subcases) of depressive neurosis, while the overall prevalence for cases of depressive psychosis of 3.0% is slightly above that for either New York or London. The City of Liverpool is noted for its high level of social problems, particularly unemployment, crime and drug dependence. However, it had been anticipated in the London/New York studies, that the prevalence of depression would be higher in New York than in London because of the additional social problems in that city, the presence of which were amply documented by the study. Nevertheless, the proportions of depression found in London and New York were similar. It therefore cannot be assumed that the level of depressive illness or depressive symptoms in a community will bear a direct positive relationship to the level of social disturbance. This may be related to the well-known finding that certain types of psychiatric morbidity are negatively correlated with social disturbances such as war. As for the affective disorders as a whole, if the diagnostic syndrome subcase levels of 10.7% and the case levels of 11.3% are added together to equal 22.0%, this figure is not far short of the 26.2% for mild-to-severe levels of affective disorder given in the Newcastle studies. The Liverpool diagnostic case level of 11.3% is close to the figure of 10.0% for moderate/severe affective disorders, found in Newcastle. The Liverpool figure of 3.0% for psychotic depression is close to the figure of 3.7% found by Blazer & Williams (1980) using DSM-III criteria for major affective disorder in a community sample aged over 65 years.

Neurotic disorders

The overall prevalence for diagnostic syndrome cases of neurosis in Liverpool of 2.4% may be compared with 1.4% in New York and 2.0% in London. There are no important differences in syndrome case levels for any of the individual neurotic disorders between the three cities. If all diagnostic syndrome levels for obsessional disorders are taken together (cases and subcases) the prevalence is 1.7% for the Liverpool sample, close to the 0.7% for the New York sample, but much lower than the figures for London of 9.8%. 2.4% is also remarkably close to 2.6% for 'other' functional diagnoses found by the Newcastle study after the removal of cases of schizophrenia, paranoid illness and affective disorders.

There are few differences between the sexes for the neurotic disorders and there is no consistent preponderance of males across the age-span as was evident in the combined London and New York samples. Neurotic symptoms may fail to reach case level because elderly people may have a lifestyle which allows them to avoid contact with provoking factors.

The measures

The GMS-AGECAT Package offers a method for undertaking comparative studies of elderly populations. It introduces a degree of standardisation not only into the collection and recording of symptoms but also into the selection of diagnosis. A range of information is provided about each subject including the identification of case and subcase levels of diagnostic confidence.

In the study described here the diagnoses were made in terms of eight diagnostic clusters. Historical data were not available at the time. If more exact distinctions are to be made between, for example, dementia and acute confusional states; and between types of dementia, other chronic organic conditions and pre-existing mental subnormality; historical data will be essential. The History and Aetiology Schedule is being used in the follow-up studies now being undertaken. Data from this interview pass into the next stage of AGECAT which aims to divide the eight diagnostic clusters into subgroups. It provides information on the form of onset and development of the illness, family history, previous illnesses, head injury and alcohol and drug abuse.

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References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) 3rd ed. Washington, D.C.: APA.

Blazer, D. & Williams, C. D. (1980) Epidemiology of dysphoria and depression in an elderly population. *American Journal of Psychiatry*, 137, 439–444.

Cooper, J. E., Copeland, J. R. M., Brown, G. W., Harris, T. & Gourlay, A. J. (1977) Further studies on interviewer training and inter-rater reliability of the Present State Examination (PSE) *Psychological Medicine*, 7, 517–523.

Copeland, J. R. M. & Gurland, B. J. (1985) International comparative studies. In *Recent Advances in Psychogeriatrics* ed. T. Arie, Edinburgh: Churchill Livingstone.

——, Kelleher, M. J., Kellett, J. M., Gourlay, A. J., Gurland, B. J., Fleiss, J. L. & Sharpe, L. (1976) A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly. The Geriatric Mental State Schedule. 1. Development and reliability. *Psychological Medicine*, 6, 439–449.

——, Dewey, M. E. & Griffiths-Jones, H. M. (1986*a*) Computerised psychiatric diagnostic system and case nomenclature for elderly subjects: GMS and AGECAT, *Psychological Medicine*, 16, 89–99.

——, McWilliam, C., Dewey, M. E., Forshaw, D. M., Shiwack, R., Abed, R., Muthu, M. S. & Wood, N. (1986*b*) The early recognition of dementia and depression in the elderly: a preliminary communication about a longitudinal study using the G.M.S. AGECAT Package. *International Journal of Geriatric Psychiatry*, 1, 63–70.

——, Gurland, B. J., Dewey, M. E., Kelleher, M. J., Smith, A. M. R. & Davidson, I. A. (1987*a*) Is there more dementia, depression and neurosis in New York? A comparative community study of the elderly in New York and London using the computer diagnosis AGECAT. *British Journal of Psychiatry*, (in press).

——, Dewey, M. E., Henderson, A. S., Kay, D. W. K., Neal, C. D., Harrison, M. A. M., McWilliam, C., Forshaw, D. & Shiwach, R. (1987*b*) The Geriatric Mental State used in the community: replication studies of the computerised diagnosis AGECAT Psychological Medicine (in press).

Dewey, M. E. & Copeland, J. R. M. (1986) Computerised psychiatric diagnosis in the elderly: AGECAT. *Journal of Microcomputer Applications*, 9, 135–140.

Forshaw, D. M. The reliability of the Geriatric Mental State (community version). (in prep.).

Gurland, B. J., Fleiss, J. L., Goldberg, K., Sharpe, L., Copeland, J. R. M., Kelleher, J. M. (1976) A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly. The Geriatric Mental State Schedule 2. A factor analysis. *Psychological Medicine*, 6, 451–459.

——, Copeland, J. R. M., Kelleher, M. J., Kuriansky, J., Sharpe, L. & Dean, L. (1983) *The Mind and Mood of Aging: The mental health problems of the community elderly in New York and London*. New York: Haworth Press; London: Croom Helm.

Henderson, A. S. & Kay, D. W. K. (1984) The epidemiology of mental disorders in the aged. In *Handbook of Studies in Psychiatry and Old Age* (eds. D. W. K. & G. Burrows. Amsterdam: Elsevier.

Jorm, A. F. & Henderson, A. S. (1985) Possible improvements to the diagnostic criteria for dementia in DSM-III. *British Journal of Psychiatry*, 147, 394–399.

Kay, D. W. K. & Bergman, K. (1980) Epidemiology of mental disorder among the aged in the community. In *Handbook of Mental Health and Aging* eds J. E. Birren & B. Sloan. Englewood Cliffs, New Jersey: Prentice Hall.

——, Beamish, R. & Roth, M. (1964) Old age mental disorders in Newcastle upon Tyne. Part I. A study of prevalence. *British Journal of Psychiatry*, 110, 146–158.

——, Henderson, A. S., Scott, R., Wilson, J., Rickwood, D. & Grayson, D. A. (1985) Dementia and depression among the elderly living in the Hobart community: the effect of the diagnostic criteria on the prevalence rates. *Psychological Medicine*, 15, 771–788.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research diagnostic criteria. *Archives of General Psychiatry*, 35, 773–782.

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Detention in Broadmoor
Factors in Length of Stay

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When the length of stay of restricted patients admitted to Broadmoor under the legal category of psychopathic disorder was examined, the factor found to be of primary importance was the gravity of the admission offence. If the men's offences did not cause personal injury, they had a good chance of early release. Patients convicted of violent or sexual offences, and particularly those who attacked strangers, made up the great majority of the long-term group. For the mentally ill there was no relationship between length of stay and admission offence: instead the data suggested that severity and chronicity of illness were the main relevant factors.

The Special Hospital Case Register was set up in 1972 and since the beginning of that year, the Special Hospitals Research Unit have been recording particulars about all patients entering the special hospitals. The information is obtained by interview and from the official records. Data about the patient's progress or treatment in hospital are not collected, but there is information about his psychiatric history, social background, criminal record, and diagnosis on admission. It was possible to use these data for the purpose of seeing whether admissions to Broadmoor had changed over the years since 1972, and also for examining the characteristics of different groups of patients, and seeing how long they were held in the hospital. Information about all the male admissions between 1972 and 1984 were made available to us.

Table I shows Broadmoor's annual figures by legal category of male admissions with a Mental Health Act classification of psychopathic disorder (PD) or mental illness.

It is clear from the table that Broadmoor's intake has fallen substantially over the years, with admissions of PD patients decreasing more than those of the mentally ill. The data were examined to see whether, within each of these Mental Health Act classifications, there was any pattern to the decline, i.e. whether receptions of certain types of patients or offenders decreased more than others. No such patterns emerged, but it is probable that there were trends in the fall of a kind not recorded in the Case Register data. There has, for example, been increasing scepticism about the treatability of psychopaths, and increasing insistence by the DHSS that mentally ill patients, however serious their offences, should not be admitted to special hospitals unless they need maximum security (Dell, 1984).

How long do patients stay in Broadmoor? The case register showed the date of discharges and transfers up to December 1982, so for patients admitted in the early 1970s some picture on the

TABLE I

Male admissions to Broadmoor 1972–1984 by Mental Health Act classification and Legal Category

Mental Illness *Year*	*S.26*	*60*	*65*	*72*[1]	*CPIA*[2]	*Other*	*Total*
1972	10	9	44	14	12	3	92
1973	8	4	46	5	8	4	75
1974	4	4	48	6	5	2	69
1975	5	4	38	6	10	2	65
1976	7	4	43	1	4	2	61
1977	6	3	29	7	5	—	50
1978	5	1	36	7	2	—	51
1979	6	4	35	9	5	2	61
1980	5	2	11	3	4	—	21
1981	3	2	15	9	6	1	36
1982	7	1	31	9	4	1	53
1983[1]	3	0	21	4	1	1	37
1984	3	1	9	4	1	2	20
Psychopathic Disorder							
1972	—	1	21	6	—	—	28
1973	—	2	43	5	—	—	50
1974	—	—	26	2	—	—	28
1975	—	1	11	3	—	—	15
1976	—	—	15	2	—	—	17
1977	—	—	14	—	—	—	14
1978	—	—	20	1	—	—	21
1979	—	—	6	1	—	—	7
1980	—	—	9	1	—	—	10
1981	—	—	9	—	—	—	9
1982	—	—	9	1	—	—	10
1983[1]	—	—	6	2	—	—	8
1984	—	—	3	1	—	—	4

1. Since October 1983, MHA 1983 section numbers apply.
2. Criminal Procedure Insanity Act 1964.

length of stay was ascertainable. We decided therefore to look at patients admitted in the years 1972–1974, for whom at least 8 years had elapsed by the time the release date was collected in 1982.

Psychopathic disorder patients

Table I showed that there were 106 men with a MHA classification of psychopathic disorder admitted in the years 1972–1974. The great majority were sent to Broadmoor by the courts on restricted hospital orders, and we confined our length of stay analysis to this group, since very different considerations are involved in the discharge of other groups, for example, of transferred prisoners who are liable to be returned to prison.

There were 81 PD patients admitted from the courts on restricted hospital orders in the years 1972–1974. For the purpose of looking at length of stay, we excluded two men who died in Broadmoor and another three who were sent to prison after committing further serious offences in Broadmoor. This left us with 76 men, and the length of time they stayed in Broadmoor (or if they moved to one, in another special hospital) is shown in Table II.

TABLE II
Section 60/65 Patients admitted 1972–1974: Length of stay

	Psychopaths (*n* = 76)		*Mentally ill* (*n* = 111)	
Length of stay	*n*	%	*n*	%
2 years or less	6	8	21	19
over 2–3 years	2	3	12	11
over 3–4 years	10	13	13	12
over 4–5 years	7	9	7	6
over 5–6 years	6	8	3	3
over 6–8 years	5	7	8	7
over 8 years	40	53	47	42

It will be seen that 25% of the men were released within the first 4 years, but that 53% were held more than 8 years. In the light of these figures we decided to divide the 76 cases into three groups: the early releases (the 18 men who were held 48 months or less), the 40 long-termers who stayed over 8 years, and the intermediate group of 18 men, who were held between 4 and 8 years. These three groups were then compared over the variables collected for the Case Register to see what, if anything, distinguished them.

Comparisons on the historical and demographic data revealed little of interest – the groups were not different on items such as age, early family history, social class or previous psychiatric hospitalisation. Criminal history distinguished the groups only in respect of previous sexual offences: 6% of the short-stay men compared with 29% of others had such convictions ($P<0.12$).

There were major differences between the groups as regards the offences for which the men had been sent to Broadmoor: Table III shows the position.

It can be seen that the long-stay group was made up largely of men who had committed offences of violence against the person, sexual or otherwise, while the men whose offences did not physically injure other people accounted for the majority of the short-stay group. The figures in Table III, showing how long different kinds of offenders were detained, have something of the air of a tariff. Long-term confinement followed 76% of homicides, 62% of non-fatal attacks, 55% of sex offences and 25% of arson cases.

TABLE III
Psychopaths admitted 1972–1974: Offence, victim and length of stay

	Short-stay *n* = 18		*Intermediate* *n* = 18		*Long-stay* *n* = 40	
	n	%	*n*	%	*n*	%
Offence[1]						
Homicide	2	11.1	2	11.1	13	32.5
Sexual offences	2	11.1	7	38.9	11	27.5
Wounding, assault	4	22.2	2	11.1	10	25.0
Arson	7	38.9	5	27.8	4	10.0
Other	3	16.7	2	11.1	2	5.0
Victim[2]						
No victim	11	61.1	8	44.4	7	17.5
Family member	4	22.2	2	11.1	9	22.5
Stranger	1	5.6	7	38.9	17	42.5
Other	2	11.1	1	5.6	7	17.5

1. Long-stay vs others for homicide/wounding, sex/arson, other offences: χ^2 10.84 2 d.f., $P<0.001$.
2. χ^2 15.34 6 d.f., $P<0.018$.

When offender/patients are admitted to the special hospitals, the consultants are asked to provide the Case Register with a diagnosis, and also with an assessment of the motive for the offence (e.g. self defence, revenge, sex, etc.). Some of these motive and diagnostic ratings proved to be related to the patient's length of stay. First when a sexual motive had been attributed to the offence (and this was done in 45% of cases) the patient was very unlikely to be in the short-stay group. Of 34 such men, only two (6%) fell into this group, whereas for 42 offenders to whom sexual motives were not attributed, 16 (38%) were released in their first 4 years ($\chi^2 = 11.07$ d.f., $P<0.004$).

Another type of motive assessment that was significantly related to length of stay was when the consultant had at admission recorded the offence as being 'apparently motiveless'. Of the 15 men so categorised, 13 (87%) fell into the long-stay group ($\chi^2 = 8.68$ 2 d.f., $P<0.013$).

Taking the long-stay group as a whole, 85% were men whose offences had been regarded as either 'motiveless' or sexually motivated, whereas only 17% of the short-term group came into this category.

Two other ratings were also found to be associated with length of stay. One was the attribution of the offence to motives of 'jealousy or revenge'. This rating was given to 22 men, and they were significantly over represented in the short-term group: more than half (56%) of the patients in this group had their offences attributed to this motive (χ^2 9,28 2 d.f., $P<0.01$). Further analysis showed that arsonists had more often ($P<0.01$) than other men had their offences ascribed to jealousy. Also, we found that men who were regarded as having committed their offences out of motives of jealousy were more often than others ($P<0.03$) given the diagnosis of 'transient situational reaction'. This diagnosis was made for seven men (9%) and five of them fell into the short-stay group ($P<0.007$). From these findings it seemed that shorter lengths of stay were associated with offences which caused no personal injury and which the doctors regarded as 'one-off' cases arising in temporary circumstances. In terms of common sense expectation, the results are unsurprising, but they do raise the question of whether some of these offenders should ever have been committed to Broadmoor as suffering from psychopathic disorder: it is not easy to see how offences which, on admission, are ascribed to a transient situational reaction can have been the result of "a persistent disorder or disability of mind" (section 4(4) MHA, 1959).

Mentally ill men

Table I showed that these men came to Broadmoor under a wider range of legal categories than did the psychopathic. How long the mentally ill men in the various legal categories were held was clearly of interest, and Table IV divides the men admitted between 1972 and 1974 into the three length of stay groupings.

It will be seen that the civil patients (S.26 in the table) tended to stay longer than offenders admitted under hospital orders. Two factors were probably relevant here. First, the civil patients had come to Broadmoor from psychiatric hospitals where they had proved difficult to manage (in over a third of cases they had attacked hospital staff), and it was therefore likely to have been difficult to return them. Secondly, there was evidence in the data that these men were on admission more severely and chronically ill than the patients who had committed serious offences: they had more often been hospitalised before.

Different considerations govern the release from Broadmoor of patients in the various legal categories. As far as hospital order cases are concerned, the release of restricted patients who are subject to statutory aftercare and supervision is obviously approached differently from the release of unrestricted offenders to whom these provisions do not apply. For these reasons, length of stay needs to be examined separately for each legal category. However, only in the case of restricted hospital order cases were our numbers large enough to permit comparisons to be made between the short- and longer-stay men. Our analyses were therefore confined to this group. It comprised all men subject to restricted hospital orders who were admitted from the courts between 1972 and 1974 with a MHA classification of mental illness. Excluding a few men who died in Broadmoor or were sent to prison after committing further offences in the hospital, the group comprised 111 men, 75% of whom had been diagnosed as schizophrenic on admission. Table II shows how long these patients were held.

As with the PD men, we divided the mentally ill into three categories: those released in the first 4 years (46 men, 41%), those held 8 years or more (47 men, 42%), and those in the intermediate group (18 men, 16%). Comparisons were then made between the three groups on the variables available, to see what distinguished the longer from the shorter stay mentally ill patients.

These comparisons revealed no significant differences in age, nor in the data regarding early family history, though there was a trend to suggest that the short-stay men were less likely than others to have suffered disruption in their upbringing. The criminal histories showed that men who had previous convictions for serious offences (violence or arson) were somewhat less likely ($P<0.07$) than others to fall into the short-stay group. Only 27% of the men (compared with 47% of the psychopaths) had such convictions on their records.

Among the PD patients, it was the data relating to the admission offence which distinguished the long- from the short-term groups, but this was not the case among the mentally ill. Table V shows the type of offence committed and what victims, if any, there were; neither of these items was significantly related to length of stay.

Only one factor relating to the offence appeared among the mentally ill to be significantly related to length of stay; this was the consultant's assessment, made on admission, of whether the offence had been sexually motivated. Fifteen men were positively rated in this way, and 11 of them (73%) fell into the long-term group (χ^2 8.49 d.f. 2, $P<0.014$). With this exception, data about their offences did not relate to the length of time that mentally ill patients were held in Broadmoor. Factors relating to the seriousness and chronicity of their illnesses appear to have been more relevant. Since the Case Register does not contain information about the patient's stay in hospital, information about mental state was not available. There were however indications in the data to suggest that length of stay was related to a severity of illness. For example, more of the long-term group (64%) than of the other men (44%) were recorded as being deluded or hallucinated on admission ($P<0.13$), and more of the short-term group (24% compared with 11%) had an admission diagnosis of paranoid state ($P<0.15$). But the previous hospital

TABLE IV
Length of stay and legal category on admission for mentally ill men admitted 1972–1974

	Short-stay		*Intermediate*		*Long-stay*		*Total*	
Legal category	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
S.26	6	28.6	3	14.3	12	57.1	21	100
S.60	7	41.2	3	17.6	7	41.2	17	100
S.65	61	45.2	23	17.0	51	37.8	135	100
S.71 or 72	7	29.2	6	25.0	11	45.8	24	100
CPIA	5	20.0	7	28.0	13	52.0	25	100

TABLE V
Mentally ill men: offence, victims and length of stay

	Short-stay *n=46*		*Intermediate* *n=18*		*Long-stay* *n=47*	
	n	*%*	*n*	*%*	*n*	*%*
Offence[1]						
Homicide	10	21.7	8	44.4	12	25.5
Sexual offences	—		—		2	4.3
Wounding, assault	24	52.2	8	44.4	23	48.9
Arson	4	8.7	2	11.1	7	14.9
Other	8	17.4	—		3	6.4
Victims[2]						
No victim	12	26.1	2	11.1	12	25.5
Family member	17	37.0	5	27.8	12	25.5
Stranger	4	8.7	3	16.7	3	6.4
Other	13	28.3	8	44.4	20	42.6

1. Long-stay vs others for homicide/wounding, sex/arson, other offences: χ^2 0.12 2 d.f., NS.
2. χ^2 6.59 6 d.f., NS.

experience of the men was perhaps the most telling factor, as representing the seriousness and intractability of their illnesses. Of 77 men who had previously been psychiatric in-patients, 49% fell into the long-term group, whereas of the 31 who had no such history only 23% were detained for more than 8 years ($\chi^2=6.88$ 2 d.f., $P<0.033$).

Discussion

Our analyses were based on the patient's MHA classification on admission to Broadmoor. In fact, many men who are admitted to Broadmoor under the PD classification are, or become, mentally ill. When two of us carried out research in Broadmoor in 1982–1984 (Dell & Robertson 1986), we found that 17% of the resident men who were classified as PD under the Act were diagnosed by their doctors as being mentally ill. Conversely, about 8% of the men classified as mentally ill under restriction orders were said by their doctors not to be suffering from mental illness. It was not usual for cases like these to be reclassified under the Act.

Analyses based on MHA classifications are thus bound to be somewhat misleading. Nevertheless the data did point to a major difference between the detention of PD and mentally ill offenders: length of stay for the former, but not for the latter, was related to the seriousness of their offences. Table III, which shows how long PD men were detained for different kinds of offences has something of the appearance of a tariff; Table V (detention of mentally ill men) does not.

How is this difference to be explained? In our view, it arises from the fundamental differences between the two groups. The mentally ill offender in Broadmoor suffers from disorders (mainly schizophrenic) about which there is an established body of psychiatric knowledge: doctors in the main agree about the recognition and treatment of the symptoms concerned. The patient is seen as having offended in the context of his illness, and if it responds satisfactorily to treatment, transfer to a less secure environment will be envisaged, whatever the offence. The course of the illness, rather than the nature of the original offence, is the dominant consideration in discharge decisions about mentally ill offenders.

For the PD men, the situation is very different. Their offences have not been committed in the context of illness. Had they not committed their crimes, few of them would have been said by doctors to require psychiatric detention. Once in hospital, the course of the disorder attributed to them cannot be monitored by the doctors in the same way as can the course of mental illness: questions of treatment, too, are more uncertain (Home Office, DHSS 1975). An offender admitted to Broadmoor with a schizophrenic illness will receive for it the standard type of drug treatment. The extent to which the illness is controlled by this treatment can then be systematically assessed by mental state examinations. For the PD offender the choice of treatment is seldom so clear, and it is certainly more difficult to establish whether change that is relevant has occurred. It is therefore not surprising to find that in this uncertain territory the seriousness of the patient's offence evidently assumes considerable importance: it is one of the few hard pieces of data available to the discharge decision makers. However, that the length of detention in a hospital should mirror the gravity of the patient's offence does raise fundamental questions about the whole process of committing offenders who are not mentally ill into indefinite psychiatric detention.

Acknowledgements

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References

DELL, S. (1984) *Murder into Manslaughter*. Oxford: OUP.
—— & ROBERTSON, G. (1986) Some aspects of treatment and discharge in Broadmoor special hospital. *Report to the DHSS*.
HOME OFFICE, DHSS (1975) *Report of the Committee on Mentally Abnormal Offenders*, Cmnd 6244, 5, 34. London: HMSO.
SPECIAL HOSPITALS RESEARCH UNIT (1979) Special Hospitals Case Register. *The First Five Years*. London: DHSS.

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Psychiatric Disorder in the Community and the General Health Questionnaire

P. G. SURTEES

Goodchild & Duncan-Jones (1985) have proposed a new scoring method for Goldberg's General Health Questionnaire (GHQ) in which increased weight is given to item responses considered to indicate current (but chronic) affective disorders. This report examines both their revised and the conventional scoring of the GHQ in the context of a longitudinal general population study of psychiatric disorder among women. Sensitivity estimates based upon advised cut-points were found to be higher for the revised than for the conventional scoring scheme, and this difference remained when allowance was made for the duration for which disorders had been present prior to assessment with the GHQ. However, Relative Operating Characteristic (ROC) analysis revealed that while both forms of scoring the GHQ discriminated affective conditions (with or without allowance for their duration) there was no significant difference in their ability to do so. The justification therefore for the post-hoc interpretation of certain item responses as indicators of enduring affective states remains controversial and an issue for future research.

The General Health Questionnaire (GHQ) was developed to facilitate the detection of cases of affective disorder among community and primary care samples (e.g. Goldberg & Blackwell, 1970; Goldberg, 1972, 1978, 1981). Since its introduction, it has been subject to a number of validity studies, used for prevalence estimation, applied in a variety of cultures and languages and evaluated in relation to prescriptive screening (e.g. see Tarnopolsky *et al*, 1979; Henderson *et al*, 1981; Benjamin *et al*, 1982; Radovanovic & Eric, 1983; Banks, 1983*a*; Chan & Chan, 1983; Skuse & Williams, 1984). In addition, several forms of the GHQ have been produced, ranging in length from 12 to 60 items to suit a variety of research needs, but Goldberg has suggested that, where possible, the longer forms of the questionnaire are to be preferred to ensure maximal sensitivity and specificity.

The years since the GHQ was first published have seen the development of a number of semi- or fully structured psychiatric assessment interviews designed to provide current symptom state profiles, to enable diagnoses to be assigned according to a variety of schemes, and to strengthen the basis for aetiological and cross-cultural studies. The availability of these diagnostic methods, with their increasing emphasis on classifying *episodes* of psychiatric disorder rather than health state during particular pre-interview periods, has enabled estimates to be made of the timing of clinical changes, and of how long given symptom states have persisted, and enabled times of remission from illness to be documented. Such information, allied to that obtained from the GHQ, provides the basis for an examination of the recurrent issue of whether this questionnaire fails to detect *chronic* affective conditions (Finlay-Jones & Murphy, 1979; Benjamin *et al*, 1982; Williams 1983; Banks, 1983*b*; Skuse & Williams, 1984). As such disorders are usually found to represent about half of those contributing to point prevalence estimates in both general population and primary care settings (e.g. see Goldberg & Huxley, 1980; Brown *et al*, 1985; Surtees *et al*, 1986), clarification of the relationship is required.

In a largely exploratory study, Goodchild & Duncan-Jones (1985) proposed a new scoring of the GHQ motivated by reports that the questionnaire frequently failed to detect chronic disorders. They challenged Goldberg's (1978) assertion that 'as usual' responses to items on the questionnaire only indicated normal functioning; believing that such responses could understandably be elicited from those chronically impaired at the time of assessment. On this basis therefore, Goodchild & Duncan-Jones divided the 30-item GHQ into equal item sets distinguished by whether or not an 'as usual' type of response could be a 'potential indicator of chronic difficulty or illness'. The revised scoring provided improved estimates of sensitivity and specificity, showed greater stability in repeated use over time and was psychometrically more satisfactory than the conventional method of scoring the GHQ (results from a general population sample incorporating a longitudinal design; Henderson *et al*, 1981). However, their research design made only limited use of a semi-structured psychiatric interview to assess present state, no attempt being made to determine either onset or remission of identified disorders. Chronic disorders meeting diagnostic criteria could not therefore be distinguished from acute disorders in the assessment of the modified scoring scheme. It is clear then that further investigations of the revised scoring scheme on new data are warranted, but such investigations must incorporate diagnostic information which allow clear case status designation. The findings would then be helpful in enabling a choice to be made between the two scoring methods. The present report therefore presents results obtained from contrasting the revised with the conventional scoring of the GHQ in the context of a longitudinal general population study of psychiatric disorder among women.

The research had the following specific aims in relation to the usual and the revised scoring of the questionnaire:

(a) to examine the psychometric properties of the two scoring methods, particularly in repeated use over time;
(b) to estimate sensitivity and specificity associated with each scoring method and to examine change in such estimates with time;
(c) to investigate the relationship between case status (with or without allowance for chronicity) at each of two interviews 6 months apart and that accorded by the two GHQ scoring methods.

These aims represent an attempt to repeat aspects of the procedure reported by Goodchild & Duncan-Jones (1985) and additionally to assess, through the specific examination of chronic conditions, whether there is any justification for adopting a modified scoring of the GHQ.

Method

The community study was undertaken in a geographically discrete area of Edinburgh and based upon a random sample of 576 women aged 18–65 years. A subsample of those women initially interviewed were approached for re-interview 6 months and 1 year later. Details of the design and methodology of the initial survey, of the follow-up and of response rates are provided in two earlier reports (Surtees *et al*, 1983, 1986). Briefly, two groups of women were selected for follow-up. In sample A were those who met *either* the Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) *or* the diagnostic scheme allied to the Present State Examination (PSE; Wing *et al*, 1974; Wing & Sturt, 1978) for a current psychiatric disorder. In sample B were those who were currently free of psychiatric disorder but had one or more of a set of putative vulnerability factors being recorded as present; *or* were included in a 40% random sample of those women who were both currently well and had none of the factors recorded as present. The ten factors chosen were (a) none or very poor relationship with a confidant, (b) none or very little diffuse support, (c) living alone, (d) reporting a poor relationship with one or more household members, (e) living with three or more children aged <14 years, (f) loss through death of one or both parents before age 11 years, (g) separation from either or both parents for ⩾1 year before age 11 years, (h) unemployment of spouse/cohabitee, (i) prior contact with health services for 'nerves', (j) low self-confidence.

At each interview, the subject's psychiatric status was evaluated using a semi-structured clinical interview, the Psychiatric Assessment Schedule (PAS). This was based on the first 40 items of the PSE and selected items from the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978). The PAS provided a detailed assessment of each respondent's psychiatric status for the month preceding each interview, and in addition sought information about her clinical state during the inter-interview time periods in order to establish accurately when the onset and remission of any illness episode had occurred. Further details describing the PAS are given in an earlier report (Dean *et al*, 1983).

The validation procedures adopted for all the interviewers' assessments completed during the follow-up were identical to those applied in the first stage of the study and reported elsewhere (Surtees *et al*, 1983). In brief, if after checking interview material, evidence of psychiatric illness was found, then the audiotape of the interview was listened to and re-rated by staff. Consensus symptom ratings were then established by joint discussion between interviewer and a staff rater.

Where respondents had provided interview information from two or three assessments, then two staff examined all available clinical material obtained from the interviews. The objectives were to re-evaluate all clinical change points, to date them in relation to each respondent's last interview and to apply the RDC scheme to all available symptom data.

At each follow-up, respondents completed the 30-item GHQ which was scored according to the conventional and, for this paper, also the revised method. Full details of the revised scoring procedure are given in Goodchild & Duncan-Jones (1985) and are therefore not repeated here. To facilitate comparison with that work, this paper will adopt an identical method of score designation. Those 15 GHQ items considered to be potential indicators of chronic illness were defined as set N, with the remainder set P. The four GHQ response categories ('not at all' (code 1); 'no more than usual' (code 2); 'rather more than usual' (code 3); and 'much more than usual' (code 4)) were then conventionally scored, with codes 1 and 2 being scored 0 and codes 3 and 4 scored 1. Under the revised scoring, all set P items were similarly scored, but set N items with assigned codes 2, 3, or 4 were scored 1.

Results

For most of the analysis the distinction was retained between those women re-interviewed who met the RDC for a psychiatric disorder at initial interview, sample A, and those also selected for follow-up but who were assessed as 'well' at initial interview, sample B. As no significant difference in case inception rates was found between the two subsamples comprising sample B (Surtees *et al*, 1986), for present purposes they were treated as one group.

Table I displays the distribution of responses to set N and set P items by sample and follow-up (F1 and F2 indicate first and second follow-up respectively). As with the Canberra study, these results reveal the 'as usual' responses

TABLE I

Distribution of responses (averaged %) within each set, for each follow-up of each sample

	Sample A		*Sample B*	
	F1 (*n*=*61*)	*F2* (*n*=*59*)	*F1* (*n*=*313*)	*F2* (*n*=*283*)
N items				
Code 1	26	31	55	53
Code 2	42	48	35	37
Codes 3 and 4	32	21	10	10
P items				
Code 1	13	23	19	15
Code 2	62	61	73	76
Codes 3 and 4	25	16	8	9

(code 2) to be more frequently found among the set of P items than among the N set, particularly among sample B.

Table II shows the mean GHQ (conventional scoring) and CGHQ (revised scoring) scores by sample and follow-up. Also indicated is the proportion of women with score values at or above the appropriate scale cut-points at each follow-up. In accordance with expectations, the proportions of women above the cut-points (on both scales) drops between first and second follow-up assessments for sample A (those all with RDC diagnoses at the start of the study), but not sample B. However, the change can be seen to be proportionately greater for the conventional scoring (GHQ) than for the revised scoring of the questionnaire (CGHQ).

A further indication of the consequence of adopting the revised rather than the conventional scoring is revealed by comparing the cumulative distributions of respondents with given scale scores. The results for samples A and B are shown in Figs 1 and 2 respectively. In Fig. 1, with the conventional scoring of the GHQ, just over 20% of sample A respondents at first follow-up and almost 40% at second follow-up obtain a zero score, in marked contrast to the results obtained with the revised method. The difference in this respect is particularly pronounced in Fig. 2, where the analysis is based upon a sample more representative of the general population. The almost linear increase in the CGHQ cumulative distribution may be an advantageous psychometric property for future research application, as it more closely approximates a normal distribution than that based upon conventional GHQ scoring.

TABLE II

GHQ and CGHQ scores

	Sample A		*Sample B*	
	F1 (*n*=*61*)	*F2* (*n*=*59*)	*F1* (*n*=*313*)	*F2* (*n*=*283*)
GHQ (conventional scoring)				
Percentage scoring $\geqslant 5$	55.7	37.3	19.5	21.2
Mean	8.6	5.5	2.7	2.9
Mean/mean F1	1.0	0.6	1.0	1.1
CGHQ (revised scoring)				
Percentage scoring $\geqslant 13$	67.2	52.5	18.5	26.1
Mean	14.9	12.8	8.0	8.4
Mean/mean F1	1.0	0.9	1.0	1.1

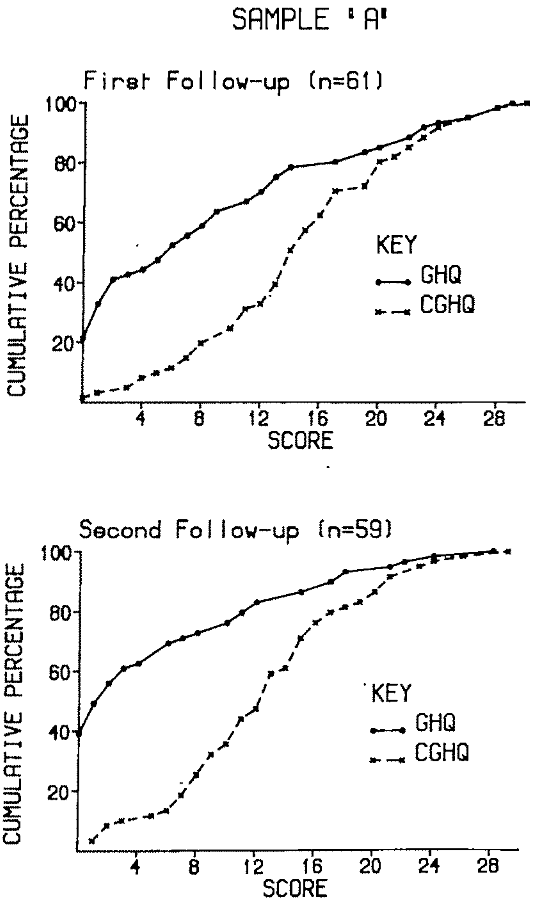

FIG. 1 Cumulative distribution of women in sample A by their GHQ and CGHQ score values at each follow-up.

Estimates of sensitivity (proportion of 'true' cases correctly identified) and specificity (proportion of 'true' normals correctly identified) associated with the advised cut-points on the GHQ and CGHQ scales were determined next. Analyses were undertaken for the samples separately and when combined. The results are shown in Table III.

The indices are intended primarily to provide a basis for comparison between the two scoring methods and not as precise estimates in view of the deliberate sampling biases adopted in this study. The results reveal that the sensitivity estimates based on the CGHQ equal or more usually exceed those determined from the GHQ. The difference is particularly marked at the second follow-up for sample A

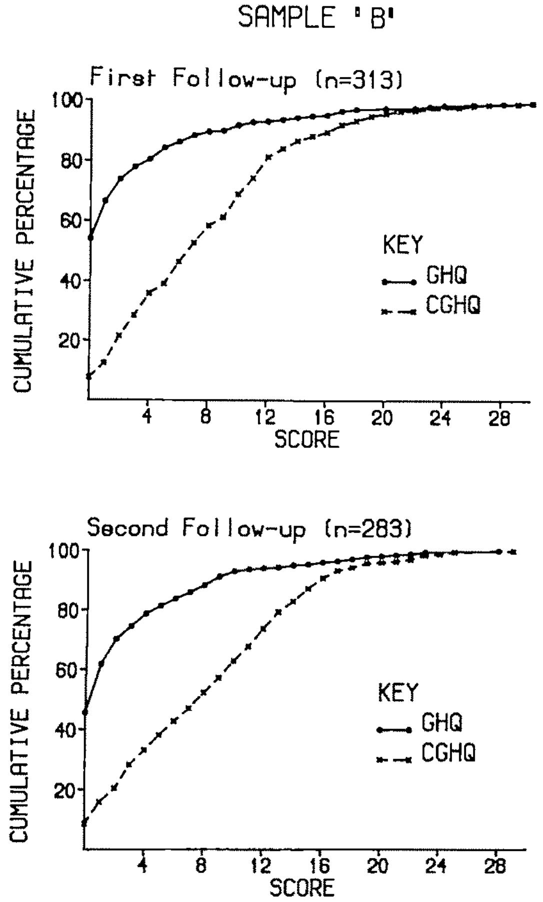

FIG. 2 Cumulative distribution of women in sample B by their GHQ and CGHQ score values at each follow-up.

where, for many, the duration of continuing RDC disorders will have already exceeded one year. While there appears to be a price in terms of specificity, these results do suggest that *perhaps* the modified scoring of the GHQ enhances detection of chronic disorders.

Table III additionally shows the percentage of each sample within an episode of RDC disorder at the time of GHQ assessment. For comparison, the percentage of women at or above the cut-points on the two GHQ scoring methods was shown in Table II. These results taken together reveal somewhat paradoxical findings. While there are a number of reports which have found prevalence estimates of psychiatric disorder to fall with repeated assessment (e.g. Henderson *et al*, 1981; Tennant *et al*, 1981; Robins, 1985), there is no obvious explanation for the phenomenon (see Henderson *et al* (1981) for a discussion of some alternatives). These results clearly show that, unlike the Canberra findings, the GHQ-based prevalence estimates in this study (sample B) remain stable or indeed increase based upon the revised scoring.

Also shown in Table III are the values of Y (Yules' coefficient of colligation) associated with each case/cut-point analysis. It has been suggested recently (Spitznagel & Helzer, 1985) that this measure has particular advantages over the more usually determined κ statistic for certain investigations of reliability and validity. In particular, for studies of validity the measure Y is totally independent of prevalence. The Y values in Table III were determined from smoothed pseudo-Bayes estimates of the cell probabilities (Bishop *et al*, 1975, equations 12.7–18 and 12.7–19), a procedure considered to be superior to that based upon raw cell values. In addition, the method enables Y estimates to be calculated from 2×2 contingency tables containing zero counts. As can be seen, the Y estimates in Table III suggest an advantage (if only marginal) in adopting the revised over the conventional scoring of the GHQ.

The results presented so far are based upon the advised or working cut-points usually adopted for estimating validity coefficients associated with the scales. An alternative method is to examine the variation in true

TABLE III

Sensitivity and specificity of GHQ and CGHQ scores to RDC case status

	Sample A		*Sample B*		*Combined*	
	F1 (n=61)	*F2 (n=59)*	*F1 (n=313)*	*F2 (n=283)*	*F1 (n=374)*	*F2 (n=342)*
Percent with RDC disorder[1]	55.7	40.7	7.0	3.5	15.0	9.9
GHQ ≥5						
Sensitivity	79.4	50.0	86.4	100.0	82.1	64.7
Specificity	74.1	71.4	85.6	81.7	84.6	80.5
Y coefficient[2]	0.51	0.12	0.71	0.87	0.66	0.46
CGHQ ≥13						
Sensitivity	88.2	75.0	90.9	100.0	89.3	82.4
Specificity	59.3	62.9	86.9	76.6	84.6	75.0
Y coefficient[2]	0.51	0.34	0.77	0.83	0.74	0.57

1. Nominal prevalence.
2. Based upon pseudo-Bayes estimation.

positive (sensitivity) and false positive (complement of specificity) proportions with changes in GHQ and CGHQ scale cut-points. This procedure, known as Relative Operating Characteristic (ROC) analysis, was originally used for research into signal detection using radar, but has subsequently been adopted in a variety of research areas where indices of comparative accuracy are required. The method has enabled, for instance, an evaluation of two diagnostic imaging techniques, computed tomography and radionuclide scanning (Swets *et al*, 1979). The application of the method to epidemiological studies is only very recent (Erdreich & Lee, 1981) and particularly so to psychiatric epidemiology (Mari & Williams, 1985).

The ROC method was applied to the GHQ and CGHQ scores obtained in this study, using a computer program developed by Dorfman & Alf (1969), but modified by Metz *et al* (1984; ROCFIT). The program provides fitted (maximum-likelihood) estimates of both sensitivity and false positive rate for all chosen scale cut-off points. ROC curves can then be plotted to these estimates. The area under each curve is used as an index of the detectability of a scale and varies between 0.5 (discrimination no better than chance) and 1.0 (scale with perfect discrimination). The ROCFIT program provides the area under the fitted curve and its standard deviation as part of the output.

Table IV shows the area estimates (and that of their associated variance) obtained for the conventional and modified scaling of the GHQ by sample and by follow-up. A two-sample Z test was applied to each pair of area estimates to establish whether they were significantly different (Erdreich & Lee, 1981). None of the analyses revealed significant differences in area estimates, and in these terms therefore both scales were found to be equally good, but indistinguishable from each other, as discriminators of women with RDC disorder.

The nature of the relationship between the fitted ROC curves for the two scales is shown in Figs 3 and 4 (for samples A and B respectively). These figures show the extent to which each ROC curve deviates from a linear ascending diagonal, and therefore the degree to which discrimination exceeds chance. The figures underpin the results shown in Table III, in that the detection of case status among sample A is clearly less successful than among sample B.

To examine the relationship between the two GHQ scoring methods and case detection further, women were classified according to their RDC case status at *each* of the two follow-ups. The percentage correct case status allocation by the advised cut-points on the two scoring methods was then determined. The results are shown in Table V. Due to missing values at either follow-up, the database available for analysis was reduced below that reported on in previous tables.

Of particular interest was the relative success with which the conventional as opposed to the revised scoring identified those women fulfilling the RDC at *both* follow-ups. Table V shows that the CGHQ was more successful than the GHQ in correctly allocating such women, and as Table III hinted, this appeared particularly so among those with enduring disorders in sample A. However, so far neither those analyses based upon advised cut-points nor those using ROC analysis have included any specific allowance for the *duration* of RDC disorders. To assess claims that a revised scoring of a widely used screening instrument will enhance detection of chronic conditions, allowance should be made for episode duration. This was attempted in the following analyses.

For sample A a chronicity criterion of two or more years was adopted, while because of constraints imposed by the limited follow-up period, when both samples were combined this was reduced to six or more months. Those women with RDC disorders but not meeting the chronicity criteria were regarded as 'acutes'. Analyses then determined the success with which the advised cut-points on the two GHQ scoring methods correctly identified the various subgroups of women. The results are displayed in Table VI.

The results, although based on relatively small numbers, suggest that adopting the revised scoring of the CGHQ increases the percentage of women identified with chronic RDC conditions. It would appear also that there is neither a compensatory loss of those women identified with acute conditions nor a substantial increase in false positives.

As before, these analyses were complemented by adopting the ROC procedure with the chronicity criteria varied as in Table VI and with scale discrimination being assessed in relation to those women with and without chronic RDC conditions. The results in terms of the areas (and their variance) under the resulting ROC curves are shown in

TABLE IV

The areas (and their variance) under the ROC curves determined according to conventional (GHQ) and modified (CGHQ) scoring of the questionnaire

	Sample A		*Sample B*		*Combined*	
	F1 (n=61)	*F2 (n=59)*	*F1 (n=313)*	*F2 (n=283)*	*F1 (n=374)*	*F2 (n=342)*
GHQ						
Area	0.803	0.698	0.960	0.952	0.907	0.805
Variance	(0.0038)	(0.0078)	(0.0004)	(0.0005)	(0.0006)	(0.0026)
CGHQ						
Area	0.787	0.747	0.921	0.971	0.907	0.860
Variance	(0.0033)	(0.0041)	(0.0011)	(0.0002)	(0.0005)	(0.0010)

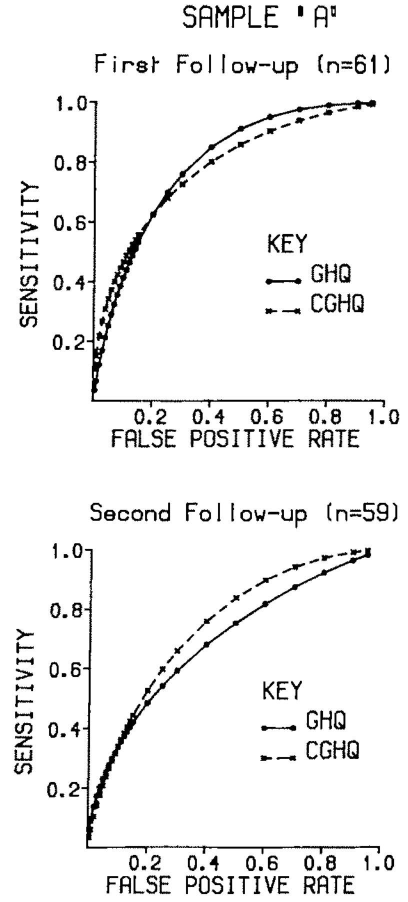

Fig. 3 Fitted ROC curves of the GHQ and CGHQ for sample A at each follow-up.

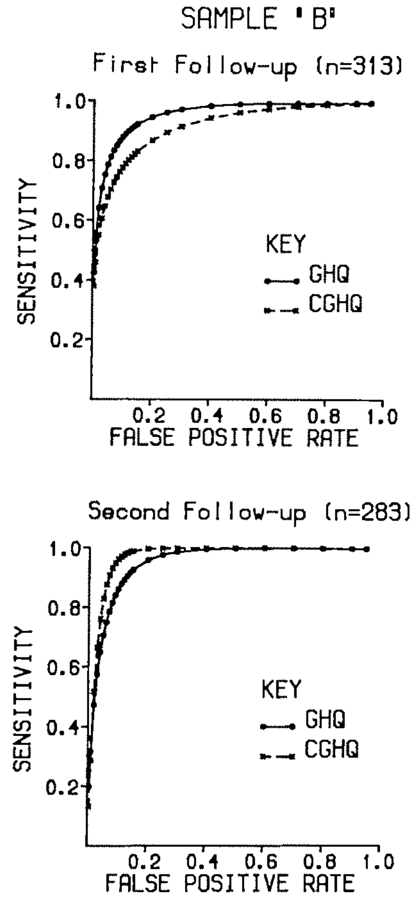

Fig. 4 Fitted ROC curves of the GHQ and CGHQ for sample B at each follow-up.

Table VII. Analyses revealed no significant differences between any of the area values (e.g. for the second follow-up, combined samples $Z = 0.97$).

Discussion

The attempt to screen successfully for affective conditions with only limited use of self-report questioning methods, while desirable from a multitude of standpoints, is necessarily hazardous. Perhaps of primary significance is that validation of the screening measure is absolutely dependent upon what Robins (1985) has called the 'erratic standard' of the clinician's interview; that is, the psychiatric assessment has to be assumed to be correct (also, see Williams, 1986). Additionally, variations in the diagnostic criteria of different schemes and differences in the conceptualisation of episodes of psychiatric disorder contribute to difficulties in understanding what may be a changing validity criterion. A further compounding factor is of course that the questionnaire-based screening method transfers responsibility to the respondent for interpreting the meaning of items; severity of and change in affective state therefore have no common reference. These issues are well known, but it is relatively difficult to investigate their specific effects.

The suggested revised scoring of the GHQ proposed by Goodchild & Duncan-Jones (1985) reinterprets certain responses to particular scale items. However, the Canberra investigation, through only limited adoption of a standardised psychiatric assessment method, did not contrast the conventional with the revised scoring method in detecting long-term as opposed to short-term affective conditions. The scoring methods were only examined in relation to psychiatric state at each interview. As Table V in this

TABLE V
Percentage correct case status assignment by GHQ and CGHQ scores

		Sample A			Sample B			Combined		
Case status at F1	*Case status at F2*	*f*	*Percentage correct by GHQ*	*Percentage correct by CGHQ*	*f*	*Percentage correct by GHQ*	*Percentage correct by CGHQ*	*f*	*Percentage correct by GHQ*	*Percentage correct by CGHQ*
Non-case	Non-case	20	55.0	45.0	256	75.8	73.0	276	74.3	71.0
Case	Non-case	13	53.8	53.8	12	8.3	25.0	25	32.0	40.0
Non-case	Case	6	50.0	50.0	2	50.0	100.0	8	50.0	62.5
Case	Case	16	37.5	62.5	8	87.5	100.0	24	54.2	75.0

TABLE VI
Percentage correct case status allocation by GHQ and CGHQ scores among sample A and combined samples A and B; chronicity criteria varied as shown

	First follow-up (F1)			Second follow-up (F2)		
	Chronics	*Acutes*	*Normals*	*Chronics*	*Acutes*	*Normals*
	(n = 21)	*(n = 13)*	*(n = 27)*	*(n = 14)*	*(n = 10)*	*(n = 35)*
Sample A (chronicity criterion ≥2 years)						
% GHQ ≥5	71.4	92.3	74.1	42.9	60.0	71.4
% CGHQ ≥13	85.7	92.3	59.3	71.4	80.0	62.9
	(n = 36)	*(n = 20)*	*(n = 318)*	*(n = 26)*	*(n = 8)*	*(n = 308)*
Samples A and B (chronicity criterion ≥6 months)						
% GHQ ≥5	80.6	85.0	84.6	57.7	87.5	80.5
% CGHQ ≥13	88.9	90.0	84.6	76.9	100.0	75.0

TABLE VII
The areas (and their variance) under the ROC curves determined according to conventional (GHQ) and modified (CGHQ) scoring of the questionnaire; RDC chronicity criteria varied as shown

	First follow-up	*Second follow-up*
	(n = 61)	*(n = 59)*
Sample A (chronicity criterion ≥2 years		
GHQ		
Area	0.609	0.638
Variance	(0.0065)	(0.0123)
CGHQ		
Area	0.646	0.675
Variance	(0.0050)	(0.0071)
	(n = 374)	*(n = 342)*
Samples A and B (chronicity criterion ≥6 months)		
GHQ		
Area	0.859	0.755
Variance	(0.0008)	(0.0045)
CGHQ		
Area	0.856	0.831
Variance	(0.0008)	(0.0017)

study showed, such analyses do not correspond to an examination of the detection of chronic conditions. The results shown in Table VI, with allowance for chronicity criteria, do lend some support to the revised scoring method.

However, the ROC analyses revealed that the conventional and the revised scoring of the GHQ were indistinguishable in their ability to discriminate between those women with and those without RDC disorders, regardless of whether or not allowance was made for the duration of illness episodes preceding GHQ assessment. These analyses, of course, took account of the full scale ranges. In many circumstances the practical application of a screening instrument for detecting psychiatric morbidity rests upon use of a specific advised cut-point. The results of this study have suggested that where the advised cut-points are adopted, there appears to be a marginal advantage in favour of using the revised scoring. However, the assumption underpinning the revised scoring of the GHQ remains controversial and not specifically addressed by this study. The extent to which it is justified must remain an issue for further research.

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References

Banks, M. H. (1983*a*) Validation of the General Health Questionnaire in a young community sample. *Psychological Medicine*, **13**, 349–353.

—— (1983*b*) Community screening for mental illness (correspondence). *British Journal of Psychiatry*, **142**, 427–428.

Benjamin, S., Decalmer, P. & Haran, D. (1982) Community screening for mental illness: a validity study of the General Health Questionnaire. *British Journal of Psychiatry*, **140**, 174–180.

Bishop, Y. M. M., Fienberg, S. E. & Holland, P. W. (1975) Discrete Multivariate Analysis: Theory and Practice. Cambridge, Mass.: MIT Press.

Brown, G. W., Craig, T. K. J. & Harris, T. O. (1985) Depression: distress or disease? Some epidemiological considerations. *British Journal of Psychiatry*, **147**, 612–622.

Chan, D. W. & Chan, T. S. C. (1983) Reliability, validity and the structure of the General Health Questionnaire in a Chinese context. *Psychological Medicine*, **13**, 363–371.

Dean, C., Surtees, P. G. & Sashidharan, S. P. (1983) Comparison of research diagnostic systems in an Edinburgh community sample. *British Journal of Psychiatry*, **142**, 247–256.

Dorfman, D. D. & Alf, E. (1969) Maximum-likelihood estimation of parameters of signal-detection theory and determination of confidence intervals: rating-method data. *Journal of Mathematical Psychology*, **6**, 487–496.

Endicott, J. & Spitzer, R. L. (1978) A diagnostic interview; the schedule for affective disorders and schizophrenia. *Archives of General Psychiatry*, **35**, 837–844.

Erdreich, L. S. & Lee, E. T. (1981) Use of relative operating characteristic analysis in epidemiology. *American Journal of Epidemiology*, **114**, 649–662.

Finlay-Jones, R. A. & Murphy, E. (1979) Severity of psychiatric disorder and the 30-item General Health Questionnaire. *British Journal of Psychiatry*, **134**, 609–616.

Goldberg, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. London: Oxford University Press.

—— (1978) *Manual of the General Health Questionnaire*. Windsor: National Foundation for Educational Research.

—— (1981) Estimating the prevalence of psychiatric disorder from the results of a screening test. In *What Is A Case?* (eds J. K. Wing, P. E. Bebbington & L. N. Robins). London: Grant McIntyre.

—— & Blackwell, B. (1970) Psychiatric illness in general practice: a detailed study using a new method of case identification. *British Medical Journal*, **ii**, 439–443.

—— & Huxley, P. (1980) *Mental Illness in the Community: the Pathway to Psychiatric Care*. London: Tavistock Publications.

Goodchild, M. E. & Duncan-Jones, P. (1985) Chronicity and the General Health Questionnaire. *British Journal of Psychiatry*, **146**, 55–61.

Henderson, S., Byrne, D. G. & Duncan-Jones, P. (1981) *Neurosis and the Social Environment*. Sydney: Academic Press.

Mari, J. D. J. & Williams, P. (1985) A comparison of the validity of two psychiatric screening questionnaires (GHQ-12 and SRQ-20) in Brazil, using Relative Operating Characteristic (ROC) analysis. *Psychological Medicine*, **15**, 651–659.

Metz, C. E., Wang, P. L. & Kronman, H. B. (1984) *ROCFIT*. Department of Radiology and the Franklin McLean Memorial Research Institute, University of Chicago.

Radovanovic, Z. & Eric, Lj. (1983) Validity of the General Health Questionnaire in a Yugoslav student population. *Psychological Medicine*, **13**, 205–207.

Robins, L. N. (1985) Epidemiology: reflections on testing the validity of psychiatric interviews. *Archives of General Psychiatry*, **42**, 918–924.

Skuse, D. & Williams, P. (1984) Screening for psychiatric disorder in general practice. *Psychological Medicine*, **14**, 365–377.

Spitzer, R. L., Endicott, J. & Robins, E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, **35**, 773–782.

Spitznagel, E. L. & Helzer, J. E. (1985) A proposed solution to the base rate problem in the kappa statistic. *Archives of General Psychiatry*, **42**, 725–728.

Surtees, P. G., Dean, C., Ingham, J. G., Kreitman, N. B., Miller, P. McC. & Sashidharan, S. P. (1983) Psychiatric disorder in women from an Edinburgh community: associations with demographic factors. *British Journal of Psychiatry*, **142**, 238–246.

——, Sashidharan, S. P. & Dean, C. (1986) Affective disorder amongst women in the general population: a longitudinal study. *British Journal of Psychiatry*, **148**, 176–186.

Swets, J. A., Pickett, R. M., Whitehead, S. F., Getty, D. J., Schnur, J. A., Swets, J. B. & Freeman, B. A. (1979) Assessment of diagnostic technologies. Advanced measurement methods are illustrated in a study of computed tomography of the brain. *Science*, **205**, 753–759.

Tarnopolsky, A., Hand, D. J., McLean, E. K., Roberts, H. & Wiggins, R. D. (1979) Validity and uses of a screening questionnaire (GHQ) in the community. *British Journal of Psychiatry*, **134**, 508–515.

Tennant, C., Hurry, J. & Bebbington, P. (1981) The short-term outcome of neurotic disorders in the community: demographic and clinical predictors of remission. *Australian and New Zealand Journal of Psychiatry*, **15**, 111–116.

Williams, P. (1983) Community screening for mental illness (correspondence). *British Journal of Psychiatry*, **142**, 310.

—— (1986) The problem of diagnosis in psychiatric research. *Journal of the Royal Society of Medicine*, **79**, 1–2.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

—— & Sturt, E. (1978) *The PSE-ID-CATEGO System: Supplementary Manual*. London: Medical Research Council, Social Psychiatry Unit.

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Long-stay Patients in a Psychiatric Day Hospital
A Case Note Review

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The case notes of all non-psychogeriatric patients attending the Psychiatric Day Hospital at the University Hospital of South Manchester during one week in October 1982 were reviewed. Clinical, demographic, social and occupational data were gathered for all patients, and their attendance at the day hospital monitored for the year following the census period. No significant differences between patients could be found on any of the factors that it was predicted would distinguish short from long-stay patients. The best predictor of length of day hospital attendance in the current admission was the total quantity of previous hospital treatment whether as an in-patient or a day-patient. It is concluded that in the day hospital studied all patients had similar characteristics, and the majority could be seen as 'new long-stay' patients. The implications of this finding are discussed.

The Government White Paper (DHSS, 1975) on day care for the mentally ill endorsed the policy of dividing day care into two sectors; acute treatment to be provided by District Health Authorities with local Social Services providing social care for the chronically disabled. This philosophy of community rather than hospital care reflected the aim of closing large mental institutions and replacing them with psychiatric units attached to District General Hospitals. Recently both the philosophy and the practice of psychiatric day care has been called into question (Royal College of Psychiatrists, 1980; Bender, 1983; Vaughan, 1983*a*,*b*; 1985; Wilkinson, 1984).

A number of studies have highlighted the difficulties in pursuing the government's expressed aims. Edwards & Carter (1979) found in a national survey of day units that over 30% of users of day hospitals had been attending over 1 year compared with 50% in day centres, and that these patients did not make full use of the facilities offered. Pryce referred to the expanding 'stage army' of new long-stay day patients, noting the trend for the psychiatric day patients increasingly to have been in contact with day services for over a year (Pryce, 1982; Pryce *et al*, 1983). These authors doubt the adequacy of service provision for long-stay patients at day hospitals. There are also doubts as to whether day care provides any effective treatment for these patients (Herz *et al*, 1971, 1976, 1977; Vaughan, 1983*a*, 1985).

In a series of papers published in the early 1970s, Gath, Hassal, and Cross (Cross *et al*, 1972; Hassal *et al*, 1972; Gath *et al*, 1973) reported on a study of patients at seven day hospitals in Birmingham, four in traditional mental hospitals and three relatively new units attached to District General Hospitals (DGH units). They found that the two types of hospital day units served two radically different patient populations. Patients attending the day hospitals in traditional large mental institutions were older, had more severe mental illnesses, showed greater morbidity in terms of their past psychiatric history, were more likely to have been attending for long periods before the census and were much more likely to be still attending at the end of the one year follow-up period.

This distinction between the patient populations of traditional and newer DGH day units was felt to be highly relevant to planning of day services. Gath *et al* concluded that it was ill-advised to close down mental hospitals until other provision was proved to be better.

The study in this paper to some extent repeats that of Gath and his colleagues, but in a Psychiatric Day Hospital attached to a District General Hospital in a Health District where there is no large mental institution and no other Health Service provision for day care of psychiatric patients. The prediction was that the two radically different patient populations identified in the Birmingham study could be identified in one DGH unit, with implications for the kind of service provided.

Present study

The day hospital used in the study occupies part of the Department of Psychiatry in the University Hospital of South Manchester providing 80 places per day for psychiatric patients (0.3/1000 resident population). There is no alternative health service facility such as a local mental hospital for the treatment of mental illness in the South Manchester district. There are five clinical teams admitting to the day hospital, totalling nine consultants and ten junior doctors, each team having one full-time nurse. These teams each continue to look after their own patients and thus effectively operate individual services within the day hospital. A full range of occupational, social and recreational activities is offered, and there are wood and metal workshops and an industrial therapy unit.

There are no other health service day facilities for the mentally ill in South Manchester. There is one local authority-funded day centre in the district, plus two day centres in adjoining districts which accept a limited number of patients from South Manchester. There are a few places available in sheltered employment, primarily in Cheadle Royal Industries. All day places are shared with other health districts of Manchester, so the number of day places available for after care from the day hospital varies with demand from other areas.

It was predicted that if patients attending the psychiatric day hospital are divided into subgroups on the basis of their length of stay and rate of discharge then these groups will be distinguishable on a number of variables relating to severity of illness, social and demographic factors, employment before and after discharge and associated physical ill-health.

Method

The study included all 109 patients recorded in the day hospital attendance roll at any time in the week beginning 1 October 1982, excluding those under the care of the psychogeriatric services.

The case notes of all patients were reviewed and the following data collected:

(a) Demographic – age; sex; marital status; recent and past occupations; length of time in work; housing; family.

(b) Clinical – current diagnosis; past psychiatric history; current treatment; date of admission; date of day hospital enrolment and frequency of attendance.

In addition, case notes and other records were perused for the 1-year period following the census to obtain the following information:

(c) Attendance – weekly attendance was obtained from day hospital records for the year following the census week.

(d) Discharge – for those patients discharged information was collected on occupation, housing and disposal following discharge.

Results

Length of stay

Of these patients, 39 (36%) continued on the roll of the day hospital throughout the year of the follow-up period. The 'length of stay' refers to the time from the date of enrolment in the day hospital to the date of discharge or the end of the study, whichever was the longer.

The frequency of lengths of stay did not, however, bear out the prediction that there would be two separable populations of attenders; short-stay patients who were discharged in a matter of months, and long-stay patients. When the cumulative frequencies of length of stay were plotted (as the percentage of patients remaining on roll against time (Fig. 1)) there was no incisura which would have indicated the presence of two populations. The distribution of lengths of stay closely approximated to an exponential as can be seen from its linearity when plotted on a semi-log scale (Fig. 2; correlation coefficient = 0.98). There is a departure from linearity beyond 60 months, suggesting that there may be a distinct very long stay group, but the number of patients involved (five) is very small.

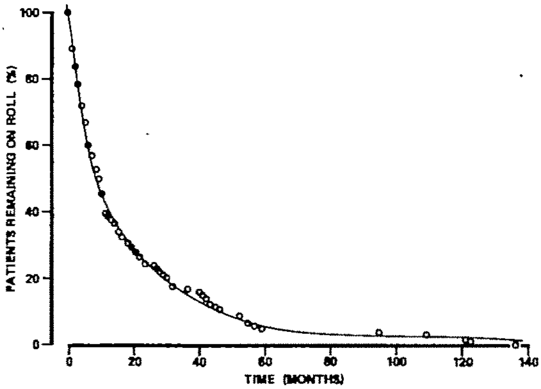

FIG. 1 Percentage of patients remaining on day hospital roll (Cumulative frequencies of months on roll during index admission) vs time from enrolment – linear scale.

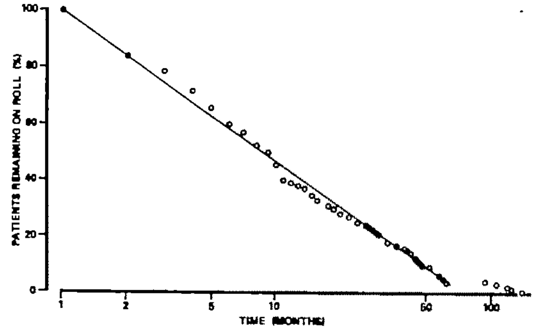

FIG. 2 Percentage of patients remaining on day hospital roll (Cumulative frequencies of months on roll during index admission) vs time from enrolment – log-linear scale.

TABLE I
Comparison of patient characteristics with length of stay

	Test	*Value*	*Significance*
Age at entry to study	R	0.156	0.052
Marital status	χ^2	8.28	NS
Last employment: months before current admission	R	0.328	0.001
Accommodation during day-hospital attendance	χ^2	10.03	NS
Primary diagnosis	χ^2	1.83	NS
Whether sectioned in lifetime	χ^2	1.899	NS
First psychiatric contact: months before current admission	R	0.3818	0.001
Total number of previous in-patient admissions	R	0.399	0.001
Total length of previous in-patient stay	R	0.227	0.01
Total number of previous day-hospital admissions	R	0.47	<0.001
Total length of previous day-patient stay	R	0.521	<0.001
Prescription of four or more categories of drugs	χ^2	28.95	0.0056
Frequency of attendance: days per week	R	0.257	0.003
Occupation after discharge	χ^2	16.18	0.04

Discrete variables analysed using χ^2 with short, medium and long-stay groups. Continuous variables compared using correlation with length of stay.

In the absence of two naturally defined groups, the 109 patients were arbitrarily divided, for the purposes of further data analysis, into the same three groups used in the study of Gath *et al* (1973); short-stay (length of stay $<=3$ months), medium stay (4–9 months) and long-stay (>9 months). Statistical analyses were performed to generate χ^2 for discrete variables and Pearson correlation coefficients for continuous variables.

Demographic and clinical factors

There were almost twice as many women (71) as men (38) attending the day hospital, but the proportion of men to women was the same in all variables subjected to analysis, so the results are presented for the whole population (Table I).

The majority (nearly 60%) of patients attending the day hospital were admitted during the transition between in-patient care and discharge. There was relatively little use of day care as the primary treatment strategy. Of all patients, 62% had diagnoses of schizophrenia or manic-depressive psychosis. It was anticipated that factors related to the severity of the illness would distinguish short-stay from long-stay patients and these factors would include diagnosis, duration of illness and the history of medical intervention, including history of involuntary admission. In fact, no significant differences were found between groups. Nor were the long-stay patients more likely to be single or living alone.

Several factors related to previous psychiatric contact correlated significantly with length of stay. These were: duration of treatment (time between the first recorded contact with psychiatric services and the current admission), number of previous in-patient admissions and total previous in-patient stay, number of previous day-hospital admission and total duration of previous day hospital attendance. Whether a day patient remains on the day hospital roll for a long period thus seems to be most closely related to previous contact with the hospital.

There was a weaker positive correlation between age and length of stay, and this may have been a confounding factor. In order to check this, and determine the relative contributions of the different variables to the overall variance, multiple regression analysis was performed. Length of previous day-hospital stay accounted for 37% of the observed variance, and overall less than half the variance was accounted for by the variables measured in this study. Age, when forced through first in the analysis, accounted for approximately 4% of observed variance and the contribution of other variables was reduced proportionally. Thus age does seem to be a confounding variable, but its contribution is small.

There were no significant differences between the groups on whether or not they were prescribed neuroleptics (oral or depot), anxiolytics, lithium, ECT or antidepressants during the period of the study. The long-stay group were significantly more likely to have received hypnotics ($P=0.01$) and drugs for physical complaints ($P<0.001$) and were more likely to have received four or more different kinds of treatment than the short-stay group. Exactly half the patients in all groups were given an antidepressant in the course of the study, despite the major diagnostic group being schizophrenia.

Much of the treatment provided in a day hospital is social and occupational. However, although an attempt was made to elicit information about non-drug treatments given to patients, these were insufficiently well recorded for this to be possible. For example it was rare for either clinical notes or nursing records to detail occupational or social therapy programmes, and the occupational therapy department was unable to provide this information.

The frequency of attendance during the follow-up period was also recorded. It might be expected that the short-stay group, receiving intensive brief treatment would attend more frequently than the long-stay group, but in fact the reverse proved to be true. Nearly 50% of the long-stay group were attending over 3 days per week whereas nearly

70% of the short-stay group attended 3 days or less. There is a statistically significant correlation between months on the day hospital roll and the frequency of attendance.

During the follow-up period 70 patients were discharged (seven were discharged twice), 15 were readmitted (two twice). Of the patients who were discharged in the follow-up period, three were discharged to in-patient care and two to supervised accommodation, the rest to their own or their family home. Out-patient appointments (usually with a junior doctor on a rotational training scheme), were offered to 61 (88%) of those patient discharged: four (6%) referred back to the care of their General Practitioner and one was followed up by a community nurse. Four patients had no disposal arrangements recorded.

Only five patients (7%) returned to open employment after discharge. 15 (22%) were accommodated at day centres or industrial rehabilitation units and four (13%) were doing voluntary work. The great majority of patients (46; 66%) had no occupation at discharge. The short- and long-stay groups differ significantly in that the short-stay group are more likely to return to open employment ($P<0.01$), but if those patients attending rehabilitation units or involved in voluntary work are classed as 'employed' this difference disappears.

Discussion

Contrary to the original hypothesis, it was not possible to separate, using social or clinical characteristics, two distinct groups of patients with different lengths of stay in the psychiatric day hospital. It may be that the measures used in this study were too insensitive to distinguish these populations, but in fact they are consistent with those that clearly distinguished the two populations in the Birmingham study of Gath *et al.* An alternative explanation is that to all intents and purposes the patients in this study represent a single population. This is supported by the lack of clear differences between long- and short-stay populations, and also by the close similarities between the patients in this study and those described by Gath *et al* (1973) and Pryce *et al* (1982, 1983) as being typical of the long-stay day-hospital patient population.

Length of stay in the day hospital was significantly related to age, duration of illness, length of time unemployed before admission and the amount of hospital treatment received by patients prior to the current day hospital admission. This latter relationship holds both for number of admissions and total duration of stay, whether as an in-patient or a day-patient, although the correlation is strongest with previous day care, which alone accounted for 37% of the variance. These factors may simply reflect the severity of the illnesses suffered by the patients. However, length of stay is not related to differences in other clinical or social factors that might be related to the severity of the illness, such as diagnosis or the use of compulsory orders.

There are other possible explanations of this finding. First, it could be that the majority of patients being referred for day care are those with chronic disabilities. As the day hospital under study is operated by nine different consultants it is not the case that it constitutes a special unit with a defined policy of care for long-stay patients. Second, it is possible that the environment of the day hospital encourages dependency on the service such that patients become institutionalised, and makes it increasingly unlikely that they will be able to find acceptable alternatives. This proposition could be tested by relating the numerical order of previous admissions to the duration of each admission, when the length of stay should increase with successive admissions. Third the average length of in-patient stay in UHSM is relatively short (29.8 days; North West Regional Health Authority, 1986), raising the possibility that with the rapid turnover of in-patient beds the day hospital is increasingly being used for more disabled chronic patients who have gradually replaced the neurotic patients. It is not possible from our data to distinguish between these alternative explanations.

It may be that what determines whether a patient is discharged from the day hospital is the availability of alternative suitable occupation such as employment or a day centre, or some other opportunity arises that provides a more appropriate way of life, such as acceptance at a rehabilitation hostel or group home. There is a significant correlation between the duration of previous unemployment and day hospital stay. In addition two-thirds of patients are discharged to no employment at all. Although there was no difference in the proportions of patients in the artificial short and long stay groups who were in open employment at the time of admission, those who returned to work were all in the short-stay group. The majority of patients were living alone or with other family members and very few (7/109 by the end of the study period) had been in sheltered hostel accommodation. These findings imply that the day hospital setting is not providing the kinds of social and occupational treatments necessary for the effective rehabilitation of long-stay patients. This is supported by the absence from the medical and nursing records of a clear description of non-physical treatments being offered to patients. On the other hand, length of stay is significantly related to the number of different categories of physical treatments given, especially the addition of hypnotics or drugs for physical illnesses to regular psychotropic medication. The lack of adequate records of non-drug treatments and non-medical disposal and the poor recording of social and occupational histories implies that medical staff in ward rounds place comparatively more weight on the medical interventions that may well have the least to offer in the long-term management of such patients.

The implications of these findings are that if day hospitals similar to the one in this study are to be effective then the model of short-term care for acutely ill patients is not an appropriate one. If these patients are only attending the day hospital for lack of suitable day centres or occupational training in the community then there is an urgent need for more alternatives to hospital for such patients. If they are generally too ill or too disabled to function adequately in such alternative settings then a coordinated programme organised toward long-term rehabilitation is required.

References

BENDER, M. (1983) For what and for whom? *Community Care*, January 13, 20–21.

CROSS, K. W., HASSAL, C. & GATH, D. (1972) Psychiatric day care: the new chronic population? *British Journal of Preventive and Social Medicine*, **26**, 199–204.

DEPARTMENT OF HEALTH AND SOCIAL SECURITY (1975) *Better Services for the Mentally Ill*, (Cmnd 6233). HMSO.

—— (1985) *Mental Illness: Policies for Prevention, Treatment and Care*. DHSS Circular dated September 1985.

EDWARDS, C. & CARTER, J. (1979) Day Services and the Mentally Ill. In *Community Care for the Mentally Disabled*. J. K. Wing & R. Olsen eds, Oxford University Press.

GATH, D., HASSAL, C. & CROSS, K. W. (1973) Whither Psychiatric Day Care? A Study of Day Patients in Birmingham. *British Medical Journal*, **i**, 94–98.

HASSAL, C., GATH, D. & CROSS, K. W. (1972) Psychiatric day care in Birmingham. *British Journal of Preventive and Social Medicine*, **26**, 112–120.

HERZ, M. I., ENDICOTT, J. & SPITZER, R. L. (1971) Day versus inpatient hospitalisation: A controlled study. *American Journal of Psychiatry*, **127**, 1371–1382.

——, —— & —— (1977) Brief hospitalisation: a two-year follow up. *American Journal of Psychiatry*, **134**, 502–507.

——, ——, —— *et al* (1976) Brief versus standard hospitalisation: the families. *American Journal of Psychiatry*, **133**, 795–801.

NORTH WEST REGIONAL HEALTH AUTHORITY (1986) Mental Illness Services; Short-term Strategy for Run Down of Large Hospitals. NWRHA Planning Document. Manchester.

PRYCE, I. G. (1982) An expanding 'stage army' of long stay psychiatric day patients. *British Journal of Psychiatry*, **141**, 595–601.

——, BAUGHAN, C. A., JENKINS, T. D. O. & VENKATESAN, B. (1983) A study of long attending psychiatric day patients and the services provided for them. *Psychological Medicine*, **13**, 875–884.

ROYAL COLLEGE OF PSYCHIATRISTS (1980) *Psychiatric Rehabilitation in the 1980s*. Report of the Working Party on Rehabilitation of the Social and Community Psychiatry Section.

VAUGHAN, P. J. (1983*a*) The disordered development of day care in psychiatry. *Health Trends*, **15**, 91–94.

—— (1983*b*) Day care or confusion? *Bulletin of the Royal College of Psychiatrists*, **7**, 184–185.

—— (1985) Developments in psychiatric day care. *British Journal of Psychiatry*, **147**, 1–4.

WILKINSON, G. (1984) Day care for patients with psychiatric disorders. *British Medical Journal*, **288**, 1710–1712.

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Mania Following Head Injury

A Report of Two Cases and a Review of the Literature

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Secondary mania has been described in association with a variety of physical conditions. While there have been a number of reports of mania occurring in individuals with intracranial cerebral lesions, there have been few reporting its occurrence in association with non-penetrating cerebral trauma. Two further cases of mania following non-penetrating head injury and the efficacy of ECT in its management are reported, and a brief review of the literature relating to the subject is given.

Krauthammer & Klerman (1978) reviewed the literature on the occurrence of mania in association with physical illness and drugs. Their criteria for a diagnosis of mania were at least 1 week of elated or irritable mood, and at least 2 of hyperactivity, 'push of speech', flight of ideas, grandiosity, decreased sleep, distractibility and lack of judgement. A previous history of affective disorder or the presence of confusion excluded the diagnosis of secondary mania being made. In their search they found cases of mania thought to be secondary to drug use, metabolic disorders, infections, intracranial neoplasms and epilepsy. Further reports have since added cerebrovascular disease to the list of physical conditions reported (Jampala & Abrams, 1983) and have extended the range of drugs implicated (Stasiek & Zetin, 1985).

Head injury, however, appears to be an uncommon precipitant of mania. There are some reports in the early literature (Symonds, 1937; Tennent, 1937), but these patients are also reported as having clouding of consciousness, and would therefore be excluded on the criteria suggested by Krauthammer & Klerman (1978). Certainly the latter authors were unable to find any instances of mania following head injury to include in their review of the literature; nor were Stasiek & Zetin (1985) in their review of more recently reported cases. However, Whitlock (1982) found two reports of mania occurring after head injury: Dalen (1965) reported two cases of mania in association with both epilepsy and a previous head injury, although the dual pathology and the lack of a clear temporal association make the nature of the relationship unclear, while Cohn *et al* (1977) reported manic symptoms in a 12-year-old male following multiple injuries sustained in a road traffic accident. Two large follow-up studies of patients admitted to neurological and neurosurgical units following head injuries failed to find any cases of mania as either an immediate or long-term complication (Fahy *et al*, 1967; Roberts, 1979). In his series of 1168 adult patients who had suffered head injuries, Ota (1969) reported a single case of mania, but this occurred many years after a 'very slight head injury' and was thought to be unrelated to the insult.

There have been two recent case reports associating mania and head injury. Sinanan (1984) reported a 21-year-old female who developed mania after a head injury sustained in a road traffic accident, and Khanna & Srinath (1985) also reported a case of symptomatic mania in an adolescent female following head injury. Despite a computer search of the literature we have been unable to identify any other reports. In a 6-year retrospective study of all referrals to a psychiatric unit based in the same hospital as the regional neurosurgical unit, two cases of mania closely associated with non-penetrating head injury were identified and are reported here.

Case histories

Case one

A previously fit 70-year-old male was admitted to the neurosurgical unit, Newcastle General Hospital, in October 1981 with a 6-week history of headache, nausea, vomiting and increasing confusion and disorientation. He had suffered a non-penetrating head injury 4 months previously.

Prior to this illness he had been living alone and coping well with home-help and family support. He had been retired for 5 years and widowed for 2 years. He had two daughters living close. Neither he nor any of his family had any prior history of psychiatric disorder.

On admission he was conscious but disoriented in time and place and had impaired short and long term memory.

He was noted to be hallucinating but the nature or modality of the hallucinations was not recorded. Physical examination was unremarkable but a CT scan showed bilateral subdural haematomata. These were evacuated the following day; he improved initially but began to deteriorate 2 weeks later, when a repeat scan showed reaccumulation of the haematomata, which were again drained. A week later a corrugated drain was inserted when a second reaccumulation was discovered. From this time he made an unremarkable recovery and was discharged home 6 weeks after admission, with no neurological or cognitive deficit. He was started on phenytoin, 100 mg four times daily, as a prophylactic.

In February 1982 he was readmitted with dizziness, unsteadiness of gait and a coarse tremor. His serum phenytoin level was 33.0 mg/l and a diagnosis of phenytoin toxicity was made. The phenytoin was immediately stopped and his symptoms rapidly and fully resolved.

In April 1982 he was again readmitted, having become overtalkative and overactive for the preceding 4 weeks. He was described as having become irritable and disinhibited and as behaving 'completely out of character'. He had talked of becoming a missionary. He had begun to give his money away and had ordered new cars and gold teeth, none of which he could afford. He saw no reason to stay in hospital and believed that the ward sister was in love with him. His mood was euphoric. Neurological examination and CT scan were both normal. Psychological assessment showed some impairment of visuospatial abilities but no cognitive deficits. In particular, attention, orientation and long and short-term memory were all normal.

A psychiatric opinion was requested and a diagnosis of hypomania was made. He was started on thioridazine, 25 mg twice daily, which was increased to 50 mg twice daily when he failed to respond. On this dosage he became less grandiose and disinhibited and was discharged to out-patient follow-up.

Two months later he became socially withdrawn and lost most of his interests. He felt miserable and was frightened of meeting people. His sleep and appetite were not disturbed. At interview he looked depressed and there was marked psychomotor retardation, with little spontaneous activity or conversation. His thioridazine was reduced to 25 mg twice daily, and 50 mg of amitryptiline at night was added. This was increased to 100 mg at night as there was only a partial response.

Two months later he was thought to be fully recovered, with no evidence of mood disturbance. He was again coping well at home with home-help and family support. In November 1982 he was discharged to the care of his general practitioner, while still maintained on amitryptiline, 100 mg at night, and thioridazine, 25 mg twice daily.

Case two

A 60-year-old male Punjabi bus-driver was admitted to the neurosurgical unit, Newcastle General Hospital, after a fall in which he had struck his head and been unconscious for 10 minutes. On admission he was oriented in place and person but not in time. He was unable to give a coherent personal history. He could move all his limbs, although there was mild left-sided weakness. There was also some left-sided sensory inattention. A CT scan showed a diffuse area of low density in the right parietal region, consistent with an old infarct. His family subsequently confirmed that he had suffered a left hemiparesis 3 years previously but had made a good recovery, with little residual weakness. There was no other past medical history nor was there any personal or family history of psychiatric disorder.

He was observed in the neurosurgical unit for a fortnight, during which time his conscious level returned to normal. When discharged he was fully oriented with no intellectual defect, although his mild left-sided weakness persisted.

Two months later he became overactive, elated and overtalkative. He began telephoning the Pakistani High Commission in London late at night and became irritable if prevented from doing so. On one occasion he threw his son out of his house following a minor disagreement. During this period he was fully oriented, with no apparent memory impairment. A diagnosis of acute mania was made and admission to the local psychiatric unit arranged, where he was prescribed major tranquillisers. An EEG performed shortly after admission showed an area of delta activity in the right temporal region, which was thought to be related to his previous infarct. An ECG showed him to be in atrial flutter with variable block. Other investigations were normal. In view of his abnormal ECG and EEG findings he was seen by both a cardiologist and a neurosurgeon. Neither felt that they had anything to offer to his immediate management, although an M-mode echocardiogram was arranged, to exclude a cardiomyopathy as a cause of an embolic cerebral infarct. After 2 weeks' treatment he was thought to have settled sufficiently to be managed as an out-patient, but had to be readmitted within a week after again becoming extremely overactive and irritable.

On readmission he was extremely disruptive, with unprovoked outbursts of verbal and physical aggression. He was disinhibited in behaviour and would freely enter other patients' rooms, becoming irritable if intercepted. At no stage during this behaviour was there any evidence of disorientation nor of clouding of consciousness. His dosage of major tranquillisers was increased.

A second EEG showed increased asymmetry compared with his initial record; there was increased delta activity on the right side anteriorly and decreased delta activity on the right side posteriorly. Therefore, a second CT scan was performed, which showed a new area of low density in the left frontal region in addition to the previously noted area in the right parietal region. There was also a mild degree of cerebral atrophy.

As large dosages of major tranquillisers were failing to control his behaviour, an attempt was made to start him on lithium. However, he would not comply with regular oral medication and therefore adequate serum levels were not obtained. Finally a course of bilateral ECT was prescribed. After two treatments he had improved dramatically, and although still elated was much less irritable and overactive and would co-operate with medical and nursing staff in his management. After four treatments he was eating and sleeping normally and was only mildly overcheerful. ECT was then discontinued, but he rapidly became irritable, overactive and restless again. Two further ECTs were given, with the same dramatic improvement.

He was now also complying with oral medication, and adequate serum lithium levels were obtained.

In view of his maintained improvement he was discharged home to continue out-patient treatment. He was seen regularly in the out-patient department over the next 6 months, during which time he remained asymptomatic and his serum lithium levels remained within the therapeutic range (0.4–0.9 mmol/litre). His echocardiogram performed in this interval showed no abnormality. After 6 months he developed a further episode of hypomania and required readmission. He was treated with a course of four ECTs and with oral neuroleptics, and made a good improvement. He was discharged after 1 month. His lithium was withdrawn during this period when he became confused. For the next year he was regularly reviewed as an out-patient and remained well. He was recently readmitted with a third episode of hypomania. The results of all investigations performed during this last admission, including CT scan and EEG, were no different from those previously obtained.

Discussion

Both these cases fulfil the criteria for secondary mania in association with non-penetrating head injury. However, each patient also had other specific intracranial pathology. It is therefore necessary to consider whether these may have contributed to the development of their manic episodes.

The psychiatric sequelae of subdural haematomata have been studied by Fleiss (1945) and by Black (1984). Fleiss reported a single case of recurrent mania associated with recurrent subdural haematoma formation. The manic episodes occurred only when the haematoma had reaccumulated, and responded to either conventional psychiatric treatment of mania or to the tapping of the subdural collection. In a follow-up study of 79 patients, Black reported three cases of depressive illness but none of mania. In our Case 1 the presence of significant subdural collection at the time of the manic episode had been excluded by CT scan. It is therefore unlikely that the presenting symptomatology was associated with the subdural haematomata rather than the head injury *per se*.

Case 2 suffered from cerebrovascular disease in addition to his head injury and developed a new left frontal infarct during the course of his illness. Whereas it is conceivable that this event was the cause of the fall precipitating his head injury, it seems insufficient to account for his subsequent illness and behaviour. However, there are two possible associations between his frontal infarct and his subsequent illness which first need to be addressed. The first is that he may have suffered an organic personality change. The organic personality changes associated with frontal lobe damage are well described by Lishman (1978), and there are many similarities between his description and our patient's presentation. However, the rapid and dramatic improvement that occurred following his courses of ECT make this diagnosis extremely unlikely. A second possibility is that his mania was secondary to the infarct itself. Secondary mania in association with cerebrovascular disease has been described (Jampala & Abrams, 1983). However, in a review of the site and nature of intracerebral lesions which have been reported in association with secondary mania, Cummings & Mendez (1984) could find none sited in the left frontal region, nearly all being right-sided. The old right parietal infarct is unlikely to be significant in this respect, and the most probable explanation of our patient's mania is that it is associated with his preceding head injury.

The pathogenesis of secondary mania is unknown and, in view of the variety of conditions with which it has been associated, probably heterogeneous. In contrast with primary mania, there does not appear to be a major genetic component; both reviews of secondary mania highlight the absence of a family history of affective disorder as a diagnostic pointer towards an underlying organic condition (Krauthammer & Klerman, 1978; Stasiek & Zetin, 1985). Both reviews also comment that it appears to be more common in the elderly, and stress that a careful search for other treatable conditions should be made in elderly patients presenting with a first episode of mania. These two cases support both these observations. It is of interest that both previously reported cases of mania associated with head injury have occurred in younger patients (Sinanan, 1984; Khanna & Srinath, 1985).

Krauthammer & Klerman (1978) suggested that the initial treatment of choice for secondary mania should be that specific to the underlying condition. Only if that led to an inadequate clinical response did they advocate the use of psychotropic medication. Others have since argued similarly and have reported short-lived episodes of secondary mania which have resolved spontaneously (Cummings & Mendez, 1984). If there is no treatment specific to the primary disorder or if such treatment fails to adequately control the manic episode, then conventional treatment of acute mania is instituted. There are a number of reports demonstrating the efficacy of lithium treatment in secondary mania (Rosenbaum & Barry, 1975; Wiesert & Hendrie, 1977; Young *et al*, 1977; Oyewumi & Lapierre, 1981; Cummings & Mendez, 1984), and some suggesting that it should be used as a first choice treatment in preference to neuroleptic medication (Gregersen & Hagen, 1983).

Both our cases developed their manic episodes at a time when there were no treatable primary conditions. Both required urgent psychiatric treatment. Case 1 responded well to neuroleptic treatment and, despite subsequently suffering a minor depressive episode, made a full recovery. Case 2 proved much more resistant to treatment. Initially unresponsive to neuroleptic therapy, he was then treated with lithium. However, this also proved ineffective, due to a lack of compliance, a problem which has not previously been reported in connection with treatment of secondary mania. ECT has long been held to be effective in primary mania, although is rarely prescribed now, with the advent of alternative treatments in the neuroleptic drugs and lithium. Therefore, a course of six ECTs was given, with dramatic effect. When the patient relapsed 6 months later a further course of four ECTs was given, with similar therapeutic effect. This appears to be the first usage of ECT in a case of secondary mania. These cases suggest that non-penetrating head injury may precipitate secondary mania, and add ECT to the treatments known to be effective in its symptomatic management.

References

Black, D. W. (1984) Mental changes resulting from subdural haematoma. *British Journal of Psychiatry*, **145**, 200–203.

Cohn, C. K., Wright, J. R. & De Vaul, R. A. (1977) Post head trauma syndrome in an adolescent treated with lithium carbonate: case report. *Diseases of the Nervous System*, **38**, 630–631.

Cummings, J. L. & Mendez, M. F. (1984) Secondary mania with focal cerebro-vascular lesions. *American Journal of Psychiatry*, **141**, 1084–1087.

Dalen, P. (1965) Family history, the electroencephalogram, and perinatal factors in manic conditions. *Acta Psychiatrica Scandinavica*, **41**, 527–563.

Fahy, T. J., Irving, M. H. & Millac, P. (1967) Severe head injuries. *Lancet*, **2**, 475–479.

Fleiss, A. N. (1945) Psychiatric manifestations of chronic subdural haematomas. *Psychiatric Quarterly*, **19**, 187–194.

Gregersen, B. & Hagen, S. (1983) Secondary mania after cerebral injury. *Ugeskrift fur Laeger*, **145**, 3661.

Jampala, V. C. & Abrams, R. (1983) Mania secondary to right and left hemisphere damage. *American Journal of Psychiatry*, **140**, 1197–1199.

Khanna, S. & Srinath, S. (1985) Symptomatic mania after head injury. *Canadian Journal of Psychiatry*, **30**, 236–237.

Krauthammer, C. & Klerman, G. L. (1978) Secondary mania. *Archives of General Psychiatry*, **35**, 1333–1339.

Lishman, W. A. (1978) *Organic Psychiatry*. Oxford: Blackwell Scientific Publications.

Ota, Y. (1969) Psychiatric studies in civilian head injuries. In *The Late Effects of Head Injury* (eds A. E. Walker, W. F. Caveness & M. Critchley). Springfield, Illinois: Charles Thomas.

Oyewumi, L. K. & Lapierre, Y. D. (1981) Efficacy of lithium in treating mood disorder occurring after brain stem injury. *American Journal of Psychiatry*, **138**, 110–113.

Roberts, A. H. (1979) *Severe Accidental Head Injury*. London: Macmillan Press.

Rosenbaum, A. H. & Barry, B. J. (1975) Positive response to lithium in hypomania secondary to organic brain syndrome. *American Journal of Psychiatry*, **132**, 1072–1073.

Sinanan, K. (1984) Mania as a sequel to a road traffic accident. *British Journal of Psychiatry*, **144**, 330–331.

Stasiek, C. & Zetin, M. (1985) Organic manic disorders. *Psychosomatics*, **26**, 394–402.

Symonds, C. P. (1937) Mental disorder following head injury. *Proceedings of the Royal Society of Medicine*, **30**, 1081–1092.

Tennent, T. (1937) Mental disorder following head injury: discussion. *Proceedings of the Royal Society of Medicine*, **30**, 1092–1093.

Whitlock, F. A. (1982) *Symptomatic Affective Disorders: a Study of Depression and Mania Associated with Physical Disease and Medication*. Sydney: Academic Press.

Wiesert, K. N. & Hendrie, H. C. (1977) Secondary mania? a case report. *American Journal of Psychiatry*, **134**, 929–930.

Young, L. D., Taylor, I. & Holstrom, V. (1977) Lithium treatment of patients with affective illness associated with organic brain syndromes. *American Journal of Psychiatry*, **134**, 1045–1047.

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Ethanol Retards Desensitisation of Simple Phobias in Non-alcoholics

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Twenty non-alcoholic patients with DSM-III defined simple phobias for small animals received behavioural approach tests and exposure therapy while under the influence of a moderately intoxicating dose of ethanol or after placebo administration. Ethanol did not demonstrate any acute anti-anxiety effect, but retarded the rate of behavioural *in vivo* desensitisation, without changing the final treatment outcome. State-dependent effects of ethanol on anxiety were not demonstrated. This study supports prior research indicating no anti-anxiety or 'tension-reducing' effects of ethanol for simple phobias.

Ethyl alcohol is the oldest and most widely used psychoactive substance (Goodwin & Guze, 1979). Pharmacologically, ethanol is classified as a sedative-hypnotic drug (Hoffman, 1983), and is considered to have potential 'tension-reducing' or anxiolytic properties (Cappell & Herman, 1972; Hodgson *et al*, 1979; Lipscomb *et al*, 1980; Pohorecky, 1981; Thyer & Curtis, 1984). This is supported by research suggesting that many ethanol-abusers appear to have pre-existing anxiety disorders, and that these abusers often started their ethanol use as an attempt to reduce the anxious symptoms (Quitkin *et al*, 1972; Mullaney & Trippett, 1979; Bowen *et al*, 1984; Smail *et al*, 1984; Stockwell *et al*, 1984; Thyer *et al*, 1986). However, the DSM-III defines several different anxiety disorders (Task Force on Nomenclature and Statistics, 1980), which might have different pathophysiologies and therefore different responses to psychopharmacological agents (Klein, 1964). In a prior study, two of us demonstrated that ethanol did not reduce the acute anxiety or avoidance associated with exposure to the phobic object in non-alcoholic simple phobics with small-animal phobias (Thyer & Curtis, 1984). Other investigators (Rimm *et al*, 1981) found that ethanol intoxication reduced subjective phobic anxiety but had no effect on phobic avoidance behaviour. The purpose of the present study was to replicate these observations, and to determine if ethanol intoxication influenced the course of *in vivo* exposure treatment in a separate group of non-alcoholic small-animal phobics.

Method

Subjects

Two experiments were performed. Twenty adults (3 male, 17 female; mean age 30.9 ± 8.0 (s.d.) years) were studied. All had discrete simple phobias for small animals as defined by DSM-III. The phobic objects included spiders, snakes, worms, dogs, cats, bees, wasps, fish and birds. The average duration was 23.8 ± 9.4 years. The diagnostic pretreatment evaluation included an extensive interview with an experienced clinician who made the DSM-III diagnosis of simple phobia. Any patient with a history of present or prior substance abuse was excluded. All patients completed several self-rated questionnaires prior to Experiment 1, including the Hopkins Symptom Checklist-90-Revised (Derogatis *et al*, 1973), the Spielberger State-Trait Anxiety Inventory (Spielberger *et al*, 1970) and the Fear Survey Schedule (Wolpe & Lang, 1969). All subjects participated in the first experiment; a subgroup of these ($n = 12$) participated in the second. All subjects gave written informed consent, and the protocol was approved by the University of Michigan Hospitals' Human Use Committee. Pregnant women were excluded.

Procedure

The study was designed to determine if moderate ethanol intoxication during *in vivo* exposure altered the course of desensitisation, and also to determine if state-dependent effects of ethanol on the desensitisation process occurred. State-dependent effects would be demonstrated if subjects showed a greater response to treatment when tested under the condition under which they had been treated than when tested under the opposite condition (i.e. ethanol versus placebo).

The design of Experiment 1 included (a) two behavioural approach test assessments (A1 and A2) before treatment, one while intoxicated and one after a placebo; (b) an assessment immediately after 2 hours of behavioural treatment in the same condition as the treatment session (ethanol or placebo) (A3); and (c) a subsequent assessment under the opposite condition (A4). All sessions were separated by at least 24 hours. Sessions A1 and A2 were counterbalanced so that half of the subjects had A1 under ethanol and A2 under placebo, with the other subjects in the reverse order; subjects were randomly assigned to one order or the other. After A2, each subject was then randomly assigned to ethanol or placebo treatment, which thus dictated the conditions for A3 and A4. All 20 subjects completed Experimental 1 (A1 to A4).

A subgroup of subjects ($n = 12$) participated in Experiment 2. These subjects were treated with exposure therapy in 2-hour sessions until either they calmly permitted unlimited contact with the phobic object or until 20 hours of treatment were completed. All subjects continued to be treated under the same drug condition as that of the first treatment session; seven were ethanol patients and five placebo. At the completion of treatment, subjects were evaluated under both conditions (A5 and A6), in a counterbalanced order. Finally, 10 of these 12 subjects had two further assessments at 6 months post-treatment, and at 12 months, again in counterbalanced order.

Behavioural *in vivo* desensitisation treatment of simple phobias has been described in detail elsewhere (Curtis *et al*, 1976; Marks, 1981). Prior to both assessment and treatment sessions, ethanol or placebo was administered, with monitoring of blood alcohol level using an Alcosensor II portable 'breathalyser'. Ethanol dose was based on body weight, with a goal of obtaining a blood alcohol level of approximately 0.10 g/dl, a moderate level usually associated with some intoxication in inexperienced users of ethanol, and legally associated with intoxication in most states. Ethanol was administered as vodka mixed in iced orange juice. Placebo was approximately 7.5 ml of vodka layered on top of iced orange juice. Ethanol administration, desensitisation treatment, and behavioural assessment were always performed by different staff, who were blind to the outcomes of the other activities. Patients were also procedurally blind to drug condition.

Assessment of therapeutic outcome was evaluated with a behavioural approach test (BAT; described in detail in Thyer & Curtis, 1984). Subjective units of distress (SUDs), rated from 0 (none) to 100 (most ever), and latency of approach (0–20 s) were rated at eight points: (a) outside the room where the phobic object was present, (b) 3 m from the object, with it in sight, (c) 1 m, (d) 0.3 m, (e) indirect contact with the object, (f) brief direct contact, (g) holding the object, and (h) unlimited direct contact. These tests, as well as treatment, were always performed with a live specimen of each subject's phobic object. The BAT was stopped when a subject could not reach the next step within 20 seconds of being requested to proceed. The point of maximum approach (rated 0–100) was used as the third BAT variable. For any step not reached, a SUDs score of 100 and a latency of 20 s were assigned, and average scores for SUDs and latency at the eight points, as well as the maximum approach score, were used as data to be analysed for each BAT.

Data analysis

Analyses were performed separately for SUDs, latency and maximum approach measures. Subjects given ethanol were compared with those given placebo at A1, and A1 was compared with A2 for all subjects. Because ethanol versus placebo condition and order are confounded for A3 and A4, the effects of 2 hours of treatment while intoxicated versus while sober were determined by comparing the two treatment groups for the mean of A1 plus A2 versus the mean of A3 plus A4. Means of A5 plus A6, A7 plus A8, and A9 plus A10 were also used for further comparisons. When appropriate, comparisons were performed only after initial differences had been removed with covariance analyses. Because of the confounding of order and condition, state-dependent effects could not be evaluated at A3 or A4; therefore, these were studied only at A5 and A6, and thereafter. Analysis of state-dependent effects involved determining for the two treatment groups whether subjects showed less phobic avoidance when tested in the same drug condition as treatment versus the opposite drug condition. Duration of treatment to reach therapeutic criterion was compared for the two groups, and finally scores on pretreatment self-rating scales as potential predictors of therapeutic outcome were investigated. Analyses included t-tests, analyses of covariance, and correlation coefficients. All significance levels were $P > 0.05$ for two-tailed tests.

Results

As expected, blood alcohol levels were 0.00 g/dl after placebo administration. After ethanol, levels were in the range 0.08–0.12 g/dl; thus, the goal of 0.10 g/dl was well approximated. During treatment sessions, small further amounts of ethanol, administered in the same way, were sometimes required to maintain these levels. Although some subjects showed signs of intoxication, no subject was ever unable to participate co-operatively in the assessment or treatment. The assessor was occasionally unblinded by the subject's behaviour, but usually could not differentiate reliably between drug verus placebo administration.

There were no significant differences at A1 between subjects given drug and those given placebo on SUDs, latency or approach; there was a tendency for subjects given placebo to approach the phobic object more closely than those given ethanol, and subjects given ethanol showed somewhat larger SUDs scores and longer latencies (Fig. 1). Thus, as in our prior study (Thyer & Curtis, 1984), acute administration of moderate doses of ethanol did not reduce acute phobic anxiety in non-alcoholics. For all subjects there was a significant decrease from A1 to A2 in SUDs ($P < 0.02$) and latency ($P < 0.005$), and a significant increase in approach ($P < 0.04$).

Even though significant differences between groups were not present prior to treatment, non-significant differences were present, especially for approach (Fig. 1). Therefore, comparison of the ethanol group with the placebo group after 2 hours of treatment (mean of A1 plus A2 versus mean of A3 plus A4) was done with an analysis of covariance in which the initial differences were the covariates removed. With these analyses, the placebo group showed a significantly greater reduction of SUDs ($P < 0.02$) and latency ($P < 0.04$), and a weak trend toward significantly more approach ($P < 0.31$). Thus, Experiment 1 demonstrated that intoxication retarded behaviour treatment as measured by SUDs and latency scores (Fig. 1).

Twelve subjects completed treatment in Experiment 2. The average number of hours required for patients receiving placebo to calmly permit unlimited contact with the phobic object was 5.85, while for the ethanol group it was 9.37. Because of much variation from subject to subject this difference was not significant; it might have been with a

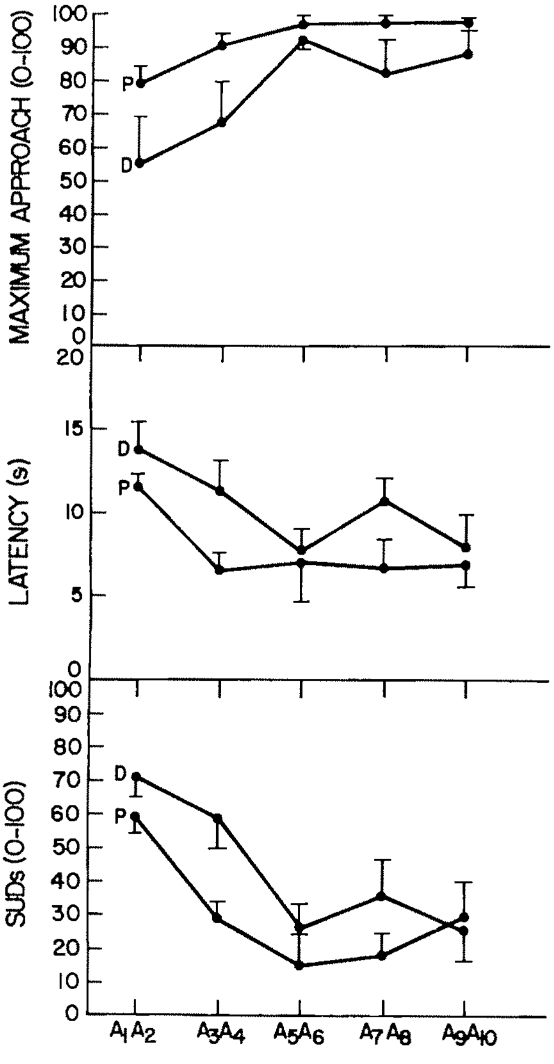

FIG. 1 Means ± s.e.m. for patients treated under drug (D) and for those treated under placebo (P), separately for subjective units of distress (SUDs), latency to approach the phobic object, and maximum approach, represented as mean of A1 plus A2, A3 plus A4, A5 plus A6, A7 plus A8, and A9 plus A10; A1A2 is assessment prior to treatment, A3A4 is after 2 hours of treatment, A5A6 is after completion on all treatment, and A7A8 and A9A10 are 6-month and 12-month post-treatment evaluations.

larger group of patients. This difference is consistent with the finding of Experiment 1 that ethanol retarded treatment progress.

No significant differences in final therapeutic outcomes on any of the three measures could be demonstrated at the completion of treatment, or at the 6-month or 12-month follow-ups (Fig. 1), and no consistent state-dependent effects could be demonstrated. No differences between patients treated with ethanol and those with placebo were observed on any of the pretreatment rating scales. Nevertheless, the State–Trait anxiety measures tended to predict outcome of treatment; higher scores were associated with poorer outcomes. Furthermore, removal of the effects of the State and Trait measures by analysis of covariance diminished the difference in latency between groups after 2 hours of treatment ($P = 0.10$ and 0.12, instead of 0.04, for State and Trait measures, respectively) and also reduced the weak trend in maximum approach ($P = 0.44$ and 0.48 instead of 0.31). However, the difference in SUDs was unchanged. Other potential pretreatment predictors were not useful.

Discussion

The results of this study replicate the prior finding that ethanol in a mildly-to-moderately intoxicating dose does not reduce avoidance behaviour or acute phobic anxiety ('tension') in non-alcoholic simple phobics. Results might be different in alcoholic patients. Furthermore, ethanol appears to slow the progress of behavioural desensitisation exposure therapy, but apparently does not impair the final outcome if extra treatment is administered. It is possible that a different dose might have a different effect. For example, the very common use of ethanol as an intoxicant in people with flying phobias in order to tolerate flying might contribute to the lack of desensitisation often observed in these individuals, especially if moderate-to-severe intoxication slows desensitisation more than a less intoxicating dose, or even totally prevents it.

Several issues in these results are important. First, even with counterbalancing of order of initial assessments with ethanol and placebo, phobic avoidance during the second assessment was significantly less than during the first. Thus, in naïve individuals, a behavioural approach test alone appears to provide some therapeutic benefit, which is probably due to both the exposure involved and the reduction in uncertainty associated with completion of the first assessment. Secondly, latency scores may have been influenced directly by the motoric impairment effects of acute intoxication; this should not, however, influence the effects seen on subjective distress or the similar trend in maximum approach. Thirdly, state-dependent effects were not demonstrated. This may reflect either a lack of state-dependent effects of ethanol in this paradigm despite its effects in other circumstances (Goodwin *et al*, 1969; Overton, 1972; Crow & Ball, 1975; Weingartner *et al*, 1976; Barry & Krimmer, 1978; Schechter, 1978), or an inability to demonstrate them despite their presence because other competing effects were more potent and masked the state-dependent effects. The fact that, under some conditions, not only were the effects not demonstrated but trends were in the opposite direction suggests that masked effects were not present. Fourthly, despite the disruption by ethanol observed in rate of treatment progress, treatment was effective for both groups, and the observed improvement was relatively stable for a year. Finally, the effects of pretreatment anxiety level (as measured by the State–Trait Inventory) on latency and maximum approach after 2 hours of treatment suggest that behavioural phobic severity (avoidance) may be an aspect of simple phobias which is less sensitive to the retarding effects of ethanol on desensitisation. However, pretreatment anxiety level did not modify the retarding effects of ethanol on reduction in subjective phobic anxiety (SUDs).

These results may or may not be generalisable to other sedative–hypnotic drugs or different anxiety disorders. In a similar study of acute phobic anxiety (Whitehead *et al*, 1978), diazepam did increase approach to simple phobic objects, suggesting that ethanol and benzodiazepines have different modes of action in the simple phobia paradigm. Further, ethanol may be an effective anxiolytic drug for other anxiety disorders (Quitkin *et al*, 1972) such as panic disorder, social phobia or generalised anxiety. If true, this finding would imply pathophysiological differences between these different anxiety states.

Our results are congruent with a number of experiments employing animal models of avoidance behaviour which have found that ethanol intoxication retards extinction of avoidance responses (Baum, 1969, 1970, 1971; Skurdal *et al*, 1975). The effects of ethanol intoxication on animal learning of avoidance or extinction are probably due to the CNS depressant effects of the drug, as opposed to the actions of its metabolites such as acetaldehyde (Snell & Harris, 1980). Thus, ethanol may be producing the effects seen in this study by slowing the 'learning' process in desensitisation.

Clinically it has been shown that the concomittant use of diazepam and exposure therapy adds little or no benefit to treatment outcomes with phobic patients, compared with exposure therapy alone or exposure plus placebo (Hafner & Marks, 1976; Bernhardt *et al*, 1980), and that diazepam does not reduce the subjective anxiety experienced by patients during the desensitisation process (Marks *et al*, 1972). Our findings with ethanol and exposure therapy are consistent with those obtained combining another CNS depressant, diazepam, with exposure. Overall our data do not support the hypothesis that ethanol intoxication reduces the anxiety associated with simple phobias in humans, and do support the clinical suggestion made by Marks (1985) that ethanol use should be curtailed by phobic patients being treated with therapeutic exposure.

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References

Barry, H. & Krimmer, E. C. (1978) Similarities and differences in discriminative stimulus effects of chlordiazepoxide, pentobarbital, ethanol and other sedatives. In *Stimulus Properties of Drugs: Ten Years of Progress* (eds F. C. Colpaert, J. H. Rosecrans). New York: Elsevier/North Holland Biomedical Press.

Baum, M. (1969) Paradoxical effect of alcohol on the resistance to extinction of an avoidance response in rats. *Journal of Comparative and Physiological Psychology*, **69**, 238–240.

—— (1970) Effect of alcohol on the acquisition and resistance-to-extinction of avoidance responses in rats. *Psychological Reports*, **26**, 759–765.

—— (1971) Effect of alcohol on the resistance-to-extinction of an avoidance response: replication with mice. *Physiology and Behavior*, **6**, 307–309.

Bernhardt, B. W., Silverstone, T. & Singleton, W. (1980) Behavioural and subjective effects of beta-adrenergic blockade in phobic subjects. *British Journal of Psychiatry*, **137**, 452–457.

Bowen, R. C., Cipywnyk, D., D'Arcy, C. & Keegan, D. (1984) Alcoholism, anxiety disorders, and agoraphobia. *Alcoholism: Clinical and Experimental Research*, **8**, 48–50.

Cappell, H. & Herman, P. (1972) Alcohol and tension reduction: a review. *Quarterly Journal of Studies on Alcohol*, **33**, 33–64.

Crow, L. T. & Ball, C. (1975) Alcohol state-dependency and autonomic reactivity. *Psychophysiology*, **12**, 702–706.

Curtis, G. C., Nesse, R. M., Buxton, M., Wright, J. & Lippman, D. (1976) Flooding *in vivo* as a research tool and treatment method of phobias: a preliminary report. *Comprehensive Psychiatry*, **11**, 101–107.

Derogatis, L., Lipman, R. & Covi, L. (1973) SCL-90: an out-patient psychiatric rating scale – preliminary report. *Psychopharmacology Bulletin*, **19**, 13–28.

Goodwin, D. W., Powell, B., Bremer, D., Hoine, H. & Stern, J. (1969) Alcohol and recall: state-dependent effects in man. *Science*, **163**, 1358–1360.

—— & Guze, S. B. (1979) *Psychiatric Diagnosis*, 2nd edn. New York: Oxford University Press.

Hafner, R. J. & Marks, I. M. (1976) Exposure *in vivo* of agoraphobics: contributions of diazepam, group exposure and anxiety evocation. *Psychological Medicine*, **6**, 71–88.

Hodgson, R. J., Stockwell, T. R. & Rankin, J. H. (1979) Can alcohol reduce tension? *Behaviour Research and Therapy*, **17**, 459–466.

Hoffman, F. G. (1983) *A Handbook on Drug and Alcohol Abuse: the Biomedical Aspects*, 2nd edn. New York: Oxford University Press.

Klein, D. F. (1964) Delineation of two drug-responsive anxiety syndromes. *Psychopharmacologia*, **5**, 397–408.

Lipscomb, T. R., Nathan, P. E., Wilson, G. T. & Abrams, D. B. (1980) Effects of tolerance on the anxiety-reducing function of alcohol. *Archives of General Psychiatry*, **37**, 577–582.

Marks, I. M. (1981) *Cure and Care of Neurosis*. New York: Wiley.

—— (1985) Behavioral psychotherapy of phobic disorders. In *The Psychiatric Clinics of North America: Anxiety Disorders* (eds G. C. Curtis, J. M. Rainey, B. A. Thyer). Philadelphia, PA: Saunders.

——, Viswanathan, R., Lipsedge, M. S. & Gardner, R. (1972) Enhanced relief of phobias by flooding during waning diazepam effect. *British Journal of Psychiatry*, **121**, 493–506.

Mullaney, J. A. & Trippett, C. J. (1979) Alcohol dependence and phobias: clinical description and relevance. *British Journal of Psychiatry*, **135**, 565–573.

Overton, D. H. (1972) State-dependent learning produced by alcohol and its relevance to alcoholism. In *The Biology of Alcoholism, Vol. 2: Physiology and Behavior* (eds B. Kissin, H. Begleiter). New York: Plenum.

Pohorecky, L. A. (1981) The interaction of alcohol and stress: a review. *Neuroscience and Behavioral Reviews*, **5**, 209–229.

Quitkin, F. M., Rifkin, A., Kaplan, J. & Klein, D. F. (1972) Phobic anxiety syndrome complicated by drug dependence and addiction. *Archives of General Psychiatry*, **27**, 159–162.

Rimm, D., Briddell, D., Zimmerman, M. & Caddy, G. (1981) The effects of alcohol and the expectancy of alcohol on snake fear. *Addictive Behaviors*, **6**, 47–51.

Schechter, M. D. (1978) Stimulus properties of ethanol and depressant drugs. In *Drug Discrimination and State-Dependent Learning* (eds B. T. Ho, D. W. Richards & D. L. Chute). New York: Academic Press.

Skurdal, A. J., Eckardt, M. J. & Brown, J. S. (1975) The effects of alcohcol on escape learning and on regular and punished extinction in a self-punitive situation with rats. *Physiological Psychology*, **3**, 29–34.

Smail, P., Stockwell, T., Canter, S. & Hodgson, R. (1984) Alcohol dependence and phobic anxiety states – I: A prevalence study. *British Journal of Psychiatry*, **144**, 53–57.

Snell, D. & Harris, R. A. (1980) Impairment of avoidance behavior following short-term ingestion of ethanol, tertiary-butanol, or pentobarbital in mice. *Psychopharmacology*, **69**, 53–57.

Spielberger, C., Gorsuch, R. & Lushene, R. (1970) *The State–Trait Anxiety (STAI) Manual for Form X*. Palo Alto, CA: Consulting Psychologist's Press.

Stockwell, T., Smail, P., Hodgson, R. & Canter, S. (1984) Alcohol dependence and phobic anxiety states – II: A retrospective study. *British Journal of Psychiatry*, **144**, 58–63.

Task Force On Nomenclature And Statistics (1980) *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn (DSM-III). Washington, DC: American Psychiatric Press.

Thyer, B. A. & Curtis, G. C. (1984) The effects of ethanol intoxication on phobic anxiety. *Behaviour Research and Therapy*, **22**, 599–610.

——, Parrish, R. T., Himle, J., Cameron, O. G., Curtis, G. C. & Nesse, R. M. (1986) Alcohol abuse among clinically anxious patients. *Behavior Research and Therapy*, **24**, 357–359.

Weingartner, H., Adefus, W., Eich, J. E. & Murphy, D. L. (1976) Encoding-imagery specificity in alcohol state-dependent learning. *Journal of Experimental Psychology: Human Learning and Memory*, **2**, 83–87.

Whitehead, W., Blackwell, B. & Robinson, A. (1978) Effects of diazepam on phobic avoidance behavior and phobic anxiety. *Biological Psychiatry*, **13**, 59–64.

Wolpe, J. & Lang, P. (1969) *Manual for the Fear Survey Schedule*. San Diego, CA: Edits.

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Electrodermal Responding and Chlorpromazine Treatment in Schizophrenia

Skin conductance level (SCL) and skin conductance responses (SCR) to a random series of tones were measured in 25 drug-free schizophrenic patients, 15 male and 10 female, before and after standard chlorpromazine treatment. The DSM-III diagnostic criteria were used for selecting subjects. Psychopathology was measured with the Brief Psychiatric Rating Scale. After treatment, patients showed an improved psychopathology and decreased SCL. There was a transition of patients to lower response categories: the number of responders decreased twice and the number of non-responders increased three times. Responders exhibited significantly higher SCL than non-responders and fast habituators were between the two groups.

Investigators in psychophysiology of schizophrenia often face the problem that the material under study is both heterogeneous and variable. For this reason some authors think it important to divide schizophrenics into subgroups (Houlihan, 1977). In recent years, more homogenous subgroups based on autonomic arousal and response patterns have appeared feasible (Venables & Patterson, 1976).

Gruzelier & Venables (1972), using the habituation rate technique, showed that 50% of schizophrenic subjects were non-responders while 50% tended to be hyper-responders with maximal electrodermal responses and diminished habituation. Patterson & Venables (1978) reported, in addition, that a further electrodermal subgroup of patients could be distinguished, showing very fast habituation and only one, or at the most two, responses to stimuli. Gruzelier & Venables (1972, 1973) also found that non-responders have a lower skin conductance level than schizophrenic or normal responders. The above data indicate that schizophrenics may be found in the two extreme poles of somatic arousal.

Other investigators (Zahn, 1976; Frith *et al*, 1979) found only a low proportion of non-responders among schizophrenic patients. Therefore, various views have been expressed on electrodermal responding in schizophrenia, with some investigators claiming non-responding to be a true finding, specific for schizophrenic disorder (Patterson & Venables, 1978; Venables, 1978; Straube, 1979), and some others insisting that this is an artifact due to various factors relevant to the technique of measurement or neuroleptic medication (Lader, 1975; Zahn, 1978). It is true that the majority of studies presenting high proportions of non-responders in schizophrenia used subjects receiving medication.

In this study we investigate the electrodermal responding of a group of schizophrenic patients before and after chlorpromazine treatment.

Method

Subjects

Schizophrenic in-patients admitted to Aeginition University Psychiatric Clinic who had been drug-free for at least 15 days were used for the study. Patients were fully informed about the experimental procedures and consent was obtained before participation in the study. The whole experiment was approved by the Hospital Committee. The diagnostic evaluation of subjects was performed by two experienced psychiatrists who used the DSM-III criteria for schizophrenia. Their diagnosis was based on standard clinical interviews and patients' records. Initially, 38 patients were considered. Six of these did not meet the DSM-III criteria. Four were very disturbed and the staff used medication to treat them before they could be included in the study. Three refused to participate. The remaining 25 patients, 15 male and 10 female, were all able to co-operate for the habituation procedure. Of these, 16 patients were first admissions. The mean age was 26.44 ± 7.71 years, the mean duration of illness was 3.66 ± 3.58 years and the mean duration of school education was 10.48 ± 3.47 years. Diagnostic categories according to DSM-III criteria were: eight undifferentiated, five disorganised, two residual and ten paranoid.

Measures

Psychophysiological and clinical measures were taken before and after treatment with a standard dose of 300 mg of chlorpromazine daily for 2 weeks. Two parameters of electrodermal activity, skin conductance response and skin conductance level, were studied in relation to treatment. The habituation rate to a random series of tones was also recorded. The degree of psychopathology was measured using the Brief Psychiatric Rating Scale (BPRS).

A Lafayette 76406 system was used and the skin resistance level and galvanic skin response were recorded (in kohms) from the palmar surface of the left hand. The standard silver–silver chloride electrodes provided by Lafayette and an electrolyte medium containing sodium chloride were applied at the distal phalanges of the first and third fingers. The skin resistance values were transformed to skin conductance values. Electrodermal response was calculated as a change in log conductance.

Electrodermal responses to tape-recorded simple tones were used for the habituation procedure. Patients were divided according to the number of responses into three subgroups: 1) non-responders, the patients who did not respond to the first three tones, 2) fast habituators, those who responded to the first or two first stimuli only, and 3) responders, the patients who responded to the tones and habituated the orienting response in three to fifteen trials. The criteria for the above subdivision have been used by other investigators (Gruzelier & Venables, 1972; Patterson & Venables, 1978).

An electrodermal response was counted only if it occurred between 1 and 5 seconds after the onset of stimulus, and was defined as a change in log skin conductance greater than 0.003 log μmhos. Following the procedure of Gruzelier & Venables (1972) the orienting stimuli were 15 one-second, 1000 Hz, 85 dB tones, presented at random intervals ranging between 30 and 60 s. The total duration of tone series was 11 minutes. The tones were presented binaurally through headphones after a 6-minute rest period during which a 'white' noise (25 dB) was heard from the tape.

Two values of skin conductance level were taken as measures of background activity, one just before the onset of auditory stimuli and the other one immediately after the end of auditory stimuli.

Psychopathology was assessed before psychophysiological measurements were taken, by two psychiatrists in a common interview. The patients were given standard information and instructions which were read out as follows: "You will be submitted to a laboratory examination with medical instruments. These instruments demonstrate the various functions of body organs as heart, eye, brain etc. During the test you must try to avoid purposeless movements. You will hear a series of sounds without any special meaning through headphones. When you hear the sounds do not do anything but listen to them as quietly as possible". The experiment took place in a psychophysiology laboratory; the room and conditions were standardised.

Results

As stated in Method, the patients were divided in three electrodermal groups. Before treatment, the highest percentage of subjects belonged to the group of responders, but after the standard CPZ treatment a one way movement of patients towards groups of decreased electrodermal responding appeared. Responders decreased from 19 (76%) to nine (36%), fast habituators increased from two (8%) to four (16%) and non-responders increased from four (16%) to 12 (48%). Table I illustrates the transition of patients to different response categories as a function of time and medication.

After treatment the skin conductance level of subjects decreased significantly and psychopathology improved significantly. Prestimulus SCL was 25.03 μmhos (s.d. = 16.37) before treatment and 17.49 μmhos (s.d. = 13.15) after treatment (paired t-test, $t = 3.39$, d.f. = 24, $P < 0.01$). Post-stimulus SCL values were 26.14 (s.d. = 18.31) and 18.37 (s.d. = 14.26) respectively (paired t-test, $t = 2.86$, d.f. = 24, $P < 0.01$). The mean BPRS score of subjects was 51.44 (s.d. = 6.98) before treatment, and 44.96 (s.d. = 9.31) after treatment; paired t-test value was 4.43, d.f. = 24, $P < 0.001$.

Before treatment, responders tended to show the higher level of autonomic arousal (as shown by skin conductance level), non-responders the lower one and fast habituators were at intermediate levels. The three groups did not differ significantly in this respect before the administration of chlorpromazine. However, the differences became significant after treatment (Table II).

No difference was observed in severity of symptoms among the three electrodermal groups. Before treatment the mean BPRS scores of non-responders was 53 (s.d. = 3.83), fast habituators 60 (s.d. = 0) and responders 50.21 (s.d. = 7.23). Values after treatment were 44.17 (s.d. = 11.08), 48.25 (s.d. = 7.80) and 44.56 (s.d. = 7.86) respectively.

Discussion

The question of high or low levels of schizophrenic physiological activity has been the subject of many discussions (Gruzelier & Venables, 1975; Tecce &

TABLE I

Transition of patients to different response categories after chlorpromazine administration

		Test 1 (before chlorpromazine treatment)			
		Response state			
		Non-responders	*Fast habituators*	*Responders*	*Total*
Test 2	Non-responders	4	1	7	12
(after chlorpromazine	Fast habituators	—	1	3	4
treatment)	Responders	—	—	9	9
	Total	4	2	19	25

TABLE II

Mean values for skin conductance level of schizophrenic electrodermal subgroups before and after treatment with chlorpromazine

				ANOVA		
	Non-responders	*Fast habituators*	*Responders*	*F*	*d.f.*	*Significance*
Before treatment	$n=4$	$n=2$	$n=19$			
Prestim. SCL	12.05 (±7.49)	23.63 (±11.49)	27.93 (±17.14)	1.65	2/22	NS
Poststim. SCL	12.31 (±9.68)	21.03 (±16.64)	29.59 (±18.93)	1.65	2/22	NS
After treatment	$n=12$	$n=4$	$n=9$			
Prestim. SCL	8.15 (±7.28)	15.17 (±7.08)	30.98 (±9.41)	20.67	2/22	$P<0.01$
Poststim. SCL	8.50 (±8.59)	16.82 (±11.07)	32.23 (±9.96)	16.21	2/22	$P<0.01$

Cole, 1972). Gruzelier & Venables (1972, 1973) suggested a bimodal distribution of arousal in schizophrenics (hypo-hyperaroused/non-hyper-responders). Venables later (1978) added a third group, the fast habituators group, and insisted on the high percentages of non-responders in schizophrenia which he regarded as a real schizophrenic phenomenon and not as an artifact produced by psychotropic drugs (Patterson & Venables, 1978; Venables, 1978).

The findings of this study support the view that the high percentage of non-responders in schizophrenia is a function of medication rather than a real schizophrenic phenomenon. After the standard chlorpromazine treatment, a transition of patients to lower response categories was observed and responders decreased twice while non-responders increased three times. Apart from drugs, a number of other intervening factors have been suggested as an explanation of this phenomenon: the type of stimuli used, the composition of experimental groups, the chronicity of the illness, the different experimental methods (Flekkøy, 1975), the sensitivity of recording method and responding criterion (O'Gorman, 1978), the size and position of electrodes, and the electrolytic medium. The majority of these factors are excluded in our experimental design (by the use of standard diagnostic criteria, same subjects and same experimental methods in both measurements, etc.). A factor of probable error not excluded in this study is that the second measurement of each subject, after chlorpromazine treatment, coincided with the repetition of the experiment. However, there are other studies that exclude such a factor by alternating experimental designs or placebo (Spohn *et al*, 1971; Kugler & Gruzelier, 1980). These studies present data showing influence of phenothiazines on the number of electrodermal responses that are in agreement with our findings.

After chlorpromazine treatment a decrease of autonomic arousal and an improvement of psychopathology of schizophrenics was observed. It is known that chlorpromazine has both central and peripheral effects. Kornetsky & Mirsky (1966) support the view that improved behaviour of schizophrenics after drug treatment is relative to decrease of hyperarousal. It is also noticeable that before treatment responders tended to be more aroused than non-responders and fast habituators tended to have an intermediate position. Those differences became statistically significant only after treatment. Such a finding may lead to the interpretation that different electrodermal groups express different levels of autonomic arousal (Gruzelier & Venables, 1972; Patterson & Venables, 1978). Another possibility, which might better explain our results, is that electrodermal responses do not have any relation to levels of arousal but that a relation does appear if patients take drugs and in particular chlorpromazine (Spohn *et al*, 1971). However, it should be noted that some groups have a small number of subjects and, statistically, this is a shortcoming for an ANOVA analysis.

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References

FLEKKØY, K. (1975) Psychophysiological and neurophysiological aspects of schizophrenia. *Acta Psychiatrica Scandinavica*, **51**, 234–248.

FRITH, C. D., STEVENS, M., JOHNSTONE, E. C. & CROW, T. J. (1979) Skin conductance responsivity during acute episodes of schizophrenia as a predictor of symptomatic improvement. *Psychological Medicine*, 9, 101–106.

GRUZELIER, J. H. & VENABLES, P. H. (1972) Skin conductance orienting activity in a heterogeneous sample of schizophrenics. *Journal of Nervous and Mental Disease*, **155**, 277–287.

—— & —— (1973) Skin conductance responses to tones with and without attentional significance in schizophrenic and non-schizophrenic psychotic patients. *Neuropsychologia*, **11**, 221–230.

—— & —— (1975) Evidence of high and low levels of physiological arousal in schizophrenia. *Psychophysiology*, **12**, 66–73.

Houlihan, J. P. (1977) Heterogeneity among schizophrenic patients: Selective review of recent findings (1970–75). *Schizophrenia Bulletin*, **3**, 246–258.

Kornetsky, C. & Mirsky, A. F. (1966) On certain psychopharmacological and physiological differences between schizophrenics and normal persons. *Psychopharmacologia*, **8**, 309–318.

Kugler, B. T. & Gruzelier, J. H. (1980) The influence of chlorpromazine and amylobarbitone on recovery limb of the electrodermal response. *Psychiatry Research*, **2**, 75–84.

Lader, M. (1975) *The Psychophysiology of Mental Illness* ed. T. Silverstone. London: Routledge & Kegan Paul.

O'Gorman, J. G. (1978) Method of recording: A neglected factor in the controversy over the bimodality of electrodermal responsiveness in schizophrenic samples. *Schizophrenia Bulletin*, **4**, 150–152.

Patterson, T. & Venables, P. H. (1978) Bilateral skin conductance and skin potential in schizophrenics and normal subjects. The identification of the fast habituator group of schizophrenics. *Psychophysiology*, **15**, 556.

Spohn, H. E., Thetford, P. E. & Cancro, R. (1971) The effects of phenothiazine medication on skin conductance and heart rate in schizophrenic patients. *Journal of Nervous and Mental Disease*, **152**, 129–139.

Straube, E. R. (1979) On the meaning of electrodermal nonresponding in schizophrenia. *Journal of Nervous and Mental Disease*, **167**, 601–611.

Tecce, J. J. & Cole, J. O. (1972) Psychophysiologic responses of schizophrenics to drugs. *Psychopharmacologia*, **24**, 159–200.

Venables, P. H. (1978) The psychophysiology of schizophrenia. In *Current Themes in Psychiatry*, eds R. N. Gaind & B. L. Hudson. London: Macmillan.

—— & Patterson, T. (1976) *Speech Perception and Decision Processes to Skin Conductance and Pupillographic Measures in Schizophrenia*. Paper presented to Scottish Rite Schizophrenia Research Program Conference on Attention and Information Processing in Schizophrenia. Rochester, May 1976.

Zahn, T. P. (1976) On the bimodality of the distribution of electrodermal orienting responses in schizophrenic patients. *Journal of Nervous and Mental Diseases*, **162**, 195–199.

—— (1978) Sensitivity of measurement and electrodermal nonresponding in schizophrenic and normal subjects. *Schizophrenia Bulletin*, **4**, 153.

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Abnormal Intestinal Permeability

An Aetiological Factor in Chronic Psychiatric Disorders?

Abnormal intestinal absorption has been suggested as an aetiological factor in schizophrenia. A procedure for investigating intestinal permeability was carried out in a group of chronic psychiatric in-patients. A proportion of the subjects studied showed abnormal intestinal permeability which could not be attributed to established bowel disease. Patients who were receiving neuroleptic but not anticholinergic drugs were those most often showing abnormal intestinal permeability. This work is at an early stage of development but preliminary findings suggest that further investigations should be carried out to establish the circumstances in which changes in intestinal permeability may be associated with mental illness.

The relationship between schizophrenia and small intestinal mucosal abnormality is controversial (*Lancet*, 1983). Increased titres of circulating antibodies to dietary antigens have been demonstrated in patients with schizophrenia, suggesting increased antigen absorption from the gut (Sugarman *et al*, 1982; Hekkens *et al*, 1980), and the prevalence of coeliac disease is increased in schizophrenics (Dohan, 1970). Coeliac disease is also characterised by high titres of antibody to dietary antigens (Kivel *et al*, 1964), which has been ascribed to the abnormal intestinal permeability found in untreated coeliac disease. Following treatment of coeliac disease with a gluten-free diet, intestinal permeability returns to normal (Hamilton *et al*, 1982), and, in some patients, dietary gluten withdrawal has led to an improvement in the psychiatric symptoms of schizophrenia (Dohan & Grasberger, 1973; Singh & Kay, 1976), although this beneficial effect of such dietary manipulation has not been confirmed by others (Storms *et al*, 1982).

The cellobiose/mannitol test has been shown to be a reliable index of abnormal intestinal permeability in coeliac disease (Cobden *et al*, 1980), when the urinary recovery of mannitol after oral administration is decreased while recovery of collobiose is increased. Expression of the result as a ratio of urinary collobiose recovery: urinary mannitol recovery minimises the effect of extraneous factors such as gastro-intestinal transit times and renal function on the test result (Cobden *et al*, 1985), and provides good separation between normal subjects and patients with coeliac disease (Cobden *et al*, 1980). We have used this test to investigate a population of long-stay psychiatric in-patients initially diagnosed as having schizophrenia to determine whether intestinal permeability is abnormal in such patients, and have further studied a proportion of these patients to determine whether abnormalities of permeability are due to co-existing coeliac disease.

Method

Patients

Thirty-two patients in long-term residential care were studied. All had been initially diagnosed as schizophrenic, often many years before, and were interviewed at the time of study by a psychiatrist who reclassified them as having schizophrenia or non-schizophrenic psychiatric disease according to Feighner *et al*'s criteria (1972). Age, sex, duration of symptoms and admission and details of all drug treatment were recorded. All patients performed a cellobiose/mannitol test. Patients fasted overnight provided a baseline urine sample and then ingested a solution containing 2 g mannitol and 5 g cellobiose in 100 ml water, with lactose and sucrose added as osmotic fillers. Patients continued to fast over the next 5 h, during which time all urine passed was collected and cellobiose and mannitol were assayed in the 5 h urine collection as previously described (Cobden *et al*, 1980). Recovery of both cellobiose and mannitol were expressed as a percentage of the administered dose, and the collobiose/mannitol recovery ratio, the percentage recovery of cellobiose:percentage recovery of mannitol, was compared with the established normal range (Cobden *et al*, 1980).

Subsequently, 10 patients in whom the ratio was within the normal range and 10 in whom the ratio was abnormal were selected for further investigation. Each of these 20 patients underwent endoscopy and distal duodenal biopsy (using an Olympus GIFP endoscope) and metabolism and excretion of cellobiose and mannitol were studied. Each patient received an intravenous injection of 500 mg mannitol and 50 mg cellobiose, and urine was collected for 5 h. Cellobiose and mannitol were assayed as for the oral test, and urinary recovery of each expressed as a percentage of the administered dose.

This study was approved by the Hospital Medical Ethical Committee and all patients consented to inclusion.

Results

Seventeen patients were classified as having schizophrenia whilst the remaining 15 were considered to have non-schizophrenic psychiatric disease.

Intestinal permeability in patients with psychiatric disease

The cellobiose/mannitol recovery ratio in the control population ranged from 0.002–0.03.

Of the 17 patients with schizophrenia, the cellobiose/mannitol ratio was normal in 11 (65%) and was abnormally high (range 0.032–0.46) in six while in the remaining 15 patients the ratio was normal in 10 (67%) and abnormal (range 0.032–0.072) in five. Overall, the ratio was abnormal in 11 of 32 (34%) long-stay psychiatric patients. Mean mannitol recovery was 8.4 (s.d. 7.1)% in patients with an abnormal ratio and 28.0 (s.d. 24.1)% in those with a normal ratio ($P<0.001$, Wilcoxon rank sum test), while mean cellobiose recovery was 0.42 (s.d. 0.3)% in patients with an abnormal ratio and 0.21 (s.d. 0.18)% in those with a normal ratio ($P<0.05$, Wilcoxon rank sum test). Thus the abnormal ratio in this group of patients was due to both a reduced mannitol absorption and an increased cellobiose absorption, the pattern found in coeliac disease (Cobden *et al*, 1980).

Patients in whom the cellobiose/mannitol ratio was abnormal did not differ from those with a normal ratio in respect of age, sex, duration of admission or length of illness, and there was no difference between groups in treatment with phenothiazines, butyrophenones or antidepressants. Only one of the 11 patients in whom cellobiose/mannitol ratio was abnormal required anticholinergic treatment, however, compared with 14 of the 18 with normal permeability who were taking phenothiazines ($P<0.002$). No patients were treated with non-steroidal anti-inflammatory drugs.

Recovery of cellobiose and mannitol after intravenous injection

Ten patients in whom the cellobiose/mannitol ratio was abnormal (six with schizophrenia and four with non-schizophrenic psychiatric disease) and 10 (six schizophrenic and four non-schizophrenic) with a normal ration, received intravenous cellobiose and mannitol.

In the patients with an abnormal ratio, mean urinary mannitol recovery was 45 (s.d. 18)% of the injected dose in 5 h, and mean cellobiose recovery 28 (s.d. 10.8)%, while in those with a normal ratio mean mannitol recovery was 53.9 (s.d. 30.3)% and mean cellobiose recovery was 29.3 (s.d. 17.7)%. Thus there was no significant difference between these two groups of patients in the metabolism or excretion of the two probe molecules after absorption.

Small intestinal histology

Each of the 20 patients receiving intravenous cellobiose and mannitol also underwent endoscopy and distal duodenal biopsy. Small intestinal mucosal morphology was normal in all patients. In particular, there was no evidence of coeliac disease or of infestation with Giardia lamblia.

Discussion

The abnormal pattern of urinary excretion of orally administered cellobiose and mannitol demonstrated in a large subgroup (34%) of long-stay psychiatric in-patients is a manifestation of abnormal intestinal absorption of these two probe molecules since there is no evidence that either probe molecule is abnormally metabolised or excreted after intravenous injection. While the recovery of each probe molecule after injection is lower than would be anticipated in normal controls, this reflects the difficulty in obtaining complete timed urine collections in the group of patients studied, and the effect of incomplete urinary collection is to reduce the recovery of each probe molecule similarly, without affecting the cellobiose/mannitol ratio (Cobden *et al*, 1985). The metabolism and excretion of both probe molecules are similar in patients studied with an abnormal cellobiose/mannitol ratio and those in whom the ratio was normal, and there is a strong and statistically significant correlation between the recovery of mannitol and that of cellobiose after intravenous injection in both groups ($r=0.74$, $P<0.01$ for patients with an abnormal ratio, $r=0.85$, $P<0.005$ for those with normal ratio) supporting the suggestion that the low recovery is due to incomplete urine collection.

The mechanisms of abnormal intestinal permeability in psychiatric patients has not been established. While the number of patients studied is not sufficient to establish the prevalence of coeliac disease in such patients, we have failed to identify any patient with histological evidence of coeliac disease, even in the group with abnormal intestinal permeability, and there was no evidence of giardiasis. The only difference in drug ingestion identified between patients with abnormal permeability and the remainder was the reduced necessity for anticholinergics in the former group, and this difference seems unlikely to account for the observed permeability abnormalities.

Increased intestinal permeability to large molecules relative to small ones may be of relevance to the pathogenesis of chronic psychiatric disease. The findings of this study are consistent with the hypothesis that a proportion of patients with chronic psychiatric illness, including schizophrenia, have abnormal small intestinal mucosal permeability, resulting in an increased absorption of endorphin-like molecules from the intestine, which induce behavioural abnormalities, but which also protect against the development of parkinsonian side-effects of phenothiazine treatment. We believe that further investigation of the relationship between psychiatric disease and small intestinal function is indicated.

References

Cobden, I., Rothwell, J. & Axon, A. T. R. (1980) Intestinal permeability and screening tests for coeliac disease. *Gut*, 21, 512–518.

——, Hamilton, I., Rothwell, J. & Axon, A. T. R. (1985) The cellobiose/mannitol test: physiological properties of probe molecules and influence of extraneous factors. *Clinical Chimica Acta*, 148, 53–62.

Dohan, F. L. (1970) Coeliac disease and schizophrenia. *The Lancet*, i, 897–898.

——, & Grasberger, J. L. (1973) Relapsed schizophrenics; earlier discharge from hospital after cereal-free and milk-free diet. *American Journal of Psychiatry*, 130, 685–688.

Feighner, J. P., Robins, E., Guze, S. R. B., Woodruff, R. A., Winokur, G. & Munoz, R. (1972) Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, 26, 57–63.

Hamilton, I., Cobden, I., Rothwell, J. & Axon, A. T. R. (1982) Intestinal permeability in coeliac disease. The response to treatment and single dose gluten challenge. *Gut*, 23, 202–210.

Hekkens, WTh. J. M., Schipperijn, A. J. M. & Freed, D. L. J. (1980) Antibodies to wheat proteins in schizophrenia: relationship or coincidence? *Biochemistry of Schizophrenia and Addiction*, ed. G. Hemmings. Lancaster, MTP Press.

Kivel, R. M., Kearns, D. H. & Liebowitz, D. (1964) Significance of antibodies to dietary proteins in the sera of patients with non-tropical sprue. *New England Journal of Medicine*, 271, 761–772.

Lancet (1983) Gluten in schizophrenia. *The Lancet*, i, 744–745.

Singh, M. M., Kay, S. R. (1976) Wheat gluten as a pathogenic factor in schizophrenia. *Science*, 191, 190–191.

Storms, L. H., Clopton, J. M., Wright, C. (1982) Effects of gluten on schizophrenics. *Archives of General Psychiatry*, 39, 323–327.

Sugarman, A. A., Southern, D. L., Curran, J. E. (1982) A study of antibody levels in alcoholic, depressive and schizophrenic patients. *Annals of Allergy*, 48, 166–171.

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Infantile Autism in Children of Immigrant Parents

A Population-based Study from Göteborg, Sweden

A population-based study of infantile autism from western Sweden has been completed. Urban children with autism more often than age-matched children in the general population had immigrant parents from 'exotic' countries. No such trend was seen in rural children with infantile autism.

In 1979, Lena Andersson (personal communication) suggested that infantile autism (IA) might be more common in children born to immigrant parents than in children born to native parents. In 1981, she published some data from an epidemiological study of autism in southern Sweden, which tended to support her hypothesis (Andersson & Wadensjö, 1981). However, in a contemporary study of autism from western Sweden no similar trend was seen (Gillberg, 1984).

Wing (1980, 1982) also has raised the possibility that infantile autism might be more common in children born to immigrant parents. She has also hypothesised that this increase in frequency, e.g. in children born to West-Indian mothers, could be due to organic factors, such as maternal rubella infection.

Akinsola & Fryers (1986) examined all severely mentally handicapped children aged 5–19 years in the city of Manchester, UK and found an increase of 'social impairments' among children of immigrant parents.

The present study reports data as regards parental country of origin from a population-based screening of children with infantile autism.

Method

Subjects

Twenty children under age 10 years with IA (17 boys and three girls) (Rutter, 1978; American Psychiatric Association, 1980) were included in the study. These children had been detected in a screening study of IA and related conditions in Göteborg (a city of about 450 000 inhabitants) (city IA group). Fifteen further children (13 boys and two girls) with IA from the adjacent county of Bohuslän (only rural and small-town areas of about 250 000 inhabitants altogether (rural IA group) were also included. The details of the epidemiological study have been published previously (Steffenburg & Gillberg, 1986). In summary, all doctors, psychologists, social workers and teachers likely to come across autistic children completed questionnaires stating whether they knew of any autistic or autistic-like children. The 35 children detected, constituted all the autistic children living in the areas on December 31 1984, and correspond to 4.5/10 000 children in the general population (4.7/10 000 in the city and 4.2/10 000 in the rural area).

All children had been diagnosed by the same child psychiatrist as suffering from IA in accordance with the criteria of the DSM-III (APA, 1980). All the parents of the 35 children were interviewed as regards their own country of origin.

Comparison group

The numbers of children under age 10 years born to immigrant parents of various origins and living in Göteborg on December 31 1984 were detailed from the registers of the Swedish Central Bureau of Statistics. Further, the corresponding number of children under 10 years of age born to all Swedish parents was noted.

Statistical tests

The χ^2 test with Yates' correction was used for comparisons of group frequencies.

Results

Six of the 20 (30%) city children (all boys) with IA were born to immigrant parents. The parents had been born in the republic of China (1), Uruguay (1), Hungary (1), Turkey (1) and Poland (2). In all cases but one (the boy whose father was Hungarian), both parents were immigrants from that country. Two of the boys (with parents from Hungary and Turkey) were born in Sweden. The remaining four had been born in their parental country of origin.

None of the 15 rural children with IA had immigrant parents.

Altogether 25.7% of the comparison group (11 022 out of 42 886) had at least one parent who had been born abroad. The majority of immigrant parents came from Finland. The difference between the city IA group and the comparison group (30% vs 25.7%) was not statistically significant.

Only 4.6% of the comparison group had at least one parent who had been born in any one of the 'immigrant countries' of the city IA group. The difference between these two groups is statistically significant at the 0.001 level. The difference between the rural IA group and the comparison group (0% vs 4.6%) was not statistically significant.

In the rural IA group there were significantly fewer immigrant parents than in the comparison group (0% vs 25.7%, $P<0.001$).

Four of the six IA city children with immigrant parents were severely mentally retarded and belonged in the most handicapped group of IA children in the whole material. No specific aetiology common to these cases could be found.

Discussion

From these results it appears that 'exotic' immigrant parents (that is parents from other than neighbouring foreign countries) might be more common in city children with infantile autism.

The reason why we did not find a corresponding increase in our previous epidemiological study of urban autism (Gillberg & Schaumann, 1982) is possibly that at the time of that study immigrants from such countries were very few indeed in Göteborg.

In the rural areas we did not find any immigrant parents at all, the main reason for this being that immigrants are extremely rare in these rural parts of Sweden.

The findings agree with those obtained by Andersson & Wadensjö (1981) in the late 1970s in Malmöhus län in southern Sweden. Malmöhus län was possibly already at that time more of an immigrant area than Göteborg. The findings are also in accord with those of Wing in London.

The reason for our findings can only be speculative since we have no hard data to uphold any specific view. Organic factors (such as intrauterine malnutrition, infections etc.) might be important. Genetic factors with or without the combination of a bilingual family problem (that is the vulnerable child in a 'language confused' state) might also be important - or, of course, other factors, that we have no idea of at the present stage, might be important.

During 1985, that is the year after our screening had been completed, seven further children with infantile autism born in 1978–1984 and living in either of the two areas studied, have been referred for diagnosis, work-up and treatment. Five of these children were born in 1983 and 1984 and had probably been missed in the screening because they were too young to have been clearly diagnosed or suspected of suffering from infantile autism. Two children had moved to the screening areas in 1985. Of these seven children no less than six have at least one immigrant parent. All of these children except one were born in Sweden and their parents had emigrated from Spain (1), Chile (1), Sri Lanka (1), The Philippines (1), England (1) and Finland (1). Again, this 'pattern' of immigrant parents in Sweden is unusual, and the frequency is much raised as compared with age-matched population samples.

On the basis of these and other recent data, we would urge other research groups to examine or re-examine their findings with a special view to disclosing possible increases in the frequency of immigrant parents in infantile autism.

References

Akinsola & Fryers (1986) A comparison of patterns of disability in severely mentally handicapped children of different ethnic origins. *Psychological Medicine*, **16**, 127–133.

American Psychiatric Association, DSM-III (1980) *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn; Washington DC: APA.

Andersson, L. & Wadensjö, K. (1981) *Early Childhood Psychoses in Malmöhus län - A Descriptive Study*. Research report. Social Welfare Authorities, County of Mamöhus län. (in Swedish).

Gillberg, C. (1984) Infantile autism and other childhood psychoses in a Swedish urban region. Epidemiological aspects. *Journal of Child Psychology and Psychiatry*, **25**, 35–43.

—— & Schaumann, H. (1982) Social class and infantile autism. *Journal of Autism and Developmental Disorders*, **12**, 223–228.

Rutter, M. (1978) Diagnosis and definition of childhood autism. *Journal of Autism and Childhood Schizophrenia*, **8**, 139–161.

Steffenburg, S. & Gillberg, C. (1986) Autism and autistic-like conditions in Swedish rural and urban areas: a population study. *British Journal of Psychiatry*, **149**, 81–87.

Wing, L. (1980) Childhood autism and social class: a question of selection. *British Journal of Psychiatry*, **137**, 410–417.

—— (1982) *Infantile Autism*. Paper read at seminar, University of Göteborg, Sweden.

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Psychiatric Vulnerability and Cerebellar Haemangioblastoma

A Case Report

A case of cerebellar haemangioblastoma in described in a middle-aged woman with a history of multiple psychiatric admissions for depressive and psychosomatic symptomatology. The diagnosis was not made until a late stage, largely because of the knowledge of her psychiatric history and its particular slant. Similar psychiatric symptoms were present at the outset, colouring the emerging neurological picture. The clinical manifestations and management of cerebellar haemangioblastoma are described.

Case report

Jane, 51, had no family history of psychiatric illness apart from her mother's suicide. She described her childhood as unhappy because her mother was too harsh. She left school at 13 and worked in factories until, at 26, she married a Post Office driver – a move her mother opposed. Her marriage was initially happy though later less so. She had the first of five miscarriages at the age of 31 and had not worked since. She always had few social contacts outside the home and was not on good terms with two siblings living locally. Jane described her life as a failure because she had produced no children. She had had extensive contact with gynaecologists and since the age of 12 had taken medication for intermittent asthma-like symptoms.

The year 1968, when Jane was 37, marked both her fourth miscarriage and her mother's suicide, and Jane said she never recovered from this latter blow. Shortly afterwards she had her first contact with psychiatrists when she claimed to have consumed bleach in a suicide attempt. In the following year she had numerous presentations with psychosomatic symptoms. She was considered to have an atypical depression (and low-normal IQ) and was given ECT during several admissions, without marked benefit. She had a further four admissions over the next 3 years and was usually given ECT. There were no further admissions over the next 8 years though her home life remained as restricted as before. A further cluster of admissions, usually of a few weeks duration, began in 1980. She complained of depression, suicidal thoughts, poor sleep and appetite ("one slice of toast a day for two years"), lack of support from her husband and of having no friends. On another admission she complained of tightness in her throat since a hysterectomy for menorrhagia a few months earlier. As before her symptoms were said to have a hypochondriacal slant and on one admission she was diagnosed as having a hysterical personality disorder. She tended to improve with in-patient support though staff often found her demanding and histrionic. In 1981 a further two admissions followed an overdose of anti-depressants and Jane complained that she was mad and should be put away. On this occasion ECT was said to have been effective. In July 1982, now 51, she presented to a casualty department with complaints of an inability to eat, drink, urinate, defaecate or breathe. She said that life was not worth living, that she was about to die and complained that her husband hit her. She was again treated with ECT, again without clear benefit.

In September 1982 she was again unable to cope at home, felt depressed and kept her curtains drawn all day in case the neighbours spied on her. One definitely new feature was the presence of involuntary movements, choreiform in nature, particularly in her face, left arm and legs. She had not been on psychotropic medication recently. These movements persisted after admission and she had a number of falls. After several weeks there was concern about memory and orientation though she was irritable and tended to co-operate poorly at interview. Her husband volunteered that her condition at that point seemed unlike all the previous episodes requiring in-hospital treatment. Transfer to a medical ward at the local General Hospital produced no new diagnostic insights though after her return she soon needed a Zimmer frame to walk. Later she became largely confined to bed. She was re-referred to the physicians and had a further 4-week stay in their care. They treated a chest infection but found no other focal pathology and the letter from the house physician which accompanied Jane back to the psychiatric ward noted her hypochondriacal style. On arrival she was found to be emaciated, slightly drowsy, irritable, disorientated and incontinent of urine. She had sluggish pupillary reflexes though no papilloedema, coarse nystagmus on left lateral glaze, involuntary movements of tongue, left side of face, left arm and leg, right facial and arm weakness with brisker reflexes, marked cogwheel rigidity and bilateral extensor plantars. She was close to death, 3½ months after admission.

Transfer to a specialist neurosurgery unit was immediately effected and an obstructive hydrocephalus was diagnosed on CT scan. A ventriculoperitoneal shunt was inserted and a further CT scan revealed a well-circumscribed mass in the left posterior fossa. A sub-occipital craniectomy allowed excision of a cerebellar tumour which proved to be a haemangioblastoma. Her post-operative recovery was fairly uneventful and she was left with a broad-based gait but minimal other cerebellar signs and was cognitively intact. She had a brief readmission for investigation of a pyrexia of unknown origin and the shunt was removed as a precaution.

More than 3 years later, now aged 54, there has been no evidence of a recurrence of tumour or of other pathology. Though for various reasons she has still not returned to the marital home, she has remained fairly stoical, though rather dependent, and has not complained of depression.

Discussion

One view of Jane's history is of a personality vulnerable to recurrent depressive and psychosomatic symptoms in the context of a life which she seems to have experienced from childhood as depriving and thwarting. Nonetheless it is noteworthy that her psychiatric history began only at 37, shortly after her mother's suicide, and it does seem that Jane struggled to free herself from the effects of this blow. In addition the successive disappointment of six miscarriages, an indifferent marriage and a lack of other social supports all took their toll. It was to psychiatrists she turned, frequently, and she seems to have been a rather trying patient to deal with. It is against a background of multiple psychiatric admissions, and of diagnostic tags like hypochondriasis and hysterical personality disorder, that the symptoms and signs of a posterior fossa tumour gradually emerged over several months, pursuing a largely classical course. That the diagnosis was not made until the patient was virtually moribund, despite several weeks on a medical ward, is a reminder of how potent a psychiatric history, particularly if it has a histrionic slant, can be in influencing the mental set of diagnosticians in other settings (Slater 1965). This effect may have been reinforced by the fact that she was already a psychiatric in-patient at the time.

Cerebellar haemangioblastoma is uncommon, representing 1–2% of all intracranial tumours. However, in adulthood, its incidence among primary tumours of the posterior fossa is 7.3% (Olivecrona, 1952) and it is thus nearly as common as meningiomas in this compartment. It tends to present in the 4th or 5th decade though at least a decade earlier in familial cases. It is composed of angioblasts which normally form foetal blood cells. It may exude xanthochromic fluid to form a cystic structure and the tumour may be a nodule in the wall of such a cyst. It is essentially non-infiltrating although malignant transformation and spread has occasionally been reported (Mohan *et al*, 1976). Almost all examples are subtentorial and cerebellar, usually single, although multiple tumours occur and in addition there can be similar tumours in the medulla or spinal cord (Morello & Bianchi, 1963).

Cerebellar haemangioblastoma can have prognostically important associations with other abnormalities, grouped as the Von Hippel-Lindau syndrome, though none have been diagnosed so far in the case above. The easiest to diagnose is a haemangioblastoma of the retina which is a small tumour situated peripherally in the retina and supplied by an enlarged artery and vein. Recognition may be hampered by secondary proliferative change or retinal detachment. Lindau's disease (Lindau, 1931) is the co-existence of other haemangioblastoma (e.g. spinal cord), cysts of pancreas or kidneys and carcinoma of kidneys. Phaeochromocytoma, sometimes bilateral, can emerge, particularly in certain families (Nibbelink *et al*, 1969). Polycythaemia occurs and may be the result of the elaboration of a factor stimulating erythropoiesis – such a factor has been found within the cyst fluid (Waldmann *et al*, 1961). Perhaps 10–20% of cases of cerebellar haemangioblastoma will ultimately produce evidence of some (usually not all) of these abnormalities though they are commoner when there is more than one tumour in cerebellum or spinal cord. In 20% of cases Lindau's disease has an autosomal dominant mode of inheritance, with variable expressivity. Nicol (1957) has reported five generations of one affected family. In most cases the cerebellar tumour is the source of initial symptoms.

Raised intracranial pressure is more marked and occurs earlier in tumours situated below the tentorium than those above though a cystic haemangioblastoma sometimes attains a large size without causing symptoms. Sub-occipital headache, clumsiness of the ipsilateral hand and a tendency to stagger towards the side of the lesion are common early symptoms. Nystagmus is marked and maximal with gaze towards the side of the lesion. The other signs of cerebellar dysfunction, particularly ataxia, will be most evident in the ipsilateral limbs. Speech is not commonly affected. Forward pressure by the tumour may impinge on cranial nerve function (particularly 5th, 6th, 7th) and pressure on pons and medulla can produce contralateral pyramidal tract signs. Tumour growth will produce obstructive hydrocephalus; papilloedema (though not present in this case) can be severe.

There was one other atypical feature. Involuntary movements are only unusually associated with cerebral tumours, particularly of the posterior fossa, and here they were present from the start. It is unclear just how they might arise in this context. However, it is worth noting that hysterical mechanisms have been considered capable of producing involuntary movements and it is certainly accepted that such mechanisms can be facilitated by underlying brain disease. More recently psychological explanations have been invoked less frequently than organic ones, not least because drugs like the phenothiazines (not implicated in the case above) have been observed to regularly produce just such movements (Merskey, 1986).

Mental disturbances are far less likely in subtentorial than supratentorial tumours and tend to emerge later than the neurological signs and symptoms (Hecaen & Ajuriaguerra, 1956). Cognitive disturbances, usually global, are generally related to the presence of raised intracranial pressure and develop insidiously. Here they were a late feature. Lability of mood, depression or irritability often accompany the advancing neurological picture though are unlikely to result specifically from organic mechanisms: emotional responses will be shaped by premorbid personality, vulnerability to psychiatric disorder and by the way doctors and nurses handle the patient. Moreover, a tumour arising 'de novo' may precipitate an episode of psychiatric breakdown whose characteristics, at least at the outset, may be little different from those of previous episodes. This patient's history suggests she was vulnerable to even modest stressors and she had already taken up familiar postures of despair and dependence at a point when the neurological picture was only just beginning to emerge.

It remains true that cerebellar haemangioblastoma invariably presents with neurological abnormalities rather than anything else and in Palmer's series (1972) this onset ranged from 3 weeks to 7 years (average 13 months) before diagnosis was made. Clearly this tumour can grow at very variable rates though, as we have seen, the interval between neurological onset and diagnosis may also reflect factors which emerge from the mental set of both patient and doctor. In the patient described above, neurological signs and symptoms, never noted during her many previous episodes of breakdown and in-hospital treatment, were present from the start of a progressive 4-month illness. It seems unlikely that a slow growing tumour could account for a much larger slice of her 14-year psychiatric history.

The most useful diagnostic aid is the CT scan, particularly in delineating the cystic component and associated hydrocephalus. Vertebral angiography may still have a role. Prognosis following resection is favourable and cure may result if resection is complete (Palmer, 1972). Recurrence may occur or haemangioblastoma at other sites may become symptomatic. The overall recurrence rate is said to be 33% after 4 years though higher (50%) in familial cases (Yamashita *et al*, 1982). A co-existing polycythaemia will be abolished by resection but can recur with renewed growth of tumour. The cerebellar haemangioblastoma itself remains the commonest cause of death. Renal cell carcinoma, arising later, may befall a patient with Lindau's disease who had successful initial treatment (Horton *et al*, 1976). Pre-symptomatic screening and genetic counselling have been recommended for families carrying Lindau's disease.

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References

Hecaen, H. & Ajuriaguerra, J. (1956) *Mental Disturbances in Intracranial Tumours*. Paris: Masson.

Horton, W., Wong, V. & Eldridge, R. (1976) Von Hippel Lindau's disease: clinical and pathological manifestations in families with 50 affected members. *Archives of Internal Medicine*, 136, 769–777.

Lindau, A. (1931) Vascular tumours of brain and spinal cord. *Proceedings of the Royal Society of Medicine*, 24, 363.

Merskey, H. (1986) The importance of hysteria. *British Journal of Psychiatry*, 149, 23–27.

Mohan, J., Brownell, B. B. & Oppenheimer, D. (1976) Malignant spread of haemangioblastoma – 2 cases. *Journal of Neurology, Neurosurgery and Psychiatry*, 39, 515.

Morello, G. & Bianchi, M. (1963) Cerebellar haemangioblastoma. *Journal of Neurosurgery*, 20, 254.

Nibbelink, D., Peters, B. & McCormick, W. (1969) Association of phaeochromocytoma and cerebellar haemangioblastoma. *Neurology*, 19, 455–460.

Nicol, A. (1957) Lindau's disease in five generations. *Annals of Human Genetics*, 22, 7.

Olivecrona, H. (1952) Cerebellar angioreticulomas. *Journal of Neurosurgery*, 9, 317.

Palmer, J. (1972) Review of 81 cases of haemangioblastoma. *Acta Neurochirurgica*, 27, 125–148.

Slater, E. (1965) Diagnosis of hysteria. *British Medical Journal*, 1, 1395–1399.

Waldmann, J., Levin, E. & Baldwin, M. (1961) The association of polycythaemia with cerebellar haemangioblastoma. *American Journal of Medicine*, 31, 318–324.

Yamashita, J., Handa, H., Kim, C. & Kim, S. (1982) Familial occurrence of cerebellar haemangioblastoma – analysis of five families. *Neurosurgery*, 11, 761–763.

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Schizophreniform Episode Following Measles Infection

A case of schizophreniform episode following measles infection in a 27-year-old woman is presented. The possibility that the clinical picture is a first description in the literature of an association between a schizophreniform episode and possible measles-induced allergic encephalitis is discussed. Other possible connections between measles infection and schizophreniform episode are also mentioned.

Acute measles encephalitis is distinct from two rare neurological complications of measles; subacute inclusion body encephalitis in immunodeficient or non-immunodeficient patients and subacute sclerosing panencephalitis (SSPE) (Kipps *et al*, 1983). The pathogenesis of the acute post-infectious encephalitis seems to be of an autoimmune nature. This is supported by the abrupt onset of the encephalitis late in the course of the exanthema or respiratory disease, the usual failure to detect virus in the brain, and the neuropathological changes which resemble those of experimental allergic encephalomyelitis (Johnson *et al*, 1984).

As there is no invasion of the central nervous system (CNS) by measles virus, it is possible to find normal protein, pressure and cell count in the cerebrospinal fluid (CSF) and no rise in measles antibody titer (Johnson *et al*, 1984). The clinical picture of such encephalitis usually involves severe neurological changes such as depressed level of consciousness, seizures, motor deficits and movement disorders (Johnson *et al*, 1984). The clinical picture of patients with brain lesions characterised by the presence of measles-like inclusive bodies is indistinguishable from these with the allergic type of brain cell damage (Kipps *et al*, 1983). To the best of our knowledge there is no description in the literature of schizophreniform episode as the only manifestation of measles post-infectious encephalitis. Therefore, we present for the first time, a case of post-infectious encephalitis presenting as a schizophreniform disorder.

Case report

Mrs S.H., a 27-year-old, woman of Jewish Ashkenazi origin was admitted to the general hospital because of behavioural changes which included agitated psychomotor activity, incoherent thinking, labile and inappropriate affect and poor judgement. She had a negative history for medical or psychiatric disease except for hormonal treatment for primary sterility for the last year. Her family history did not reveal any psychiatric disease. Ten days before her admission, the patient developed fever up to 40°C, maculopapular rash, respiratory symptoms and Koplik spots. She was diagnosed as having measles virus infection by her family doctor. She had suffered from the above behavioural changes since the eighth day and was admitted to the general hospital, was suspected of having measles encephalitis. Neurological examination and EEG were normal. Lumbar puncture technically failed. The mental status continued to deteriorate (bizarre delusions, frightening auditory hallucinations, extreme agitation and poor cooperation with the medical staff), and there was no change in her neurological status. The next day the patient was transferred to the Geha Psychiatric Hospital. On admission she was uncooperative and agitated. Her consciousness was clear with good orientation for time and place. Memory was undisturbed for all ranges. Her thinking was rapid, tangential and loosely associated, with delusions about her ability to hypnotise other people, read their thoughts, "turn them religious", and about having her thoughts read by others. The affect was euphoric and inappropriate. She had auditory hallucinations but could not describe them because "it was a secret". She lacked any insight and her judgement was poor.

Physical and neurological examinations were normal. Laboratory findings were as follows: leukocytes – 11 800 mm^3 with 60% lymphocytes; BSR – 80/110 mm which returned to normal within 2 days. Blood chemistry values and T4, vitamin B12, ANF, and LE cells were all within normal range. Measles antibody titer was 1:16–1:32, and progressed to 1:32–1:64, 6 days later. Cerebrospinal fluid findings were: clear fluid; pressure – 10 cm water; protein – 10 mg%; sugar – 66 mg%; cell count – 14/mm^3, with 98% monocytes and 2% polymorphonuclears. Bacterial and viral cultures were negative. Viral antibodies for measles, herpes simplex and rubella viruses were negative. On admission to the psychiatric department, and 4 days later, EEGs were normal as was CT scan.

Treatment was begun with levomepromazine up to 200 mg/day, and the psychosis gradually disappeared after 3 weeks. The patient was sent home in full remission, and on 8-month follow-up there were no psychiatric disturbances.

Discussion

Some CNS syndromes associated with measles virus are atypical and do not fit the classical description of either acute measles encephalitis or subacute SSPE (Kipps *et al*, 1983). We describe for the first time in the literature, a case of an association between a schizophreniform episode and possible measles-induced allergic encephalitic infection. This clinical picture developed post-measles infection and was constituted of agitated psychomotor activity, delusions, incoherent thinking, auditory hallucinations, labile and inappropriate affect and poor judgement and lacked any insight.

The anamnesis supported a measles virus infection with upper respiratory infection signs, maculopapular rash and Koplik spots. The clinical and laboratory findings of elevated BSR, leukocytosis with lymphocytic predominance, the increased measles antibody titre, and the most significant finding of 14 cells/mm^3 in CSF supported the diagnosis of measles encephalitis presented as a schizophreniform episode.

This case might be considered as an acute measles allergic encephalitis in which there was no evidence for direct invasion of the brain. Such encephalitis, which is considered allergic is essentially a demyelinating disorder which occurs primarily as a complication of measles, but also after vaccinia, varicella, and rubella infections. The lesions resemble those of neuroparalytic accidents following immunisation with rabies vaccine made from CNS grown virus and in experimental allergic encephalitis. This similarity suggests a common immunological reaction to some neural or vascular component resulting in demyelination, necrosis, vascular damage, and haemorrhage. Although viral inclusions are not found in this disorder and measles virus has not been isolated with certainty from the CSF or brain in such cases, the possible role of measles virus in the initiation of these changes cannot be excluded (Kipps *et al*, 1983). The case presented above can be included in group A described by Kipps *et al* (1983), which includes patients in whom neurological signs developed within 3–7 days of the onset of the measles rash, with short clinical illness lasting a week or less. This case is different from group A in that the virus was not isolated and there were no measles antibodies in the CSF, and it presented as a psychiatric disorder but not a neurological disorder. On two occasions the EEG of our patient was normal.

The incidence of encephalomyelitis post-measles infection is 1:1000. The encephalomyelitis is responsible for 25% of the neurological complications of post-measles infections. The mortality rate is 10–20%. Approximately 50% of the patients have severe persistent neurological defects, such as blindness, paraplegia and ataxia; 10% have seizures and many patients remain with mental deficiency. The prognosis is usually related to the duration of stupor or coma of the patient (Johnson *et al*, 1984). The proportion of cases presenting as a purely psychiatric disorder is unknown.

In such a discussion we must mention the viral hypothesis of schizophrenia, which may support the viral aetiology of the schizophreniform episode in this case report.

According to the developments in the field of virology and neurologic disease, there is preliminary evidence in support of the hypothesis that some individuals who are diagnosed as schizophrenics have a chronic brain disease, possibly acquired *in utero* or early infancy, the cause suspected as being a viral agent, according to findings in virology, clinical, epidemiological and laboratory spheres (Torrey & Peterson, 1976).

Common viruses including measles were suggested as possible aetiological factors of schizophrenia (Krauss, 1957; King *et al*, 1985). King *et al* inferred that an impaired immune response might be involved in schizophrenia. However, if the present case was an allergic encephalitis it would imply an augmentation of the immune response.

This case supports the concept that some schizophreniform-like disorders are associated with viral infection especially encephalitis (Wilson, 1976; Sobin & Ozer, 1966). Since the specific viral agent was not always identified, some of these cases might also have been due to measles.

References

Johnson, R., Griffin, D., Hirsch, R., Wolinsky, J., Roedenbeck, S., De Soriano, I. L. & Vaisberg, A. (1984) Measles encephalomyelitis – clinical and immunological studies. *New England Journal of Medicine*, 310, 137–141.

Kipps, A., Dick, G. & Moodie, J. (1983) Measles and the central nervous system. *Lancet*, ii, 1406–1410.

King, D. J., Cooper, S. J., Earle, J. A. P., Martin, S. J., McFerran, N. V. & Wisdom, G. B. (1985) Serum and CSF antibody titers to seven common viruses in schizophrenic patients. *British Journal of Psychiatry*, 147, 145–149.

Kruass, S. (1957) *Report of 2nd International Congress for Psychiatry*, Zurich, Vol. II, pp. 100–102.

Sobin, A. & Ozer, M. N. (1966) Mental disorders in acute encephalitis. *Journal of Mount Sinai Hospital*, 33, 73–82.

Torrey, E. F. & Peterson, M. R. (1976) The viral hypothesis of schizophrenia. *Schizophrenia Bulletin*, 2, 136–146.

Wilson, L. G. (1976) Viral encephalopathy mimicking functional psychosis. *American Journal of Psychiatry*, 133, 165–170.

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Clonidine

An Effective Anti-manic Agent?

Three patients suffering from bipolar affective disorder, manic phase, and who were resistant to conservative treatment were successfully treated with clonidine. This would support the nor-adrenergic hyperactivity theory of mania. The rapidity of action of clonidine and its lack of relatively serious side-effects renders it a potential alternative to conventional anti-manic agents.

According to the catecholamine theory of affective disorder, increased central nor-adrenergic transmission has been implicated in the genesis of mania (Schildkraut, 1978). Clonidine is a long-established anti-hypertensive agent with presynaptic agonistic effect resulting in inhibition of the neural release of nor-adrenaline. Side-effects during treatment such as depression and sedation are well documented and have resulted in suggestions for its use as both a possible anti-manic and anxiolytic agent (Hoehn-Sarie *et al*, 1981; Lloyd, 1985). Increasing reports in recent literature suggest that clonidine is a potential adjunct in the treatment of mania (Giannini *et al*, 1983; Zubenko *et al*, 1984). We report here our experience with clonidine in the treatment of three patients. They presented with mania and with a history of bi-polar affective disorder.

Case reports

A 29-year-old male with a negative family history of affective disorder was first diagnosed as a unipolar depression in 1979. He developed his first manic episode in 1982 and has subsequently had several admissions with both manic and depressive episodes. He was maintained on lithium carbonate 1200 mg per day since June 1983 with serum lithium levels consistently ranging around 0.8 to 0.9 mmol/litre. His mania was treated at varying stages with chlorpromazine, haloperidol and lithium with only transient effect. His depressive episodes since 1982 were always post-mania and of brief duration. His last admission was in September 1982 when he was again noted to be markedly loud and vociferous with pressure of speech, flight of ideas, elation and grandiose delusion. He failed initially to respond to haloperidol in doses of 60 mg per day combined with chlorpromazine in doses of 1200 mg per day and his mood began to cycle rapidly between mania and apathetic depressive state. Because of his minimal response he was treated after one month with lithium carbonate 1600 mg per day (serum level 0.9 to 1.14 mmol/litre) for a period of 8 weeks but his mood continued to cycle rapidly as before. He was then given, in addition to lithium carbonate, clonidine 0.1 mg *b.i.d.* because of reports of its anti-manic effect. At this time his score on the Bech-Rafaelsen (BR) mania rating scale was 29. Clonidine doses were increased to 0.2 mg *b.i.d.* within 2 days. Following the increase in the dose of clonidine his BR mania rating scale score dropped to 7. His blood pressure did not alter. He was maintained on clonidine for 3 weeks and it was then withdrawn gradually over one week. Subsequently he was maintained on lithium carbonate 1200 mg per day. He was discharged and has remained asymptomatic up to the time of this report.

A 60-year-old woman with a long history of manic-depressive illness, depressed type, presented with her first episode of mania in April, 1985. She had one previous admission for the treatment of depression in 1970 and was treated on an out-patient basis for several depressive relapses up to the time of her latest admission. Her Bech-Rafaelsen mania rating scale score was 32. She was commenced on chlorpromazine 400 mg daily and chlopenthixol 40 mg daily with no effect after 1 week. At this stage haloperidol 5 mg orally with an intravenous dose of 10 mg was added on a daily basis. She failed to respond for a further 1 week. Haloperidol and chlopenthixol were discontinued and she was commenced on clonidine 0.2 mg daily. She became much more settled within 48 h and was normothymic 4 days later. Her BR mania rating scale score dropped to 5 and she was subsequently discharged on clonidine 0.2 mg daily with thioridazene 100 mg daily and has remained asymptomatic up to the time of report.

A 38-year-old married male with a negative family history of affective disorder was first diagnosed as psychotic depression in October 1983 and subsequently had several admissions for depression and mania. In February, 1985 he was again admitted with another episode of mania. His Bech-Rafaelsen mania rating scale score was 31 at the time of admission. He was commenced on chlorpromazine 300 mg daily and 100 mg intramuscular every 6 h with no improvement. After 5 days, haloperidol 10 mg intravenous daily was added to the regimen. He failed to respond for a further 4 days. Haloperidol was discontinued and he was commenced on clonidine 0.1 mg *b.i.d.* He became much settled within 3 days. The dose of chlonidine was increased to 0.2 mg *b.i.d.* and chlorpromazine was reduced to 100 mg a day. He became normothymic within 1 week. His BR mania rating scale score dropped to 6 and he was subsequently discharged on a maintenance dose of clonidine 0.2 mg daily and chlorpromazine 100 mg daily. He has also remained asymptomatic up to the time of report.

Discussion

The three case reports suggest that clonidine may have a role in the treatment of mania and are consistent with previous reports. (Giannini *et al*, 1983; Zubenko *et al*, 1984). All the three cases responded rapidly following commencement of treatment with clonidine and without apparent sedative effect. It is also worth noting that no patient developed hypotension or depression. The specific anti-manic effect appears to support the noradrenergic hyperactivity theory of mania (Schildkraut, 1978). Reports of relapse into mania following withdrawal of clonidine would also support this theory (Zubenko *et al*, 1984). Furthermore, Farsang & Kunos (1979) observed that clonidine may act as an endorphinergic agent, particularly in view of the fact that its hypotensive effectives are antagonised by naloxone, as a consequence it has been suggested that clonidine may exert its antimanic effect by stimulating central endorphins. It would appear likely that it has a dual endorphinergic and noradrenergic antagonistic mechanism. While it is preemptive to suggest that clonidine should be used as a standard treatment in mania, our study would suggest that it is worth considering in refractory cases or in patients who cannot tolerate lithium. The associated risks of neuroleptics such as anticholinergic effects, extrapyramidal symptoms and the potential risk of tardive dyskinesia are markedly more serious than those attributed to clonidine such as sedation, hypotension and less commonly depression. Our findings are consistent with other preliminary reports and the possibility of clonidine being used in place of neuroleptics and as an adjunct or substitute for lithium warrant further research.

References

Farsang, C., Kunos, G. (1979) Naloxone reverses anti-hypertensive effect of clonidine. *British Journal of Pharmacology*, **67**, 161–164.

Giannini, A. J., Exetein, I., Gold, M. S., Pottash, A. L. C. & Castellani, S. (1983) Clonidine in mania, Drug development research. 3, 101–103.

Hoehn-Sarie, R., Merchant, A. F., Keyser, M. L. & Smyth, V. K. (1981) Effects of clonidine on anxiety disorders. *Archives of General Psychiatry*, **38**, 1278–1282.

Lloyd, G. F. (1985) Emotional aspects of physical illness. In *Recent Advances in Clinical Psychiatry*, ed. K. Granville-Grossman. Edinburgh: Churchill Livingstone.

Schildkraut, J. J. (1978) Current status of the catecholamine hypothesis of affective disorders. In *Psychopharmacology: A Generation of Progress*, ed. M. A. Litpon, A. Di Mascio & R. F. Killam. New York: Raven Press.

Zubenko, G. S., Cohen, B. M., Lipinski, J. F. & Jones, J. M. (1984) Clonidine in the treatment of mania and mixed bi-polar disorder. *American Journal of Psychiatry*, **141**, 1617–1618.

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The Role of Food Substitutes in a Token Economy System

Data from five behaviour management (token economy) units were analysed for periods of 10–20 weeks before and after the use of a food substitute (Complan®), provided when insufficient tokens had been earned to buy a full- or part-meal. The efficacy of this form of negative punishment to control disturbed and aggressive behaviour was examined. No evidence was found to suggest that the contingent use of a food substitute made any contribution to the therapeutic process.

It was demonstrated in the early 1960s that food could function as a powerful reinforcer of desirable behaviours among patients with psychotic disturbances (Ayllon, 1963; Ayllon & Haughton, 1962). Consequently, several early Token Economy Programmes (TEPs) utilised meals as one form of token-contingent reward. The techniques of behaviour modification have been a cause of ethical concern to several authorities (Wexler, 1973, 1975; Stolz, 1976; Kazdin, 1977; Innis, 1981). To support these forms of negative punishment as useful clinical procedures we need more evidence that to facilitate behavioural change, it is necessary to restrict access to certain basic amenities (Higson *et al*, 1985).

Behaviour management techniques can be divided into four basic conditions. Firstly, to increase behaviour one can (a) present something desirable (positive reinforcement) or (b) end something undesirable (negative reinforcement). Secondly, to decrease behaviour one can (c) present something undesirable (positive punishment) or (d) withhold something desirable (negative punishment). Withholding meals in lieu of the required tokens falls into this last category of negative punishment.

Higson *et al* (1985) investigated the effects of introducing meals as a 'free' item in the treatment of nine female chronic schizophrenic patients. The meals had previously been contingent upon the patients achieving a number of target behaviours in the area of self-help skills and social activities. Contrary to expectations, they found no systematic effects of meals on patients' performance on these specific target behaviours.

Two reservations may be expressed over the Higson study, however. The first is that subjects were all chronic schizophrenics. No attempt was made to investigate the effectiveness of food as a reinforcer with other types of patient treated in behaviour modification settings (e.g. those with conduct disorders, physical aggression and 'acting out'). Secondly, 'other consumables' (e.g. sweets) were available to these patients even when a meal was unavailable and food substitutes were not used.

The use of solid food as a method of reinforcement was a major feature of a behaviour management procedure at St Andrew's Hospital until March 1985. St Andrew's Hospital accepts very seriously disturbed patients for treatment in a controlled and locked setting using behaviour as a management structure (see Moyes *et al*, 1985). The practice of this negative punishment procedure was limited by a '15% rule', whereby staff were not to deny patients – or, in other words, patients were not permitted to lose – more than 15% of their meals per month. Where insufficient tokens were held for purchasing either a 'full' or 'part' meal, a milk-based nutritious substitute (Complan) was offered. The intention of this procedure was not to starve an individual patient but to create an attitude that meals, like many other things that some people take for granted, are something to be earned or worked for by a display of good, cooperative behaviour.

The need for patients to *earn* a meal has always been seen by some as a contentious ethical issue. On the other hand, staff of various disciplines concerned with the residents behavioural unit felt that the use of solid food as a back-up reinforcer was not only ethically justified but an essential part of the therapeutic regime. The discontinuation of this procedure in early 1985 made it possible to evaluate the efficacy of it retrospectively. Two main questions were addressed. First, the pragmatic issue of whether or not the use of a food substitute served its purpose of motivating individuals to work towards their target behaviours, thereby making the use of food effective as a reinforcer. Secondly, there was the ethical issue of whether one is justified, in the long-term interests of the patient, to 'deprive' patients of solid food.

Method

Data from the five behavioural units within the hospital operating TEPs were used. Unit 1 accommodates young people between the ages of 15 to 25 with behaviour disorders, some of which are secondary to psychiatric illness. Most of those who are admitted are detained under Section 3 of the Mental Health Act or a Secure Care Order and have usually proved unacceptable for placement in other establishments. Unit 2 takes similarly aged, behaviourally disordered youngsters who are at a lower level of functional and social skills. These patients have a mild-to-moderate degree of mental impairment and are likely to be always in need of some minimal community care. Unit 3 accommodates adults presenting a disturbed behaviour pattern, either as part of a psychotic illness or a personality disorder. The majority are referred from the courts either on remand or on Sections 37 or 41 of the Mental Health Act. Unit 4 caters for a wide age-range of behaviourally disordered mentally handicapped individuals, while Unit 5 is a rehabilitation unit for adults who have sustained brain injury (usually in road traffic accidents) and whose behavioural sequelae generally preclude their acceptance for rehabilitation elsewhere. All units are secure and all are of mixed sex. With the exception of the Unit 5, they contain a rather heterogenous group of patients.

The 29 patients studied (20 males, nine females) had a mean IQ of 73 (s.d. = 19) and a mean length of stay of 22 months (s.d. = 12). The number of patients in Units 1, 2, 3, 4, and 5 were 7, 7, 3, 6, and 6, respectively. By diagnostic category, three patients were behaviourally disordered with schizophrenia, 12 were behaviourally disordered with mental handicap (IQ<70), six were behaviourally disordered with head injury, and eight demonstrated other behaviour disorders (hysteria, personality disorders, etc.)

The various units terminated the use of Complan as a food substitute at different dates within a 4-month period. Only patients who had been on their respective behavioural units for 10–20 weeks (X = 18.55; s.d. = 2.698) before and 10–20 weeks (X = 15.53; s.d. = 3.100) after Complan was stopped were included in this study (n = 29). Patients transferred from one unit to another within the hospital during this period were not included. A period of 15 weeks before and after discontinuing Complan yields data from over 600 meals per patient. Due to the fact that a change in hospital policy meant that Complan could not be reintroduced, the experimental design was not amenable to anything other than a 'before and after' paradigm.

Procedure

It was a hospital requirement that all incidents where Complan (or part-meals) were given, be recorded by staff in a book specifically for this purpose and which is countersigned at least once per week by the psychiatrist or psychologist in charge of the treatment. In addition, all failures to earn tokens are recorded on the individual patients' charts. Cumulative token earnings are summated every week and documented as a percentage of possible earnings. Such data is therefore readily available for subsequent analysis. These percentages are quantitative reflections of a patient's level of general behaviour. Similarly, acts of aggression, leading to 5 minutes in a Time-Out Room are recorded by staff, together with dates, times and a description of the behaviour. There is, therefore, a clear record of incidents of actual physical aggression and other behavioural disturbances of a violent kind.

If a 'square meal' can be used to motivate and/or reinforce desirable behaviour, then we can evaluate the effectiveness of a food substitute as a form of negative punishment within a behavioural system by hypothesising that after food ceased being a contingent reinforcer, behavioural indices would alter to reflect this, probably by showing an escalation of the frequency of inappropriate behaviour. Specifically, token earnings would be expected to decrease while Time-Out Room incidents for aggression would increase for a period after the withdrawal of contingent food reinforcement compared with the period immediately before.

Results

Token earnings

Analysis (one-tailed t-tests) of mean weekly token earning percentages for the five units before and after termination of the use of Complan revealed that only two of the five units (Units 4 and 5) showed a trend in the expected direction. In addition, the only significant difference was in Unit 2 and that was in the direction opposite to that predicted ($t=1.96$; d.f. $=6$; $P<0.05$).

Analysing patients by category (behaviour disorder with mental handicap, behaviour disorder with schizophrenia, other behaviour disorders) as opposed to analysing by units, reveals that only those with a degree of mental handicap showed a significant difference although, again, in the direction opposite to that predicted ($t=-1.847$; d.f. $=11$; $P<0.05$).

Time-Out Room incidents

The differences in mean weekly Time-Out Room recordings for units before and after termination of food as a reinforcer, shows that only in Unit 1 was a significant difference found ($t=1.995$; d.f. $=6$; $P<0.05$). Once more, however, this was not in the direction predicted. Analysing differences in mean weekly Time-Out recordings by patient category as opposed to units, fails to show any significant changes.

Discussion

It was hypothesised that if the use of a food substitute (Complan) in lieu of adequate tokens for a 'full-' or 'part-meal' was an effective component in the behavioural system, then this would have been reflected by changes in behavioural indices before and after its cessation. In particular, it would be expected that some behaviours (Time-Out Room incidents) would increase, while positive aspects of behaviour (weekly percentage token earnings) would decrease.

The analysis of results by units and by patient categories, showed that few significant differences emerged. These were found to be quite dissimilar to those predicted – suggesting that using food as a privilege to be earned might be associated with a slightly punitive social milieu which reduces patient motivation and co-operation. It is clear that the use of food substitute *per se* did not have a substantial role in creating behavioural change. This is in contrast with beliefs held by many staff members of the hospital under study.

Behaviour modification arose from the study of animal behaviour in controlled settings. The evidence from both the theory and practice of behavioural approaches clearly indicates that private events play an important role in the determination of human behaviour (Lowe & Higson, 1983). In a social context the patient is systematically provided with information concerning his actions and their likely outcome for himself and others. Behaviour thus comes about through an informed appraisal of social consequences rather than via a hypothetical conditioning process or through an attempt to regain pleasures and privileges which are the patient's by right. ". . . We can not continue to defend coercive systems by reference to their results since we have clearly demonstrated that programmes which involve no deprivation of basic rights and privileges achieve comparable results to those achieved in token economies. In treatment of any kind the procedures that cause least distress to the patient should always take priority".

Hall & Baker (1986) summarise four major contributions of behavioural methods to present-day psychiatric rehabilitation: firstly, an increased emphasis on careful and comprehensive assessment of the patient and his environment, using behavioural methods and relating to regular monitoring procedures; secondly, a broadening range of treatment procedures which *may* use individual or group token programmes; thirdly, a clearer formulation of patient–staff and patient–patient interactions for ward milieux; and fourthly, better guidance on the design of both patient treatment programmes and staff training programmes so that positive results of the programme generalise to the real world – "although the token economy itself, in its original 'pure' form, has fallen into abeyance".

One does not necessarily have to agree with Hall & Baker. It would be of interest to examine various other aspects of the reinforcement system, e.g. social praise, contact with staff, cigarettes for smokers, leisure time etc., in an attempt to tease out which particular components can, in themselves, demonstrate changes in behaviour. There seems little doubt that the system *in toto* modifies behaviour and that changes so gained are maintained at follow-up (see Moyes *et al*, 1985). It may be that *no one item alone* contains that essential quality for change. Rather, when various components are combined together in a 'behavioural package', the sum is greater than the total of the parts.

References

Ayllon, T. (1963) Intensive treatment of psychotic behavior by stimulus satiation and food reinforcement. *Behaviour Research and Therapy*, 1, 53–61.

—— & Haughton, E. (1962) Control of the behaviour of schizophrenic patients by food. *Journal of the Experimental Analysis of Behavior*, 5, 343–352.

Fraser, D. (1983) From token economy to social information system: the emergence of critical variables. In *Current Issues in Clinical Psychology*, ed. E. Karas, Vol. 1. New York: Plenum Press.

Hall, J. & Baker, R. (1986) Token economies and schizophrenia: A review. In *Contemporary Issues in Schizophrenia*, eds A. Kerr and P. Snaith. London: Gaskell.

Higson, P. J., Woods, P. A., Tannahill, M. M. & Ellis, N. C. (1985) The role of meCals as a reinforcing event in a token economy programme. *British Journal of Psychiatry*, 147, 170–174.

Innis, N. K. (1981) Ethics of behavior modification: Behavioral and medical psychology. *Social Science Medicine*, 15F, 69–73.

Kazdin, A. E. (1977) *The Token Economy: A Review and Evaluation*. New York: Plenum Press.

Lowe, C. F. & Higson, P. J. (1983) Is all behaviour modification 'cognitive'? In *Current Issues in Clinical Psychology*, ed. E. Karas, Vol. 1. New York: Plenum Press.

Moyes, T., Tennant, T. G. & Bedford, A. P. (1985) Long-term follow-up study of a ward-based behaviour modification programme for adolescents with acting out and conduct disorders. *British Journal of Psychiatry*, 147, 300–305.

Stolz, S. B. (1976) Ethical issues in behaviour modification. In *Ethics of Social Intervention*, eds G. Bermant & H. Kelman. Washington, DC: Hemisphere.

Wexler, D. B. (1973) Token and Taboo: Behavior modification, token economies, and the law. *California Law Review*, 61, 81–109.

—— (1975) Reflections on the legal regulation of behavior modification in institutional settings. *Arizona Law Review*, 17, 132–143.

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A Study of Schneider's First-Rank Symptoms of Schizophrenia in Nigerian Patients

A prospective evaluation of Schneider's first-rank symptoms in 56 Nigerians with RDC schizophrenia gave a prevalence of 73%. The presence of first-rank symptoms was unrelated to schizophrenic subtypes, duration or severity of illness. While some of the symptoms correlate with age of patients, the pattern of inter-correlations among them supports previous views about their empirical heterogeneity.

The first-rank symptoms of Schneider continue to be of considerable interest with regard to their value in the diagnosis of schizophrenia (Carpenter *et al*, 1973) and their prognostic implications (Carpenter & Strauss, 1972). Even though Schneider regarded these 11 symptoms as pathognomonic of schizophrenia in the absence of organic brain dysfunction, recent evidence has brought into question the validity of this claim (Carpenter *et al*, 1973).

In spite of the remarkable stability in the frequency of FRS across schizophrenic samples in many studies (Mellor, 1970; Carpenter *et al*, 1973; Carpenter & Strauss, 1974; Ndetei & Singh, 1983), there is evidence that these symptoms do not form an empirically homogenous symptom group (Mellor, 1970). Also, while there is suggestion that schizophrenics with FRS may have a more severe illness as indicated by a higher overall psychotic symptom rating and disruption of instrumental role performance (Silverstein & Harrow, 1981), many studies have shown that these symptoms have no prognostic implications (Carpenter & Strauss, 1972). Thus, so far, the most appropriate status of the FRS remains to be determined.

Only very few studies of FRS have been done among African patients (Carpenter & Strauss, 1974; Ndetei & Singh, 1983). Specifically, apart from the evaluation of the prevalence of eight FRS in patients included in the International Pilot Study of Schizophrenia (Carpenter & Strauss, 1974), there is as yet no report of any systematic study of FRS among Nigerian patients.

This report investigates the prevalence of FRS among Nigerian schizophrenics, the relationship of FRS to each other and to the major diagnostic subtypes of schizophrenia, age at presentation, duration of illness, and the severity of the illness at presentation.

Method

The subjects were 56 consecutive new patients seen at Aro Hospital over a period of 3 months. Only patients were included in the study who (a) met the Research Diagnostic Criteria (RDC) for definite schizophrenia (Spitzer *et al*, 1975), (b) had informants with them who could give a detailed description of their behaviour during current illness, and (c) were not on active medication with a known psychotropic drug. All the patients were seen by one of the authors (O.G.) either immediately or within 48 h of contact with the hospital. This was to allow for accurate assessment of symptoms before they had been substantially modified by treatment. The informant was first asked for a general description of the patient's current level of functioning. This was rated on the Global Assessment Scale (Endicott *et al*, 1976). Using the listing by Mellor (1970) but assessing 'made impulse' and 'made affect' together, the presence of FRS was established by following the interview format described by Carpenter *et al* (1973) for eight of the symptoms, and Present State Examination style routine questions for two types of auditory hallucination: voices commenting, and voices arguing. The questions in these formats were found quite easy to give in Yoruba, the local language in which most of the patients were interviewed. Yoruba is the interviewer's first language and was the language used for the Nigerian section of the IPSS. Delusional percept was established during the overall mental state assessment and from the informant's report. Special care was taken in assessing all the FRS bearing in mind the ambiguity that may arise in delineating their boundaries. Only symptoms rated as definitely present were considered positive and all other ratings, including questionably present, were considered negative.

Results

The group comprised 27 men and 29 women with mean age of 27.8 years (s.d. 8.25). Analysis into RDC schizophrenic subtypes gives 25 (45%) mixed, 21 (38%) paranoid, nine (16%) disorganised, and one (2%) catatonic. There was no significant difference among the three commoner subtypes with regard to the presence of FRS ($\chi^2=3$; d.f. $=2$, NS). Fifteen patients had no FRS, six had one, seven had two, 11 had three, ten had four, three had five, and four had six (mean $=2.41$; s.d. $=1.89$). Thus, one or more FRS were present in 41 of the 56 patients giving a prevalence of 73%. The commonest FRS were made volition, thought insertion, and voices commenting. Delusional percept was the least common (Table I).

TABLE I
Distribution of individual FRS

FRS	*No. of patients*	%
1. Audible thoughts	7	13
2. Somatic passivity	15	27
3. Thought insertion	19	34
4. Thought withdrawal	11	20
5. Thought broadcast	13	23
6. 'Made affect and/or 'Made' impulse	11	20
7. 'Made' volition	22	39
8. Voices commenting	19	34
9. Voices arguing	15	27
10. Delusional percept	3	5

Even though there were a few significant correlations among FRS, there was no consistent pattern of association between either the special forms of auditory hallucinations or those symptoms said to represent a disturbance of the ego boundary.

Of eight significant correlations, none was within the former group and only three were within the latter group: thought withdrawal with thought broadcast ($r=0.36$; $P<0.02$), made affect/impulse with made volition ($r=0.43$; $P<0.02$), and somatic passivity with made affect/impulse ($r=0.30$; $P<0.05$).

Only two symptoms showed significant positive associations with age: voices commenting ($r=0.36$; $P<0.02$) and somatic passivity ($r=0.30$; $P<0.05$). The correlations with duration of illness did not attain statistical significance.

There were also no significant correlations with the severity of illness as rated on the GAS although there was a trend for thought insertion to correlate with less severity, and delusional percept with greater severity.

Discussion

The prevalence of FRS of 73% obtained in this sample bears close similarity to the 72% obtained by Mellor (1970) among British patients and 73% by Ndetei & Singh (1983) in their Kenyan sample. An earlier study of FRS among Nigerian patients (Carpenter & Strauss, 1974) found a prevalence of 63%. The difference between that report and the present one may be due in part to the fewer number of FRS (eight of the 11) assessed in that study, and differences in the diagnostic criteria used. The high frequency of made volition and thought insertion, noted in the present study, is similar to the observation of Carpenter & Strauss (1974) and Ndetei & Singh (1983). However, their results suggesting voices commenting as the least common are dissimilar to our finding. Delusional percept is present in 5% of the current sample, similar to the 6% obtained by Mellor (1970). Unlike Carpenter *et al* (1973), we found no significant associations between FRS and the four RDC subtypes of schizophrenia present in the studied sample. We believe, as Mellor (1970) says, that "it would be fortuitous if first-rank symptoms, selected solely for their diagnostic value, were central to syndromes of the theoretical interest."

Our results suggest that even though the presence or absence of FRS in general may not bear significant relationship to age, there may be some individual FRS that do so. This finding needs further clarification by other studies. The lack of significant association between FRS and duration of illness noted in this sample is similar to the observation of Carpenter *et al* (1973). There has been a suggestion that FRS-positive patients may have a more severe illness than those without (Silverstein & Harrow, 1981). We only noticed such a trend with regard to patients having delusional percept.

The pattern of intercorrelations among FRS in this study further supports the view that FRS are an empirically heterogenous group of symptoms and that this point bears remembering in determining their clinical and research use.

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References

CARPENTER, W. T. & STRAUSS, J. S. (1972) The prediction of outcome of Schizophrenia: characteristics of outcome. *Archives of General Psychiatry*, 27, 739–746.

—— & —— (1974) Crosscultural evaluation of Schneider's first-rank symptoms: a report from the International Pilot Study of Schizophrenia. *American Journal of Psychiatry*, 131, 682–687.

——, —— & MULETI, S. (1973) Are there pathognomonic symptoms in Schizophrenia? *Archives of General Psychiatry*, 28, 847–852.

ENDICOTT, J., SPITZER, R. L., FLEISS, J. L. & COHEN, J. (1976) The Global Assessment Scale: a procedure for measuring overall severity of Psychiatric disturbance. *Archives of General Psychiatry*, 33, 766–771.

MELLOR, C. S. (1970) First-rank symptoms of schizophrenia: I. The frequency in schizophrenics on admission to hospital: II. Differences between individual first-rank symptoms. *British Journal of Psychiatry*, 117, 15–23.

NDETEI, D. M. & SINGH, A. (1983) Schneider's first-rank symptoms of schizophrenia in Kenyan patients. *Acta Psychiatrica Scandinavica*, 67, 148–153.

SILVERSTEIN, M. L. & HARROW, M. (1981) Schneiderian first-rank symptoms in schizophrenia. *Archives of General Psychiatry*, 38, 288–293.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1975) *Research Diagnostic Criteria (RDC). NY State Dept of Mental Hygiene, NY State Psychiatric Institute*, Biometrics Research.

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Books Reconsidered

Cognitive therapy and the emotional disorders: A. T. Beck*

Beck's original book on cognitive therapy was published in the USA in 1976 but not reviewed in the *Journal* until December 1979 (**139**, 582–583). The review was favourable. A second book (Beck *et al*, 1979) was not reviewed, although by then there was a substantial and growing literature on cognitive approaches to treatment.

Beck is a psychiatrist and former psychoanalyst who came to be increasingly dissatisfied with psychoanalytic theories and the results of therapeutic interpretations based on them. His earlier book on depression, published in 1967, was one of the first modern works concerned with the disorder. In it he reviewed the literature and some studies of his own, with an emphasis on symptom phenomenology. He also reported work with a symptom inventory which he had developed earlier and which has since been used widely.

Subsequently, his cognitive theories evolved from a more direct look at the depressive thought content, with considerable therapeutic contributions from behaviour therapy. In the late 1960s in the USA, moves away from psychoanalysis were gaining momentum, but were still not easy for the committed; I can remember the cool reception which a rather muted lecture at Yale around 1970 from Dr Beck (a kindly and courteous man) evoked in the senior figures in the audience.

The 1976 book set out the theoretical structure evolved by Beck and his group. The 1979 work, larger but very readable, is more a treatment manual. The theories centre around conscious ideation, moving away, as Beck pointed out, both from the unconscious thought processes of the psychoanalysts and from the neglect of thought content by the behaviour therapists. Beck directed attention to the presence of automatic thoughts, negative in content. These thoughts were regarded as preceding and giving rise to negative moods. He made a detailed analysis of what he described as systematic logical errors in thinking, such as arbitrary inferences and over-generalisation. He postulated underlying cognitive schemes which might give rise, when activated, to disorders such as depression. A method of therapy focusing on thought processes was described, aimed at recognition and control of the negative thoughts. The therapy uses techniques such as recording of thoughts, reality testing, and cognitive rehearsal. It is verbal, but in its structure and use of techniques such as homework exercises and self-monitoring has a clear behavioural component.

The 1976 book ended with the forthright claim that in comparison with psychoanalysis, cognitive therapy was readily comprehensible to patients, economical with regard to therapists' time, testable in research, and easily teachable to others.

On rereading this book one is struck by the clear, economical style. It consists of firm statements by a man who knows what he wants to say, and who is conscious of setting out for others an authoritative statement of a new theory which will have a memorable impact. Also, it is apparent that Beck was not just talking about depression. He deals with thoughts in relation to the normal emotions of sadness, euphoria, anxiety, and anger, and to the full gamut of emotional disorders: depression, anxiety, phobic and obsessional states, psychosomatic disorders, and hysteria. Only when he moves to treatment does he focus on depression.

It is not yet easy to assess the place of Beck's work: it has not reached a final point of equilibrium. It certainly took off with great success. By the late 1970s a vigorous group of young therapists and researchers were attending conferences and presenting data with some fervour. More recently, cognitive therapy has become part of the general armamentarium of the clinical psychologist. A number of controlled trials in depression have been carried out, which show the treatment to be at least as effective as drug therapy. It has led to further books, by various authors, to at least one journal, and to some distinguished research by psychologists in this country, clustering in Cambridge, Edinburgh, and Oxford.

However, its practical place in depression is still far from clear, and it is possible that reaction will set in. The treatment is intensive in its use of therapist time: drug treatment is considerably cheaper. On the other hand, there are early hints that cognitive therapy may reduce relapse rates. Studies have largely been confined to the milder range of depression, particularly general practice patients and out-patients. There has been little comparison of cognitive therapy with drugs or ECT in more severe depression. Most cognitive therapists are reluctant to tackle chronic and resistant depressions, although it is here that the need for alternative treatments is greatest. It is not certain whether the therapeutic effects are specific or reflect non-specific effects of the considerable therapeutic contact. Most studies do not show the effects particularly on thought content rather than other aspects of depression. Results so far available from the recent large American collaborative trial comparing cognitive therapy, interpersonal psychotherapy, drug therapy, and placebo suggest that all active treatments have approximately equal and similar effects but that cognitive therapy may have less effect in the severe depressions. Comparisons of recovered depressive patients with controls do not show the proposed abnormalities of thinking which might predispose to depression. This has led to a greater emphasis on the latent nature of the depressive schemas: a dangerous move towards the unverifiability of unconscious processes. Beck's own position appears to have changed over the years from regarding negative cognitions as causing depression to the view that they are not necessarily causal but part of the depressive syndrome and a convenient point of leverage for effecting change in it.

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*International Universities Press, New York, 1976.

These are more than mere cavils, but less than fatal flaws: they are current hesitations. The real place of cognitive hypotheses and of cognitive therapy in depression will take longer to establish. Meanwhile, there is no doubting that the impact has been enormous. We have seen the birth of a modern movement. Others, particularly among clinical psychologists, were moving towards cognitive approaches at the same time as Beck, but it was his early work and his formulation which brought the therapy into being. Whatever its ultimate place as a treatment in depression, it has brought thought content back to centre stage in concepts of disorder. It has produced a new acceptance that psychological treatments can have major effects in a disorder where biological mechanisms appear important. It is also rapidly extending outside depression, for instance in the treatment of appetite disorders, and in a common disorder where alternative treatments are not satisfactory: generalised anxiety. Cognitive approaches are a major part of the currently evolving packages of anxiety management programmes. The approach itself may turn out to be the greatest contribution.

Reference

BECK, A. T., RUSH, A. J., SHAW, B. L. & EMERY, G. (1979) *Cognitive Therapy of Depression*. New York: Guildford Press.

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Consensus Statement: Panic Disorder

SIR: We welcome the consensus statement (*Journal*, April 1987, **150**, 557–558) from a group of British psychiatrists that the status of panic disorder as a separate entity is not strongly supported by available clinical and scientific evidence.

Their statement, arising from a meeting arranged by a pharmaceutical company, devotes a fair amount of space to drug therapy for panics, but very little to non-drug methods of treatment. It omits to mention that systematic exposure lastingly relieves the most common type of panic – phobic panic. Evidence for the durable value of systematic exposure rests on numerous controlled studies and follow-ups four to seven years later: far longer than after any drug study. In chronic panic, enduring improvement after exposure was achieved without the side-effects and with less of the relapse on ceasing medication that can be troublesome in drug treatment.

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Temperament, Personality and Personality Disorder

SIR: Rutter (*Journal*, April 1987, **150**, 443–458) discusses a paper we have written on the subject of childhood temperament (Graham & Stevenson, in press). We have suggested that temperamental characteristics can be helpfully viewed as minor variations of behaviour which, in extreme form, can be regarded as psychiatric disorders, and have put forward evidence to support this view. We have not, as Rutter suggests, conceptualised temperamental characteristics as "mini-disorders" (his term, not ours) and indeed, in minor form, we see no good reason at all to think of such characteristics in pathological terms. Rutter rejects our view on five grounds, none of which, for reasons we state here, seems to us to constitute a valid objection.

Extremes of temperamental traits do not in themselves constitute disorder: This categorical statement by Rutter carries with it little meaning without definition of the terms concerned. We would argue that temperamental traits, such as activity, emotionality and 'socialisability', when shown in extreme form, do indeed represent handicapping forms of behaviour, and have cited evidence in the paper to which he refers.

Most of the symptoms of child psychiatric disorder are not part of temperamental concepts: We have not argued that *all* psychiatric disorder can be viewed as extremes of temperamental characteristics. Such a view would be absurd, and we have made this clear in our paper in which we cited anorexia nervosa and autism as two disorders that could not be so conceptualised. We have argued that extremes of the common varieties of temperament do constitute common disorders, and that much childhood depression, anxiety, hyperactivity, and antisocial behaviour can usefully be conceptualised in this way.

Patterns of correlation with change in behaviour rather than current disorder do not fit easily with our hypothesis: We are unaware of any significant body of work examining the relationship between background factors such as family disharmony or school failure with *change* of behaviour, although some such work certainly exists. Indeed, our own longitudinal study (Richman *et al*, 1982) represents one of the most substantial in this area. Most examination of background factors in relation to childhood disorder has concerned disorders of varying duration. There is no reason, however, why, given our hypothesis, change in background factors should not relate to a change in behaviour along a continuum from everyday behaviour to trivial, mild, moderate, or severe disorder, nor why temperament construed in the way we suggest should not be related to later behavioural change.

Our suggestion bypasses evidence on the indirect path by which temperament may lead to disorder via influences on other peoples' reactions: In fact, our hypothesis would have no difficulty in accommodating such evidence. If, for example, everyday but subclinical behaviour of an irritating type leads to parental rejection of child A more than everyday behaviour of a less irritating type in child B, it would not be surprising if child A's reaction to rejection involved a more extreme and disordered form of the same behaviour.

Our hypothesis fails to take account of the repeated finding that the effects of temperament may vary by sex: We are not suggesting that our hypothesis can be used to explain all phenomena that relate to temperament – it would be highly suspect if it did. On the other hand, the hypothesis is certainly not disconfirmed by differential responses to temperamental characteristics in boys and girls.

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References

GRAHAM, P. & STEVENSON, J. Temperament and psychiatric disorder – the genetic contribution to behaviour in childhood. *Australian and New Zealand Journal of Psychiatry* (In press).

RICHMAN, N., STEVENSON, J. & GRAHAM, P. (1982) *Pre-school to School: A Behavioural Study*. London: Academic Press.

SIR: I am sorry that Graham & Stevenson feel that my use of the term 'mini-disorder' does not represent their concept of temperament. Nevertheless, it does seem in keeping with their argument in the earlier paper I cited (Stevenson & Graham, 1982). With respect to the observed association between temperament and the later development of behaviour disorder, they stated: "in fact we are merely observing a mild (or not-so-mild) problem turning into a larger one".

Their letter is helpful in clarifying their theoretical position, but let me comment briefly on the implications of the view that "much" child psychiatric disorder constitutes extremes of temperament. Firstly, the notion that psychiatric disorder implies more than just the extreme of a single behavioural trait is not just mine. The criteria for most psychiatric disorders in both ICD–9 and DSM–III require patterns of multiple symptomatology and social impairment. Moreover, as noted in my paper (Rutter, 1987), many *common* disorders are defined in terms of types of behaviour that are not part of any concept of temperament.. That is obviously the case with problems such as enuresis or encopresis, but it is also so with many conditions, such as phobic states, that have a closer connection with temperamental features. The development of, say, school phobia or agoraphobia involves something different from a general escalation in anxiety; that is why they are differentiated from anxiety states.

Secondly, with acute disorders there is the need to account for onset and remission. It does not seem likely that this is accountable for in terms of accentuations and reductions in temperamental features. Nevertheless, I agree that evidence both for and against is lacking. Also, it is common for psychiatric disorders in childhood to exhibit considerable situation-specificity. This phenomenon requires a form of explanation that does not fit readily into a concept of extremes of temperament.

Thirdly, with pervasive chronic disorders there is the assumption of continuity between normal variations in the temperamental feature and its apparent equivalent in the pathological condition. Again, the empirical evidence is sparse, but it seems likely that often this is not the case. For example, Graham's demonstration that some cases of the hyperkinetic syndrome respond to an oligoantigenic diet (Egger *et al*, 1985) suggests that the disorder is not just the manifestation of a high level of temperamental activity.

Fourthly, I appreciate that the Graham & Stevenson view by no means excludes circular processes involving interactions with environmental influences. However, what it surely finds more difficult to encompass are processes that have results that involve the emergence of types of behaviour that are different from accentuations or diminutions of temperamental features.

Finally, there is the most basic point of all: the assumption that, if psychiatric disorder is just an extreme of temperament, both should share the same set of correlates. But is that so? Certainly there is a lack of supporting evidence. The implication, for example, is that the genetic basis of the two is the same and that the environmental features associated with psychiatric disorder should apply similarly to temperament.

I chose to discuss Graham & Stevenson's concept because of my high respect for their research and I would like to take the opportunity of publicly thanking them for their courtesy and helpfulness in letting me see and comment on their paper in advance of its publication. Our views on temperament differ in crucial respects but we are entirely at one in the view that empirical evidence will decide between our concepts. Such evidence is being obtained by them (as well as by other investigators), and I await their findings with great interest.

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References

EGGER, T., CARTER, C. M., GRAHAM, P. J., GUMLEY, D. & SOOTHILL, J. F. (1985) Controlled trial of oligoantigenic treatment in the hyperkinetic syndrome. *The Lancet*, *i*, 540–545.

RUTTER, M. (1987) Temperament, personality and personality disorder. *British Journal of Psychiatry*, **150**, 443–458.

STEVENSON, J. E. & GRAHAM, P. J. (1982) Temperament: a consideration of concepts and methods. In *Temperamental Differences in Infants and Young Children*. Ciba Foundation Symposium 89 (eds R. Porter and G. M. Collins). London: Pitman.

Huntington's Chorea

SIR: Martindale (*Journal*, March 1987, **150**, 319–323) has raised an important issue that is relevant not only to Huntington's chorea but also to child psychiatry. It is well known that parents often have difficulty in recognising depression in their children. Professional responses to child psychiatric difficulties are less often discussed, and it seems worth drawing attention to a suggestion recently put forward by Emde *et al* (1986). Childhood depression was described many years ago, but it took a long time to gain general acceptance both by clinicians and by research workers. Emde *et al* hypothesised that this might be because depressed children threaten to arouse painful feelings in the professionals with whom they come into contact, and this may lead the professionals to fail to recognise the condition.

We are all familiar with 'observer effects' in research and these often seem to be regarded as contaminations which prevent us from discerning the truth. Both Martindale and Emde *et al* seem to suggest that this is only partially true and that some emotional responses (such as denial) will blind us to the truth, but others, such as the ability to tolerate feelings of hopelessness, helplessness and sadness in ourselves, may enable us to make a diagnosis which would otherwise elude us.

If this is correct it would seem to have several implications. Firstly, regarding teaching, perhaps we should spend more time helping our trainees to be more aware of their emotional responses to their patients; and secondly, as regards research, it would at least seem worth recording the emotional reactions of the interviewer to the patient.

My clinical impression is that patients often evoke similar emotional responses in a large number of professionals, and it could be that we should be taking more notice of this.

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Reference

EMDE, R. N., HARMAN, R. J. & GOOD, W. V. (1986) Depressive feelings in children: a transactional model for research. In *Depression in Young People, Developmental and Clinical Perspectives* (eds M. Rutter, C. E. Izard and P. B. Read). New York & London: The Guilford Press.

Anorexia and Antidepressants

SIR: We wish to take up and extend Crisp's theme (*Journal*, March 1987, **150**, 355–358) regarding the mode of action of antidepressant drugs in patients with anorexia nervosa. We have recently studied a group of four male patients with eating disorder, depressive symptoms, and obsessional behaviour.

All four have several features in common, including early age of onset (13–15 years), dietary restriction with consequent weight loss, and obsessional, repetitive exercise routines. One patient exercises with such force that he has sustained several muscle injuries. Three of the patients have been admitted to hospital in the past with transient weight gain. All patients have experienced marked anxiety in relation to loss of control of body weight. This has occurred, for example, when exercise was disrupted by injury or infection, and has led to anticipatory ruminations about the risk of contracting illness and losing control over body weight or body 'fat content'.

In all four, obsessional behaviour has extended beyond the spheres of diet, preparation of food, and preoccupation with exercise to the extent that they are unable to make simple decisions – almost amounting to 'folie de doute'. Three of the four patients are high-achievers academically, pursuing courses in tertiary education; the fourth has been sufficiently ill to interrupt his schooling, although he was regarded premorbidly as being of above average potential. Their academic success has been jeopardised by their obsessional insistence on studying every subject in excessive detail, which leads to their falling behind in their study schedules, causing anxiety and precipitating further ruminations.

All four are currently improved in their academic and social functioning (but not in their core anorexic psychopathology) following the administration of drugs which block 5HT re-uptake with varying degrees of specificity (e.g. fluvoxamine or clomipramine).

Crisp justifies the use of clomipramine by drawing attention to the possible primary phobic and secondary avoidance behaviour and obsessional features within anorexia nervosa. We concur with the view that in some anorexic patients obsessional features play an important role (Dally, 1969), particularly in male patients (Beumont, 1972), and such symptoms contributed significantly to the difficulties encountered by our patients. Taking Crisp's argument that tricyclic drugs have an immediate effect on drive behaviour further, we suggest that in addition they reduce repetitive, 'stereotyped' behaviour as manifested in impaired decision-making seen in these patients.

It is likely (Dally, 1969) that many such patients do not seek, or even actively avoid, in-patient treatment and in these circumstances the prescription of 5HT re-uptake blocking drugs may prove to be of considerable value in their academic and social functioning, although not in their core anorexic psychopathology.

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References

DALLY, P. (1969) *Anorexia Nervosa.* London: Heinemann.

BEUMONT, P. J. V., BEARDWOOD, C. J. & RUSSELL, G. F. M. (1979) The occurrence of the syndrome of anorexia nervosa in male subjects. *Psychological Medicine,* **2,** 216–231.

Zinc in Senile Dementia

SIR: McLoughlin (*Journal,* March 1987, **150,** 409) appears to have selected information from various studies quoted by Srinivasan (1984) and concluded that lower zinc levels are present in the elderly functionally ill and in those with confusion. These studies cover a wide range of diagnoses and relate both to adult and psychogeriatric populations.

Our paper clearly describes the work of Bunker *et al* (1984), who suggest that the mean level of plasma zinc in healthy elderly adults is 11 μmol/litre (s.d. = 1.2). The levels reported in our study compare well with Bunker's elderly group. It is reasonable, therefore, to imply that the low levels of zinc in senile dementia which McLoughlin suggests he would have demonstrated using a healthy elderly control group would not have materialised.

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References

SRINIVASAN, D. P. (1984) Trace elements in psychiatric illness. *British Journal of Hospital Medicine,* **32,** 77–79.

BUNKER, V. W., HINKS, L. J., LAWSON, M. S. & CLAYTON, B. E. (1984) Assessment of zinc and copper status of healthy elderly people using metabolic balance studies and the measurement of leucocyte concentrations. *American Journal of Clinical Nutrition,* **40,** 1096–1102.

Life Events and Mania

SIR: We are surprised that the *Journal* published an article which purports to measure life events prior to the onset of mania (*Journal,* February 1987, **150,** 235–240) when the method of life event assessment is simply that of searching case notes. Ambelas makes some important deductions from his assessment of life events, but we believe that the weakness of the life event assessment method is such that these could be misleading.

We are in the early stages of measuring life events prior to the onset of mania and are using the reliable method of Brown & Harris (1978) (LEDS). After assessment of only 11 patients two factors have become clear. Firstly, in the year prior to onset of mania 26 life events (according to the LEDS) have been elicited at interview, but only two of these were recorded in case notes. Secondly, the time between onset of symptoms and admission varied between 2 and 90 days (median 13 days), which indicates that measuring life events prior to admission, as performed by Ambelas, could be grossly misleading as there will be many illness-related events following the onset of a manic illness.

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BROWN, G. W. & HARRIS, T. O. (1978) *Social Origins of Depression.* London: Tavistock.

Khat-Induced Paranoid Psychosis

SIR: In February 1984 we reported (Gough & Cookson, 1984) a case of khat-induced psychosis in a 27-year-old man originally from North Yemen who presented with paranoid delusions and thought broadcasting similar in nature to those described by Critchlow & Seifert (*Journal,* February 1987, **150,** 247–249). In our patient a urine drug screen at admission was positive for amphetamines but negative for opioids, cannabinoids, cocaine metabolites, and barbiturates. This is to be expected, as khat contains a number of amphetamine-like substances such as d-norpseudoephedrine.

Our patient's family stated that khat is widely used by Yemeni people in Liverpool, fresh supplies being flown in daily. It commonly produces some degree of elation and hyperactivity, but not frank psychotic symptoms.

In January 1985 we were contacted by journalists from *The Sunday Times* (20 January 1985) in connection with an investigation they were carrying out into the use of khat in Britain. They had found out that large quantities of the substance were being imported into London without restriction.

The Regional Drug Information Service informs us that no further cases have come to light in Liverpool, and it would appear that at present the use of the drug is mainly confined to certain ethnic communities. Possibly because of this it may be less likely to come to the attention of medical services.

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GOUGH, S. P. & COOKSON, I. B. (1984) Khat-induced schizophreniform psychosis in the UK. *The Lancet*, *i*, 455.

SIR: This case report (Critchlow & Siefert, *Journal*, February 1987, **150**, 247–249) does not accurately describe a typical case of khat psychosis. We reported three cases of catha (khat) psychosis (Dhadphale *et al*, 1981): our patients had paranoid psychosis with florid delusions which occurred in clear consciousness, and excessive use of khat was suspected as a possible causal agent. In a five year follow-up we did not find any relapse of psychosis in our subjects. Since then we have seen more than a dozen patients with khat psychosis, and all of them have shown a similar clinical picture as well as outcome.

As khat chewing is blamed for every social evil in our society by the lay press, our department has been involved in substantial research on various aspects of khat chewing (Omolo, 1985; Omolo & Dhadphale, 1987*a*). Our country grows and exports good quality khat leaves and a substantial number of our people in khat-growing areas chew khat for its amphetamine-like stimulant effect. Many use alcohol to counteract the sleeplessness which inevitably follows khat use (Omolo & Dhadphale, 1987*b*).

Critchlow & Seifert's case appears to us to be a mixed psychosis, probably toxic in origin, and due to several factors such as respiratory infection, possible morphine or dihydrocodeine use or abuse, or subtle malnutrition due to poor appetite and sleep deprivation – the latter two being the result of chewing khat. The authors should have inquired about the quantity of leaves chewed by the patient during the weeks prior to admission.

However, they have rightly alerted the medical and psychiatric communities in the UK to the possibility of khat being involved when a young person presents with a paranoid psychosis. Evidence of khat use may be found if the clinician examines the oral cavity of the patient for evidence of ulcers or stained teeth, which are common among habitual users.

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DHADPHALE, M., MENGECH, H. N. K. & CHEGE, S. W. (1981) Miraa (catha edulis) as a cause of psychosis. *East African Medical Journal*, **58**, 130–135.

OMOLO, O. E. (1985). *Medical Problems Among Khat Users*. M.Med. dissertation, University of Nairobi, Kenya.

—— & DHADPHALE, M. (1987*a*) Prevalence of khat chewers among primary health clinic attenders in Kenya. *Acta Psychiatrica Scandinavica* (In press).

—— & —— (1987*b*) Alcohol use among khat chewers in Kenya. *British Journal of Addiction* (In press).

Capgras' Syndrome in a Patient with Dementia

SIR: We report a patient who typifies some of the issues raised by Kumar (*Journal*, February 1987, **150**, 251).

Case report: A 79-year-old woman presented with a three-year history of the gradual onset of impaired memory. Two years after onset her husband was admitted to hospital for a few days for a minor operation. On his return he noticed that his wife made repeated references to her 'first' husband, whom she claimed had died some years before. She now referred to her husband as her second husband, despite acknowledging the very close resemblance in physical appearance, name, and profession between the two. Even though the husband could not convince her of his true identity, she showed no hostility towards him; as she said, "the new man is quite nice to be with". The husband's attempts to remind her of their earlier married life were always met with the reply: "but I was married to John in those days". At the time of presentation this belief had persisted unchanged for six months.

On examination, she had moderate impairment of memory for recent and remote events. She was mildly dysphasic and dyspraxic and could not name, or recognise as familiar, any of the subjects in a Famous Faces test. She did not score above chance level on a forced choice test of facial recognition. There was no evidence of depression or other abnormal beliefs, and her insight was limited to concern about her poor memory. CT brain scan revealed mild ventricular dilatation and sulcal widening but other investigations were normal, and a presumptive diagnosis of senile dementia of Alzheimer type was made.

This case emphasises some of the problems with regard to the nosological status of Capgras' syndrome in the cognitively impaired. There may be a spectrum of disorders of recognition. In our case there was no suggestion that the patient believed her husband was an imposter or that he had been maliciously replaced. Our case is similar to those described by Pick (1903) in introducing the concept of reduplicative paramnesia. While resisting a psychoanalytical interpretation, and in the absence of evidence of focal brain lesions (Lewis, 1987), the sequence of events in the genesis of our patient's belief might be as follows: the couple are reunited after a separation but because of a combination of poor visual memory and failure of facial recognition the wife does not recognise her partner's face. However, a sense of familiarity is retained because of other cues, e.g. mannerisms, and has to be explained. The patient's interpretation of events may then take one of two paths: either a confabulation involving a 'duplication' of the same person experienced at two different points in time (or in two different settings as with Pick's cases), or a delusional elaboration that the original partner has been replaced by an imposter. This latter is the classic Capgras' syndrome and may arise more readily in patients with premorbid paranoid traits (Burns, 1986). The status of such phenomena in the cognitively impaired may have implications for treatment and requires further elucidation.

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References

BURNS, A. (1985) The oldest patient with Capgras' syndrome? *British Journal of Psychiatry*, **147**, 719–720.

LEWIS, S. (1987) Brain imaging in a case of Capgras' syndrome. *British Journal of Psychiatry*, **150**, 117–121.

PICK, A. (1903) Clinical studies III. On reduplicative paramnesia. *Brain*, **26**, 260–267.

ECT in the Elderly

SIR: The report by O'Shea *et al* (*Journal*, February 1987, **150**, 255–257) of cognitive improvement in a very elderly depressed patient treated with ECT is useful in that it reminds us that elderly people should not be denied an effective treatment solely on the grounds of age.

Clinical features which predict a good response to ECT in younger patients also predict a good response to ECT in the elderly (Fraser & Glass, 1980), and the presence of co-existing dementia does not adversely affect the efficacy and safety of ECT (Weiner, 1982). Age alone is not a contra-indication to the use of ECT (Benbow, 1985).

The clinical details quoted by O'Shea *et al* give little support to a diagnosis of dementia in their 91-year-old patient. She had previously responded to dothiepin, and the episode which led to the use of ECT seems to have been of less than 12 months duration. Her cognitive function was impaired as tested by the Mini-Mental State at a time when she had a frankly psychotic depressive illness, and improved as she recovered in response to treatment. This is not surprising. Fraser and Glass (1980) studied 29 elderly patients (aged 64–86 years) treated with ECT and found that they all had impaired memory function before treatment. Three weeks after ECT all scores had reached levels acceptable as normal. The clinical experience of many psychogeriatricians bears this out.

One problem is that the literature regarding the use of ECT in the very elderly is limited. In a study of 122 patients treated with ECT by the psychogeriatric service at the University Hospital of South Manchester only 16 were aged over 80 years and the oldest was a woman of 88 years (Benbow, 1987).

It is important that ECT is made available to elderly people with depressive illnesses, where the clinical features suggest a likely response and antidepressant drugs have not been successful or cannot be tolerated. Babigian & Guttmacher (1984) demonstrated a substantially decreased mortality risk in women over 75 years of age treated with ECT in comparison with a non-ECT group. Perhaps age could be regarded as an indication for ECT, rather than the reverse?

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References

BABIGIAN, H. M. & GUTTMACHER, L. B. (1984) Epidemiologic considerations in electro-convulsive therapy. *Archives of General Psychiatry*, **41**, 246–253.

BENBOW, S. M. (1985) Electro-convulsive therapy in psychogeriatric practice. *Geriatric Medicine*, **15**, 19–22.

—— (1987) The use of electro-convulsive therapy in old age psychiatry. *International Journal of Geriatric Psychiatry* (In press).

FRASER, R. M. & GLASS, I. B. (1980) Unilateral and bilateral ECT in elderly patients. A comparative study. *Acta Psychiatrica Scandinavica*, **62**, 13–31.

WEINER, R. D. (1982) The role of electro-convulsive therapy in the treatment of depression in the elderly. *Journal of the American Geriatrics Society*, **30**, 710–712.

Admission Rates and Lithium

SIR: The argument seems to be about the 'efficacy' and perhaps the 'efficiency' of this drug (*Journal*, February 1987, **150**, 264–265). We would like to add further data to this discussion.

Recently, we examined infradian rhythms of mood and allied factors in out-patients with major affective disorders and matched healthy controls (Eastwood *et al*, 1985). Each person was interviewed with the Schedule for Affective Disorders and Schizophrenia, and the index group had to meet the Research Diagnostic Criteria. A sub-group from this study consisted of 11 patients who had been on lithium for an average of seven years, and these were contrasted with 22 healthy controls. These patients were treated independently by other psychiatrists and took only lithium during the study period. Patients, all of whom had bipolar affective disorders, and controls completed visual analogue scales daily (Eastwood *et al*, 1984) for 14 months. The mood ratings for index and control groups were compared using the means, standard deviations, and delta squares. The variability of mood, using chi-square analysis, was significantly skewed towards zero in the lithium group, compared with controls, for the standard deviations and delta squares.

So cases of bipolar affective disorder had less mood variability over 14 months on lithium than normal controls. The Folstein *et al* (1982) short-term finding in this regard (one month) is more than confirmed. Our study speaks to efficacy, in some cases, but says nothing about efficiency. Elsewhere we have shown that prescribing is not completely efficient, since in Toronto approximately two-thirds of patients with bipolar affective disorders receive lithium and two-thirds of those on lithium have bipolar affective disorders (Eastwood *et al*, 1981).

The very effectiveness of lithium could create problems by making patients overly controlled, without spontaneity and creativity (Jamison *et al*, 1979), and cause them to stop taking it. This would fit with Kendell's idea of withdrawal causing relapse and ever more mania. How ironic if patients' non-compliance in this disruptive illness was a function of effectiveness of treatment.

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References

EASTWOOD, M. R., STIASNY, S. & TICE, S. (1981) Estimates of the prevalence of bipolar affective disorder: methods based on treatment with lithium. *Acta Psychiatrica Scandinavica*, **63**, 83–90.

EASTWOOD, M. R., WHITTON, J. L. & KRAMER, P. M. (1984) A brief instrument for longitudinal monitoring of mood states. *Psychiatry Research*, **35**, 773–783.

EASTWOOD, M. R., WHITTON, J. L., KRAMER, P. M. & PETER, A. M. (1985) Infradian rhythms: a comparison of affective disorders and normal persons. *Archives of General Psychiatry*, **42**, 295–299.

FOLSTEIN, M. F., DEPAULO, J. R. & TRIPP, K. (1982) Unusual mood stability in patients taking lithium. *British Journal of Psychiatry*, **140**, 188–190.

JAMISON, K. R., GERNER, R. H. & GOODWIN, F. K. (1979) Patient and physician attitudes toward lithium. *Archives of General Psychiatry*, **36**, 866–869.

Psychiatric Morbidity and the Mentally Handicapped

SIR: Day's (1985) reported morbidity of 30% and 20% for psychiatric disorder in mentally handicapped patients of a mental handicap hospital and a day hospital respectively, may be equally valid with respect to mentally handicapped out-patients.

In a random analysis of one year's attendance of mentally handicapped patients at a small community-based mental handicap out-patient clinic (January–December, 1982) I found that 19% of the 115 out-patients seen were suffering from a significant mental illness: depressive illness 10 (i.e. manic depressive 2, agitated depression 6, depression with epilepsy 1, depression after brain tumour 1); schizophrenia 9 (i.e. paranoid 6, hebephrenic 2, simple 1); and severe personality disorders 3. This excludes behaviour disorders which were present in 36%. Diagnoses were made on the basis of a comprehensive history (from patients' relatives, social workers and community nurses), appropriate clinical criteria and, in doubtful cases, by admission for assessment, observation, and follow-up.

Sturmey's claim (*Journal*, February 1987, **150**, 270) that the behaviour problems due to painful physical conditions (abscesses, nasal infection, etc.), learned behaviour, and anti-social behaviour (wandering, stealing, etc.) may inflate the serious psychiatric morbidity in the mentally handicapped is not borne out by the many well-conducted studies in this field. In fact, most of these studies include major psychiatric illnesses such as schizophrenia, affective disorder, and personality disorder, and exclude the behaviour problems which he mentions.

The diagnosis of major psychiatric illness in mentally handicapped patients has always been difficult, as the signs and symptoms of the illness may be masked by the behavioural expression, as illustrated below.

Case report: A young severely mentally handicapped patient attending a special needs unit expressed his mood changes in a regular pattern of periods of overactivity, agitation, aggression, sleeplessness, and exhaustion, which resulted in loss of weight, chest infections, etc. (hypomanic phase), alternating, after a brief period of stability on medication, with phases of withdrawal, irritability, loss of his usual sociability, and tearfulness (depressive phase). This cycle lasted between 9 and 15 months. His response to lithium therapy was remarkable, and showed in his physical and emotional well-being, as well as in improvement in his learning, social and personal skills.

I hope that Sturmey is not attempting to minimise the importance of the recognition of treatable major mental illness in mentally handicapped patients, many of whom have suffered greatly in the past from the lack of appropriate treatment (mainly psychotropic medication) simply because their mental disorders were unrecognised.

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DAY, K. (1985) Psychiatric disorder in the middle-aged and elderly mentally handicapped. *British Journal of Psychiatry*, **147**, 660–667.

Is the Positive-Negative Distinction in Schizophrenia Valid?

SIR: The longitudinal study by Johnstone *et al* (*Journal*, January 1987, **150**, 60–64) raises questions on the validity of the concept of positive and negative symptoms in schizophrenia. Their original proposition that this dichotomy represents different pathogenic mechanisms had been predicated largely on the assumption that positive symptoms are treatment responsive, and hence reversible, whereas negative symptoms are not. The current findings indicate, however, that positive symptoms are not uniformly treatment responsive and that negative symptoms are not universally immutable.

Our longitudinal studies of schizophrenia also challenge the validity of the positive-negative model as originally proposed, and suggest that other dimensions, notably chronicity of illness and diagnostic sub-type, may interact with this construct. In a two year follow-up of young patients with acute schizophrenia (Lindenmayer *et al*, 1986), both positive and negative syndromes proved longitudinally unstable and showed changing patterns of external correlates over time. Contrary to assumptions from studies in the chronic phase, it was the base-line negative rather than positive presentation which anticipated a favourable outcome. A cross-sectional survey of 134 patients with schizophrenia classified as acute (up to 2 years history of illness), chronic (3–10 years), and long-term chronic (over 10 years) similarly suggested that an early negative syndrome carries a better prognosis. In patients with acute schizophrenia such a profile was associated with depressive and atypical catatonic features as well as absence of psychosis in first-degree relatives (Kay *et al*, 1986). Thus, the positive-negative dichotomy may have a quite different clinical significance in the acute phase of psychosis to that in the chronic phase.

Recent pharmacological analysis has suggested, moreover, that the positive-negative classification delineates homogeneous psychobiological sub-types in schizophrenia only if considered along with diagnostic sub-type (Singh *et al*, 1987). The addition of anticholinergic drugs to neuroleptic treatment in a mixed population of patients with acute, subacute and chronic schizophrenia produced a significant worsening of positive symptoms overall, but this occurred mainly among patients with catatonic symptoms. In paranoid patients, to the contrary, it was the negative symptoms that worsened significantly, whereas in hebephrenic patients neither syndrome was significantly affected. Cumulatively, our results suggested that the natures of positive and negative syndromes vary according to chronicity and subtype of schizophrenia, and perhaps only in the chronic patients and the non-paranoid forms of illness is the present concept of the positive-negative dichotomy applicable.

The data from Johnstone's group underscore qualifications on the generality of the construct. They observed that delusions – a cardinal feature of paranoid schizophrenia – were significantly more prevalent in neuroleptic-treated than neuroleptic-free patients with schizophrenia, as were also symptoms of anxiety and depression. The latter finding may be understood within the context of a post-neuroleptic emergence of dysphoria and elevation of tonic autonomic arousal, which characterises only non-paranoid patients and portends poor therapeutic response (Singh & Kay, 1979). Furthermore, our work has suggested that non-paranoid neuroleptic-resistant patients with schizophrenia may be distinguished from others by clinical worsening under blind wheat gluten challenge (Singh, 1978; Singh & Kay, 1984). Research on the psychobiology and course of schizophrenia, therefore, casts doubt on the sufficiency of the positive-negative model. Study of its interaction with diagnostic sub-type and phase of illness, on the other hand, may offer new insights not afforded by examining any of these dimensions separately.

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References

KAY, S. R., FISZBEIN, A., LINDENMAYER, J.-P. & OPLER, L. A. (1986) Positive and negative syndromes in schizophrenia as a function of chronicity. *Acta Psychiatrica Scandinavica*, **74**, 507–518.

LINDENMAYER, J.-P., KAY, S. R. & FRIEDMAN, C. (1986) Negative and positive schizophrenic syndromes after the acute phase: a prospective follow-up. *Comprehensive Psychiatry*, **27**, 276–286.

SINGH, M. M. (1978) Some insights into the pathogenesis of schizophrenia. In *The Biological Basis of Schizophrenia* (eds G. Hemmings and W. A. Hemmings). Lancaster: MTP Press.

—— & KAY, S. R. (1979) Dysphoric response to neuroleptic treatment in schizophrenia: its relationship to autonomic arousal and prognosis. *Biological Psychiatry*, **14**, 277–294.

—— & —— (1984) Exogenous peptides and schizophrenia. In *Psychoneuroendocrine Dysfunction* (eds N. S. Shah and A. G. Donald). New York: Plenum Press.

——, —— & OPLER, L. A. (1987) Anticholinergic-neuroleptic antagonism in terms of positive and negative symptoms of schizophrenia: implications for psychobiological subtyping. *Psychological Medicine* (In press).

Neuroleptic Malignant Syndrome (NMS) and Tardive Dyskinesia

SIR: Haggerty & Gillette (*Journal*, January 1987, **150**, 104–105) seem to have concluded that NMS developed while the patient was having reserpine and lithium. The details they give do not support such a conclusion, as they mention in their discussion. For example, they do not mention any alteration in consciousness after lithium and reserpine were started: besides rigidity, hyperpyrexia and autonomic dysfunction, alteration of consciousness in varying degrees is a *sine qua non* for the diagnosis of NMS (Caroff, 1980). Here, only autonomic dysfunction was noticed after starting lithium and reserpine. Moreover, the progression of NMS is known to be very rapid after onset. In more than 90% of 120 cases reviewed, the full syndrome developed within 48 hours of the first symptoms (Shalev & Munitz, 1986). It is naïve to conclude that mild NMS developed over a period of two weeks and that the full-blown syndrome began later, when thioridazine and haloperidol were started.

What is of interest in this report is the unusually long duration of the NMS episode (seven weeks). Generally, it is presumed that NMS lasts for 5–10 days after discontinuation of oral neuroleptics (Sternberg, 1986). I have come across another patient in whom the syndrome persisted for three weeks after cessation of haloperidol and remitted after a 17-day course of amantidine (Woo *et al*, 1986). Treatment was continued for a further period of five months. Such an abnormally long duration may have something to do with the patient's metabolic characteristics, as differences in absorption and first pass metabolism are known to cause wide inter-individual variations in the plasma levels of neuroleptics. Moreover, neuroleptics, being highly lipophilic, can be detected for weeks after discontinuation.

Caution is required before concluding that unexplained fever is due to NMS. This is clear when one considers the authors' suggestion that reports by family of transient diaphoresis and elevations of temperature in the months prior to admission were probably because of NMS in "milder form". In the absence of any alteration of consciousness such an interpretation is not justified. At the moment, there is not enough clinical data to justify the spectrum concept of neuroleptic toxicity (Corlon, 1986) which the authors seem to have in mind.

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References

CAROFF, S. N. (1980) The neuroleptic malignant syndrome. *Journal of Clinical Psychiatry*, **41**, 79–83.

CORLON, P. (1986) The spectrum concept of neuroleptic toxicity. *American Journal of Psychiatry*, **143**, 811.

SHALEV, A. & MUNITZ, H. (1986) The neuroleptic malignant syndrome: agent and host interaction. *Acta Psychiatrica Scandinavica*, **73**, 337–347.

STERNBERG, D. E. (1986) Neuroleptic malignant syndrome: the pendulum swings. *American Journal of Psychiatry*, **143**, 1273–1275.

WOO, J., TECH, R. & VALLENCE-OWEN, J. (1986) Neuroleptic malignant syndrome successfully treated with amantidine. *Postgraduate Medical Journal*, **62**, 809–810.

Continuum of Psychosis and the Gene

SIR: Crow's theory (*Journal*, October 1986, **149**, 419–425) bears a striking similarity to the theory of Britain's best-known psychologist, Hans Eysenck, to the effect that a heritable trait of psychoticism (*P*) underlies much psychotic (and indeed psychopathic) disturbance (Eysenck & Eysenck, 1976, 1985). Like Crow's continuum, Eysenck's *P* has sometimes been considered to be related to cerebral lateralisation of function (Brand, 1981) and to forms of creativity and achievement (Claridge, 1985) that might explain the persistence of high-*P* genes in the population despite the lowered average fertility of people who suffer outright psychotic disturbance.

I wonder, could the theories of Crow and Eysenck by any chance be related?

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References

BRAND, C. R. (1981) Personality and political attitudes. In *Dimensions of Personality* (ed. R. Lynn). Oxford: Pergamon.

CLARIDGE, G. (1985) *Origins of Mental Illness: Temperament, Deviance and Disorder*. Oxford: Blackwell.

EYSENCK, H. J. & EYSENCK, S. B. G. (1976) *Psychoticism as a Dimension of Personality*. London: Hodder & Stoughton.

—— & EYSENCK, M. W. (1985) *Personality and Individual Differences: A Natural Science Approach*. New York: Plenum.

Koro-Like States

SIR: We have under our care a 25-year-old male patient with complaints of penis and testicles shrinking into his abdomen.

Case report: This man, from a working-class English background, initially presented in 1983 at the age of 21, with an anxiety state and reactive depression for which he was admitted to hospital for further assessment. He had had both psychic and somatic anxiety symptoms. At that stage there was no evidence of a major affective or schizophrenic illness. Like the patient described by Modai *et al* (*Journal*, October 1986, **149**, 503–506), this young man was physically well-built but emotionally immature. He presented a poor self-image and usually responded to any threat to his ego by being verbally aggressive. His parents' marriage broke up when he was nine. He was left with his father, whom he always perceived as being a domineering man. He could not forgive his mother for leaving him and still harbours a strong feeling of rejection. He had a history of faecal soiling and repeated hospital admissions for successive eye-operations (following an accidental injury) around the age of ten. He first complained of his penis and testicles retracting into his abdomen while in hospital in 1983, coincidental with the break-up of his first serious relationship with a girlfriend. His mood subsequently deteriorated. He was treated with amitriptyline (50 mg/day) and stelazine (15 mg/day). The depression improved, but his belief about his genitalia remained unaltered. He was re-admitted a year later, and again in 1986, with clear-cut episodes of hypomania. His hypomania has now remitted, but during both these admissions he maintained his belief about his retracting genitalia. His concern is such that he avoids places where his genitalia may be exposed and he despairs of ever having a girlfriend again.

In contrast to classical koro, there does not appear to be a cultural factor in this case. Although koro is rare in the Western hemisphere, koro-like states may not be uncommon and may be under-reported.

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HIV and the Psychiatric Hospital

SIR: Most readers are aware of the controversy concerning the treatment of patients infected with the human immunodeficiency virus (HIV) presenting with various medical problems. There has been little discussion of the problems encountered when psychiatric disorder arises in these patients. Colleagues in virology and venereology assure us that the transmissibility of HIV by any route other than by sexual contact or transfusion is small. We may therefore rest assured that the aggressive self-mutilating or spitting patient presents little risk to staff or other patients provided that proper precautions are taken. I have been assured that needlestick injuries are ineffective in transmitting HIV, although I would still like to see some evidence of this, given the frequent need to sedate acutely psychotic and resistive patients by intramuscular injection.

The major problem in dealing with HIV-positive patients lies elsewhere, and has moral and practical dimensions. It is not uncommon for psychiatrically ill patients to be in a state of sexual hyperarousal, most often seen in mania. Equally, most long-stay facilities have a significant sexual sub-culture where intercourse is traded e.g. for the price of a few cigarettes. While staff try to prevent such activities, it is impossible to achieve 100% success. Introduction of HIV carriers to a psychiatric unit therefore poses the threat of transmission to patients whose judgement is impaired by virtue of mental illness.

Morally, detention of a patient under a section of the Mental Health Act implies a responsibility to guard that patient against the consequences of his or her impaired judgement. The sheer terror induced by being diagnosed as HIV positive, plus the profile of *some* high risk groups (promiscuous homosexuals and drug addicts), suggests that as the HIV epidemic continues, psychiatric hospitals will have a higher prevalence of HIV carriers than the population at large. Furthermore, HIV-associated dementia will probably require psychiatric placement, again increasing the prevalence. The moral question I would ask is: 'How can we defend detaining a patient who is at risk to his or herself by virtue of mental illness and whose condition entails a tendency to sexual promiscuity, in a facility where the prevalence of HIV carriers may be above that in the community?"

Practical problems abound. Closer supervision through augmented staff levels could, in theory, preclude the possibility of intercourse in hospital. If a middle line is taken and only those who are HIV positive are "specialed", we run into the problem of consent for screening. Also, it is a criminal offence to disclose the HIV status of a patient to unauthorised people (not the case with Australia Antigen).

I believe that the special problems faced in psychiatric units as a result of HIV merit urgent discussion by the College, the Mental Health Act Commission and the DHSS. In particular, I wonder whether Parliament needs to review the problem of consent for HIV screening when patients are to be admitted to psychiatric hospitals, and also the question of the degree to which the dissemination of their HIV status can be permitted. The use of 'high risk' categories in determining levels of surveillance, while still of use, cannot be considered adequate, given the spread of HIV into the heterosexual non-drug-abusing community.

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First Admissions of Native-Born and Immigrant Patients to Psychiatric Hospitals

SIR: In their study of first admissions of native-born and immigrant patients to mental hospitals in South-East England, Dean *et al* (*Journal*, 1981, **139**, 506–512) found increased rates of admission for most immigrant groups, particularly for illnesses diagnosed as schizophrenia.

However, their findings, which were based on routine data from the four Thames Regions, differed from more locally organised studies in showing substantially smaller excesses, particularly for Asians. For example, for all diagnoses for Indians Dean *et al* found a 50% excess of first admissions for men and a 20% increase for women. For immigrants from Pakistan and the rest of the Asian New Commonwealth they found a 40% deficit.

By contrast, for example, Carpenter & Brockington (*Journal*, 1980, **137**, 201–205) found, for all Asian groups combined, excesses of 200–500% at different age groups. The only exception was the 15–24 year-olds, who showed a 50% excess.

A possible explanation for the discrepancy is the notorious incompleteness of the data source Dean *et al* used (Mental Health Enquiry (MHE)). The authors attempted to address this problem by organising a campaign to improve completeness of recording. However suspicion must remain that while overall they achieved quite good results – 91% completion of the place of birth field – this success may have been patchy, giving rise to systematic distortion in their conclusions. A closer examination of the data for one of the four Regions they studied for 1976 suggests this is likely.

Dean's study excluded all but first admissions so as to avoid multiple counting of individuals. This meant that it depended not only on the birthplace field in each record, but also on the previous admissions question. Permitted responses to this include "not known", and people so coded are not included in first admission statistics. A survey in Newham Health Authority (Glover, 1985) found that about two-thirds of admissions so coded almost certainly were first admissions. Dean also excluded records with the birthplace omitted.

A study of N. W. Thames regional MHE data for 1976 by the Area Health Authority showed that the urban Areas had a higher proportion of first admission records with birthplace uncoded (ranging from 1.4% in Bedfordshire (Beds) to over 10% in Ealing, Hammersmith and Hounslow (EHH) and in Kensington, Chelsea and Westminster (KCW). Urban Areas also had a higher proportion of records with previous admission status "not known" (ranging from 2% in Beds to 16% in Brent and Harrow and 33% in KCW).

In general, but particularly in EHH, birthplace was more frequently missing from records with previous admission status "not known". The same urban areas had rather higher proportions of immigrants in their resident populations than the rural ones.

It seems reasonable to imagine that language difficulty is one obstacle records clerks may encounter in eliciting a patient's previous admission status. Clerks may also be unwilling to ask about some patient's birthplace if racial tension is prominent in the district served. The hospitals serving the Notting Hill Gate area, one of the more racially disturbed at the time, had particularly low rates of birthplace recording.

These findings suggest that the figures Dean *et al* produced are probably a serious underestimate and that the local studies should be considered to be more reliable. They particularly call into question the widely quoted idea that Asians make less use of psychiatric services. This view is based mainly on MHE studies, and runs counter to the impression of many psychiatrists who have worked in areas with large Asian populations.

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Dates of Acceptance

SIR: Readers with an eye for detail might have noticed a change in the March *Journal*. Would-be contributors are no longer allowed to muse over the gestation period of papers accepted for publication: the date which used to be given at the end of each article has disappeared. Eighteen months ago dates of receipt were transmuted into dates of acceptance. Since, notwithstanding, the *Journal* continues to enlarge, perhaps the next step might be to omit page numbers.

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CORRIGENDA

Journal, October 1986, **149**, 535. Review of *Handbook of Psychiatry*. 'Jarrold Woolfson' should read 'Gerald Woolfson'.

Journal, March 1987, **150**, 412. Letter by Jacob *et al.* "Neurotic individuals appear to be more prone to develop AIDS-phobia in India than in the West" should read "... *and* in the West."

Journal, May 1987, **150**, 723–724. The letter entitled "Neuroleptic Malignant Syndrome and Compulsive Water Drinking" is by D. N. Anderson, *Sefton General Hospital, Smithdown Road, Liverpool L15 2HE.*

A HUNDRED YEARS AGO

A curious outbreak of convulsionist mania, analogous to those which occurred from time to time during the Middle Ages, is reported to have shown itself at Agosta, in the province of Rome. For some weeks past the country people have been labouring under the delusion that the district is under the immediate government of the Evil One, and before retiring to rest they carefully place on the threshold the broom and the salt, which are credited with the power of keeping off evil spirits. Many of the younger women have epileptiform attacks, during which they utter piercing shrieks, and are violently convulsed. So serious had the condition of things become, that the syndic of Agosta found it necessary to inform the Prefect, who sent detachments of soldiers into the district in order to calm the apprehensions of the inhabitants. The contrast between this strictly physical way of dealing with the disease and the more imposing but less effectual religious ceremonies formerly employed must be interesting to historical students. As a natural consequence of this condition of mental perturbation, the country is overrun with quacks who claim to possess the only infallible remedy for the seizures. One of these nostrums, the vendor of which was making a rich harvest from its sale, was found on analysis to consist of earth, snuff, and borax. Three medical men who were commissioned to investigate the cause and nature of this extraordinary affection, came to the conclusion that it was an epidemic of hysteria. They examined a number of the sufferers, mostly young women, some of whom were alleged to have vomited nails, horseshoes, and other equally indigestible substances, while others barked like dogs. Several of them were removed to Rome for treatment in the hospitals there, and measures have been taken to check the spread of the mischief. In a milder degree, this contagious form of hysteria is not infrequent, especially in places where ignorance and superstition favour manifestations of nervous disorder. The worst excesses of popular outbreaks, like the French Revolution, have been attributed to similar influences, and with every appearance of justice.

Reference

British Medical Journal, 1887, *ii*, 94.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

British Journal of Psychiatry (1987), **150**, 884–891

Book Reviews

Editor: Sidney Crown

The Breaking of Bodies and Minds: Torture, Psychiatric Abuse, and the Health Professions. Edited by ERIC STOVER and ELENA O. NIGHTINGALE. Oxford: W. H. Freeman. 1985. Pp 319. £21.95 (hb), £11.95 (pb).

This book is testimony to modern man's potential for physical and mental abuse and inhumanity to his fellow man – conducted usually under the jurisdiction of repressive regimes or institutions and, unfortunately, facilitated willingly, unwillingly, or unwittingly by medical and biomedical professionals. Psychiatrists and their powerful tools are liable to co-option by the state or by institutions, and their participant role may be central or supportive. Some non-participants subsequently may profess no knowledge about such acts; others may turn a blind eye; but there are the courageous few who (at considerable personal risk) note and protest such abuses and attempt to arouse international awareness.

Indubitably such acts abrogate universally accepted norms of human rights. Individual practitioners and medical associations are confronted with the challenge of abolishing and resisting complicity in state or institutional abuses. One way of helping such individuals is by the availability of internationally accepted guidelines and codes of ethics which define professional complicity, and which unequivocally assert that such abuses are morally wrong. For instance, the brief set of principles adopted by the United Nations General Assembly (18 December 1982) merits wide publicity. How many medical journals have troubled to publish these? And yet I believe they should be mandatory reading for all health workers.

While there should be no compromise about such codes of ethics in practice, the book hints at some unresolved issues. Firstly, the concept of misuse of medical resources to suppress political and religious dissent is often widened to include variations in quality of psychiatric care. However, a distinction needs to be made between misuse of medical skills and maldistribution of health resources to the poor in the face of their misery and powerlessness.

Secondly, there are the complex political issues. What constitutes a repressive regime? How should ideological differences be perceived and questions of infringment of national sovereignty be answered? How do endeavours which are aimed at political change differ from the health profession's action on behalf of human rights? A formula must be found for dealing with these issues in a non-political way: in particular, codes of ethics should be non-political. But what other means are available? Some of these issues were addressed at a recent meeting convened by the Royal College of Psychiatrists (White, C. (1986) *British Medical Journal*, **293**, 1524); they include "the anomalies of trying to bring pressure to bear while preserving dwindling contacts, and of the importance of being seen to be fair rather than sanctimonious, and professional rather than political".

Thirdly, what should be the reaction to a medical association in a particular country when a small core of their health professionals systematically abuse their medical skills? Excommunication of the whole association is likely to be counter-productive: it may stigmatise even those who silently or openly condemn such abuses.

Borrow or buy this book; it costs little enough. The chapter on 'Unwilling patients' by the psychiatrist Anatoly Koryagin provides compulsive reading; the reader may wish to dip further.

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Professional Supervision in Group Care: A Contract-Based Approach. By JAMES S. ATHERTON. London: Tavistock. 1986. Pp 202. £6.95(pb).

James Atherton is a teacher of social studies who has led workshops on supervision. This book has arisen from the ideas and approaches used during these courses. It is aimed specifically at social work staff in residential homes and day centres. For this reason it is a practical and didactic work, rather than a reflective one, and at the level it is pitched it works well.

The book is divided into three parts. The first is the meat of the book, dealing with what supervision is all about and how it should be conducted. The short second part discusses some of the topics of supervision, and the third part looks at problematic areas and develops some of the themes touched on earlier in the book.

The writing is admirably clear, the approach to supervision is sensibly argued, and a wide range of accessible references for further reading are given. The two criticisms I have are very specific, but important. Firstly, the topic of transference is poorly dealt with, and I can only think that anyone coming upon the idea for the first time would end up with a very muddled view. Secondly, there is a fundamental error in the section devoted to evaluating supervision, where a specimen questionnaire is shown with a three-point scale in which one point signifies negative and two points positive evaluations. Such a scale is inherently biassing, as opposed, for example, to a five-point scale with a neutral mid-point.

Can a book targetted at so specific a readership be of interest to others, such as psychiatrists? I think it is not of general interest, but anyone contemplating supervising staff in a residential or day care setting (such as a therapeutic community) would find plenty of useful advice.

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Attack on the Self: Adolescent Behavioural Disturbance and their Treatment. By DEREK MILLER. Northvale, NJ: Jason Aronson. 1986. Pp 340. $32.50.

Miller suggests in this book that two crucial ideas are required if adolescent psychopathology is to be truly understood. The first is the biopsychosocial basis of all psychiatric disorders. The second is the need, particularly with adolescents, to view any disorder from a developmental perspective. It is because of the adolescent's particular phase in development that emotional disturbance is expressed overwhelmingly through visible behaviour. The severity of the behavioural problems may be quite out of proportion to the severity of that underlying emotional disturbance. Relieving the symptoms may, through its effects on those around the adolescent, truly cure the disease. The first three chapters expand on these arguments and deal with the developmental and social issues. The next two deal with general principles of assessment and treatment, and the remaining eight with specific disorders.

What comes over strongly in this book is the author's deep compassion and concern for the well-being of adolescents and his anger over the way they can be treated (or mistreated) by adults. Equally, the author's great clinical experience is apparent from the many brief clinical vignettes used to illustrate his points. Despite this, the book as a whole is surprisingly unsatisfactory. One major problem is the question of to whom it is addressed. The only hint is in the acknowledgements, where medical students at North Western University are thanked for their comments on the draft. The level is indeed appropriate for an undergraduate audience: the discussions on aetiology, prevention, and treatment are insufficiently detailed to be of any real help to professionals. I would however have great reservations about recommending it for an undergraduate class of medical or other students; there is a narrow selectivity in the choice of references used to illustrate the author's arguments. Key pieces of work which do not fit in with his thesis are ignored. In many places controversial statements are made with no attempt to support these with evidence from research. The book is really the philosophy of adolescent psychiatry of one experienced practitioner. I feel that by not defining a target audience, it has left itself without one. I would have enjoyed reading Miller's detailed thoughts behind some of the ideas he puts forward, which are often arguments going against conventional widsom. Alternatively, on a simpler level, the book could have been a useful guide for the perplexed parent or teacher. As it is, I can unfortunately think of no group to whom I would recommend it as an introduction to adolescent psychiatry.

STEPHEN WOLKIND, *Consultant Child Psychiatrist, Maudsley Hospital, London*

Handbook of Depression: Treatment, Assessment, and Research. Edited by E. EDWARD BECKHAM and WILLIAM R. LEBER. Chicago: The Dorsey Press. 1985. Pp 1079. £51.00, $72.00.

This is a big book; its 1079 pages are assembled into 27 chapters and are written by 51 American and 2 British authors. Obviously, in a multi-author work of this size there will be good parts and bad parts, but the editors should be congratulated for persuading all the contributors to come right up-to-date with their references; all the chapters are very well referenced. Both the editors are psychologists, and the emphasis is on the psychological aspects of depression; however, the biological aspects are not neglected and the chapter on biological processes (Thase, Frank & Kupfer) is one of the most concise and up-to-date accounts that exists anywhere.

The book starts with ten chapters on therapies: cognitive (Beck), behaviour (Hoberman & Lewisohn), long-term analytic, brief psychodynamic, interpersonal psychotherapy, family and marital, and alternative (transactional analysis, gestalt, and reality therapies). The common failing of all these accounts is that authors do not delineate the areas of clinical usefulness of their approaches; most of them seem to imply that the therapy they describe is generally effective in a condition termed 'depression' (without further qualification), although sometimes a caution is given about trying to treat the acutely suicidal patient by a psychotherapeutic approach. Bellack provides a useful overview of psychotherapy research and points out that little is known about how psychotherapies compare with one another, for whom and conducted by whom they are effective, and what they all hold in common; surprisingly, on the last of these issues he does not quote Jerome Frank, and nor does anyone else. Bellack points out that many of the claims of proponents of psychotherapies are not established, and he closes his chapter with a consideration of four broad 'mechanisms'; he omits a fifth, which is that most depressive disorders remit spontaneously and that natural recovery may well occur in the course of these expensive encounters. The conduct of somatic therapy (apart from lists of drugs) is poorly described, and the authors of a chapter on the management of refractory depression (Akiskal & Simmons) seem to spend a lot of time, and therefore their patients' patience and money, in taking them off drugs only to prescribe them again but without clear and well documented credentials for what they are doing; in the end they frequently prescribe the long delayed electroconvulsive therapy which "more often than not" proves effective.

The problem of definition of depressive disorders continues to becloud the whole area, despite all the grandeur of DSM–III and the Research Diagnostic Criteria. Some authors come close to acknowledging that these systems do not define homogeneous disorders and Kathol, in the chapter on depression in the medically ill, underlines their failure to identify a psychiatric disorder requiring treatment. Batteries of psychological and physical investigations lead to confusing results of uncertain application, because they are applied to heterogeneous disorders which hold in common only the universal term 'depression'. No author in this book has taken up the idea, put forward by Klein over a decade ago, of a biogenic depression styled 'endogenomorphic' which might be distinguished from other depressive states on clinical grounds. Until some such characteristics are firmly established, research and treatment of depression will continue to hobble along, swayed now by one and then by another enthusiasm; indeed, this book shows that this is exactly what is happening. The two British contributors, Bebbington and Wing, have provided a good chapter on epidemiology in which they acknowledge, partly due to this failure to identify homogeneous states, that "the epidemiology of depression has hardly begun".

I would recommend the book as a convenient source of up-to-date references, generally well written and very readable. It would be most useful in those clinical psychology departments which organise training courses, but all clinicians will find one or more of the chapters to be helpful.

PHILIP SNAITH, *Senior Lecturer in Psychiatry, University of Leeds*

Ethical Issues in Epidemiologic Research (Series in Psychosocial Epidemiology, Vol. 7). Edited by LAURENCE TANCREDI. New Jersey: Rutgers University Press. 1986. Pp 203. $15.00 (pb).

If you would like to ponder on some contemporary questions about psychiatry and research, this book could be a place to start. If, on the other hand, you already know a fair amount about ethics, epidemiology or sociology you should probably steer clear of it. In eight chapters, two of which have been published before, Tancredi has brought together authors from medical and legal backgrounds but without a unifying intellectual perspective. Tancredi himself starts with a thoroughly positivist view of epidemiological research designs, but later acknowledges the contribution, at least in forensic psychiatry, of ideas from the Frankfurt School philosophers. Other chapters include discussions on access to public records for longitudinal studies, confidentiality in clinical settings, and the patient's rights to refuse treatment – all issues current in Britain. Natalie Abrams, a lawyer, uses the example of research into the genetic basis of schizophrenia to raise the ethical question of such research: would or should our society take action as a result of the information gained? But a rambling discussion of the use of diagnoses in psychiatry by Walter Reich, once again presenting Soviet psychiatry as an Evil Empire and conveniently ignoring wider questions of how all States maintain social control, points to the failing of this book: it lacks critical sociological thought.

MARK MCCARTHY, *Senior Lecturer, Department of Community Medicine, University College, London*

Neuropsychological Assessment of Neuropsychiatric Disorders. Edited by IGOR GRANT and KENNETH M. ADAMS. Oxford: Oxford University Press. 1986. Pp 536. £40.00.

This is a major book which should find a permanent place in any neuropsychiatric reference library. It brings together a series of chapters on the neuropsychological assessment of different neuropsychiatric disorders. The references alone make its contribution valuable. Unfortunately, not enough of the authors are able to go beyond a detailed description of the literature to analyse the current conceptual position of their subject, and fewer still to use this analysis to carry understanding of the subject forward. One of the exceptions is Eric Caine, whose chapter on the neuropsychology of depression is the most forward-looking account of this subject currently available. The whole book is interesting historically in that different chapters represent both the traditional approach to psychology and psychiatry and the more recent neurological approach to these two disciplines. For example, Ralph Reitan on his neuropsychological test battery and Albert Carlin on the neuropsychological consequences of drug abuse still adhere to the nineteenth century model, equating mental with cortical function and localising psychological functions in topographical cortical areas. Pat McKenna and Elizabeth Warrington, in contrast, represent a more modern analytic approach to psychological assessment, and the Queen Square Battery, based on solid clinical research, deserves to become the standard adjunct to the WAIS for general neuropsychological assessment. It is only a pity that their assessment of impairment in psychiatric disorder is limited to describing it as 'functional', although they recognise that increasing knowledge of neuropsychological function increases the chances of being able to provide a cerebral basis for otherwise apparently bizarre symptoms. The attempt to distinguish between 'functional' and 'brain related' dysfunction by others such as Milterg, Hebben, and Kaplan on the Boston Process Approach to neuropsychological assessment and Larry Squire on the assessment of memory dysfunction is unworthy of a neuropsychiatric text. Allan Yozawitz, in his account of applied neuropsychology in a psychiatric centre, points the way forward in this respect. In the preface the editors place psychiatry firmly in the medical fold with neuropsychiatry as one of the neurosciences. They are to be congratulated in having produced a book worthy of this aspiration.

DANIEL ROGERS, *Consultant Neuropsychiatrist, Burden Neurological Hospital, Bristol*

A Handbook of Communication Skills. Edited by OWEN HARGIE. Beckenham: Croom Helm. 1986. Pp 480. £35.00 (hb), £14.95 (pb).

This is an important book for those who engage in professional encounters which demand a degree of skilled communication: it will be particularly enjoyed by those who recognise the need to improve social skills during processes of communication in order to achieve higher levels of verbal and non-verbal competence in their daily work.

There are 19 chapters under the four main headings of 'Background', 'Core communication skills', 'Group skills', and 'Dimensions of communication'. Topics covered range from communication as skilled behaviour to handling strong emotions, from questioning to case conference presentation. As a member of a professional group who spends much time communicating with patients, students, and colleagues I found this book very interesting. Most of the contributions are a pleasure to read, and offer useful summaries of the skills we need to be aware of when communicating with others. Some of the chapters also offer insights into areas that contain depths we may not always appreciate when we participate in the routine of our professional encounters. The book offers some critical analysis of social interaction, which many of us have recognised as important but have up until now perhaps treated only with anecdotal criticism.

As with most multi-author books there are chapters which fail to rise to the levels set by other contributors; these are usually recognisable by their uncomfortable written style. Perhaps writers who are intent on developing debate about social skills in verbal communication should ensure first of all that their written skills match their verbal aspirations! The chapter on questioning, for example, required great efforts on my part before meanings became clear. Occasionally the thinking of some writers appears to be woolly or only half-formulated. In the chapter on self-disclosure, for example, we are told that "Facts can be friendly. Judgements may or may not be." This seems awfully like one of those anecdotes we are used to hearing that hide more than they reveal. However, most of the chapters are of a high standard, and of these I would like to single out Chapter 2, written by Hargie & Marshall and entitled 'Interpersonal communication — a theoretical framework'. This chapter ranges over a wide field which the reader is able to follow effortlessly: there's much to consider, reflect upon, and take note! Possibly the best chapter was George Brown's 'Explaining'. Brown provides several informative examples, and supplies references to texts written specifically for different professions; the perceptive reader should pick up plenty of ideas for possible research topics. Another chapter which workers in health or education are likely to find helpful is that on 'Chairmanship'. It follows a main strand running through this book, which is to develop awareness of ways we communicate in order that such awareness might lead to changes for the better. It is this last pursuit which makes the book such an impressive and important one for anyone working in the health service.

B. A. WILSON, *Principal Clinical Psychologist, Charing Cross Hospital, London*

How to Stop Taking Tranquillisers. By PETER TYRER. London: Sheldon Press. 1986. Pp 96. £2.50.

This is Tyrer's third contribution to the Sheldon Press *Overcoming common problems* series. It is not the first self-help guide to tranquilliser dependence and withdrawal, but will be a useful addition to the literature available to the general public.

The first chapter, 'Tranquilliser dependence – the facts', describes what tranquillisers are and defines the concept of drug dependence. It concludes by assessing the frequency of tranquilliser dependence, putting it into perspective without raising the anxiety level of the readership. Chapter 2 provides reasonably comprehensive tables of commonly prescribed psychotropic medication, along with associated dependence risks. Through the use of seven convincing case histories the author describes possible responses to chronic tranquilliser (or hypnotic) usage. Chapter 3 is aimed at enabling readers to decide which of Tyrer's two approaches to withdrawal (rapid or slow) they should follow. They are able to give themselves scores on type, dosage, and length of usage of drugs, previous withdrawal symptoms, life problems, and personality in order to aid the decision. The following two chapters outline the two withdrawal programmes, and are on the whole clear and easy to follow, bringing in self-monitoring of symptoms as well as straight advice on dose reduction. Chapter 6 has a cognitive–behavioural flavour and is concerned with adjuncts to the withdrawal programme; it includes explanations of the body's response to stress and ways of dealing with anxiety and related symptoms, an essential component of the approach. It also mentions self-help groups, the possibility that depression may follow the withdrawal, and discussion of the withdrawal symptoms themselves. The final brief chapter discusses the possibility that the programmes may not be a success and emphasises that other addictions are far more dangerous. The appendix contains some useful addresses and a short list of references.

This is a non-sensational book with sensible advice for tranquilliser users. It is easy to read, and would be especially useful for patients seeking advice on the topic of withdrawal needing not intensive psychiatric input but rather encouragement to take responsibility for their drug intake and their symptoms.

ANNA HIGGITT, *Locum Consultant Psychiatrist, The Maudsley Hospital, London*

Handbook of Eclectic Psychotherapy. Edited by JOHN C. NORCROSS. New York: Brunner/Mazel. 1986. Pp 500. $47.50.

In some circles in the psychotherapy world, eclecticism is a dirty word; it is held to be synonymous with muddle-headedness or the last refuge for mediocrity. For others, it heralds the coming of a bright new future where rapprochement, convergence, and integration will still the clamouring of the dissenting psychotherapy tribes, who number more than 400 at the last count. In North America the Society for the Exploration of Psychotherapy Integration has been formed; several of its distinguished members have contributed to this somewhat grandiosely titled *Handbook of Eclectic Psychotherapy*. This substantial, well-referenced volume delineates the area to be explored – Norcross is to be commended for listing the chapter heading guidelines that he provided for contributers, a model of comprehensiveness and rigour – but demonstrates that we have a long way to go.

Eclecticism may mean the selective use of the most appropriate approach for a particular patient, or the integrative amalgam of what is best in various systems, or an idiosyncratic practice that takes from many sources and reflects the style of the therapist. Actually, most experienced therapists practice idiosyncratically, but the systematic eclectic looks to see how various orientations complement each other. The book itself may be seen as a series of Venn circles. In the centre there are nine chapters of the integrative-idiosyncratic variety; they include Lazarus on multi-model therapy, Beutler with a systematic approach, Garfield in iconoclastic form, and presentations on time-limited and family therapy and case work. The inward-looking attitude of many of these presentations is brought out in a critique by Dryden, and this is wrapped around by more crossed-referenced criticisms in excellent chapters on the concepts and history of eclecticism, possibilities and promises, training, relationship to outcome studies, and a lucid, astringent account by Messer of assumptions, problems, and trade-offs. I was particularly taken with Messer's analysis of the way in which different psychotherapies view the possibilities of life according to romantic, ironic, tragic, and comic visions.

While dynamic therapy, especially in its brief form, becomes more behavioural and behavioural therapy more dynamic, through its interest in cognition and its substrate, psychotherapy still lacks a comprehensive, integrated theory. Many hurdles need to be surmounted before the eclectic ideal, a direction which this reviewer generally supports, is reached. Not least is the problem of doing justice to concepts from different frameworks in a single language; at this stage, uniqueness may be revelatory. Much comparative and process research needs to be done before we will know if the future lies with selective eclecticism in which the best fit is made between patient, approach, and talent of that therapist, or, in recognition of the fact that technique only contributes 15% to the outcome variance, with an integrative summation of the best of the most. Futurologists will want to read this book; libraries will need it as the reference source of what may become a mighty river.

MARK AVELINE, *Consultant Psychotherapist, Nottingham Psychotherapy Unit*

Contemporary Perspectives on Psychotherapy with Lesbians and Gay Men. Edited by TERRY S. STEIN and CAROL J. COHEN. New York: Plenum. 1986. Pp 271. £29.50.

Stein and Cohen are practising psychodynamic psychotherapists in North America. Their starting point for this book is the belief that "homosexuality is a variation of human behaviour and is neither a sign of illness nor an underdeveloped form of sexual expression." As "homosexuality co-exists in individuals who also demonstrate psychopathology... it is essential for the psychotherapist to understand the relationship of homosexuality to identity." The papers collected here make it clear that it is also essential for the therapist to understand the implications of homophobia, not only in the outside world but also in his or her own practice.

The book includes papers on historical and theoretical considerations, but most of the papers use extensive clinical material to illustrate the issues that arise in therapy with lesbians and gay men. There are particularly interesting papers on the dynamics of merger in lesbian relationships; psychotherapy and AIDS; individual, couple, and group psychotherapy with gay men; and psychotherapy with lesbian mothers. Among the many important questions considered are the relevance of the sexual orientation of the therapist, and the effect of self-revelation by the therapist, on transference and counter-transference in the client-therapist relationship.

In spite of the rapidly changing situation with regard to HIV infection, the paper on psychotherapy and AIDS condenses into a short space most of the important issues to consider when dealing with gay men who have the disease. The impact of the AIDS epidemic on gay relationships is also considered in a paper on psychotherapy with gay couples. Any future edition would need to consider the impact of the availability of screening for HIV antibodies on therapy with gay men.

With few exceptions, the authors are clear about the issues that are particular to lesbian and gay relationships, although occasionally the distinction between these and issues common to all relationships are left blurred.

This book may be dismissed as irrelevant or unimportant by those who might benefit most from reading it, but it is an intelligent, sensitive, and stimulating book which should be essential reading for any therapist working with lesbian or gay clients.

JUDY BURY, *General Practitioner and Psychosexual Counsellor, Edinburgh*

Psychiatric Disorders: Neurotransmitters and Neuropeptides (Progress in Brain Research Vol. 65). Edited by J. M. VAN REE and S. MATTHYSSE. Amsterdam: Elsevier Biomedical Press. Pp 275. $83.25, Dfk225.00.

This volume continues the high standard of the *Progress in Brain Research* series. The individual chapters are for the most part well written and extensively referenced. Most of them are detailed examinations of neurochemical hypotheses, rather than general reviews, and this is to be welcomed. The topics examined are wide-ranging – from neuropeptide localisation in the CNS to nutrition and its role in psychosis. Most of the chapters start with a detailed description of an aspect of basic neuroscience and follow with a discussion of putative clinical applications, and in many instances a stimulating and rewarding account results.

However, the volume suffers from the usual symptoms of a multi-authored book in that coverage of many topics (for example Alzheimer's disease) is patchy and highly selective. The chapters are written by well-known proponents of various biological hypotheses of psychotic illness, but there is no exchange of views or sign of critical editing. Thus the dopaminergic, noradrenergic, and peptidergic hypotheses of schizophrenia are outlined, but run in parallel with no point of contact.

This volume can be highly recommended for those with a specific interest in a research topic related to the application of basic neuroscience to psychiatry; if there is a chapter in this book relating to their own research interest they should obtain a copy of that chapter, because it is guaranteed to be detailed and an excellent source of ideas and references. However, the volume should carry a health warning for both trainees in psychiatry and in neuroscience who wish to have a concensus or critical view of the current status of biological psychiatry.

I. N. FERRIER, *MRC Clinical Scientist and Honorary Consultant Psychiatrist, MRC Neuroendocrinology Unit, Newcastle*

Psychotherapy with Adolescent Girls (2nd edition). By DORIS LAMB. New York: Plenum. 1986. Pp 265. $29.50.

This is a practical guide to a form of psychotherapy in which the emphasis is on helping the adolescent girl to complete her developmental tasks rather than on the achievement of insight: attempts at the latter can impede progress more often than they assist. At a time when conjoint family therapy has become the fashion, this text illustrates the place for individual therapy, particularly when done in a family context. The chapter on advising parents is especially helpful. As the title indicates, the book is about psychotherapy with girls; it does not explain how this differs from that with boys, and most of the material seems to apply equally to both sexes. That many of the references were specific to boys strengthened this impression of an artificial distinction.

The author makes her points using a large number of vignettes as a basis. The style is that of a supervision session, and this resemblance is heightened by the conversational quality of the text. Although pleasantly free of jargon and easily digested, there is a rhetorical repetitiveness and gossipy informality which suggests that this is a transcription of a series of monologues. The same points are made repeatedly. Although this may be excusable in America where the cost of the book is slight, its more substantial cost here makes this lax presentation a major flaw. The work needs to be edited radically, with the obvious stated more tersely and points made less frequently. With the removal of irrelevant detail, a review of the grammar, and careful proof-reading the reader would be free to enjoy the content of this book without the distraction of the form in which it has been thrown at him.

Overall this is a very useful book for anyone starting to learn the skills necessary for the intimacy of adolescent psychotherapy. Although it is suggested by the introduction, I would not recommend this to parents. It does not have the breadth to leave a balanced impression of adolescence, particularly with parents whose perspective has been distorted by their own difficulties. This book does provide a good concrete framework for therapy and indicates the commoner pitfalls. As such I recommend it as a book which most adolescent services will find of use to their staff whatever the discipline.

T. P. BERNEY, *Consultant Psychiatrist, Prudhoe Hospital, Northumberland*

Clinical Gerontology: A Guide to Assessment and Intervention. Edited by T. L. BRINK. New York: The Haworth Press. 1986. Pp 517. $49.95.

This book is divided into five sections. Section I, 'The assessment of the geriatric patient', begins with a chapter about the reason for the low utilisation of mental health services by the elderly, questioning traditional, 'ageist', and self-fulfilling assumptions about the type of care older patients need. There are chapters on assessment of spatial competence, neuropsychological test batteries, and the use of projective techniques. Section II, 'Depression scales for use in later life', contains an excellent chapter on clinical psychosocial assessment of depression in the elderly. Section III is titled 'Therapy in later life'. The first article reviews psychodynamic therapy with the aged, a neglected subject. A chapter on cognitive therapy has vivid case illustrations, with an encouraging description of an 83-year-old with forgetfulness, hearing, and visual problems who nevertheless benefited from cognitive therapy for depression. The article on group psychotherapy with cognitively impaired older adults is welcome. Section IV, 'Family therapy', includes a chapter on the techniques and contexts of multigenerational therapy with emphasis on practical aspects of therapy with extended families. Section V, 'Related topics', is a miscellany of articles ending with a chapter discussing ethical issues in dementia care. Care examples illustrate the difficult decisions that can face those involved in the care of cognitively impaired individuals.

The editor's introduction to each chapter and the author's conclusion at the end provide a good summary. This would be a good library book, but is probably not for personal shelves.

JANE GARNER, *Consultant Psychiatrist, Chase Farm Hospital, Enfield*

Police Selection and Training: The Role of Psychology (NATO Advanced Science Institutes Series. Series D: Behavioral and Social Science). Edited by JOHN C. YUILLE. Dordrecht, The Netherlands: Martinus Nijhoff. 1986. Pp 379. Dfl 200.00, $97.00, £63.00.

Until recently there were few texts on police psychology and none evaluating the role of psychology in the selection and training of police officers. This book, which documents the proceedings of a conference attended by 63 police officers and psychologists from eleven nations, aims to remedy this deficiency. Its special interest lies in the recognition that despite local and national differences, police psychologists share a wide variety of common concerns, and indeed have an important role to play – not least in the areas of stress management, therapeutic intervention, selection procedures, and aspects of training such as communication skills, de-escalation techniques, community relations, and interviewing techniques.

Several chapters report the findings of current research projects. While the majority of the contributions focus on selection and training, the book traverses a far wider field, providing a broad overview of the complexities of contemporary policing. A number of initiatives recorded were introduced partly as a response to criticisms that the police were becoming distanced from the communities they served and partly as a result of an increased awareness of the significance of the service role of the police.

Although the papers are of variable texture and depth and the index is somewhat rudimentary, in view of the burgeoning interest in exploring ways of enhancing the quality of interactions between the police and the public this text is a welcome addition to all those concerned with police training and selection, as well as to academics and researchers in related disciplines.

DIANNA M. YACH, *Lecturer in Law, Queen Mary College, London*

Family Care of Schizophrenia. By IAN R. H. FALLOON, JEFFREY L. BOYD and CHRISTINE W. MCGILL. New York: The Guilford Press. 1984. Pp 451. £28.50.

The title of this book reflects the positive attitude of the authors to the potential role of families in the treatment of schizophrenia. They review the literature on the aetiology and management of schizophrenia, noting how the family has been implicated and often berated, and conclude that negative attitudes are unwarranted. Instead, a model is adopted of schizophrenia as a chronic illness that increases stress in the family; the other family members are seen as potentially able to provide the best support for the patient.

Therapy with the family has as its cornerstones education about schizophrenia, enhancement of communication skills, and development of problem-solving ability as individuals and as a family. The aim is to reduce stress levels for patients in their day to day lives, by improving the quality of family interactions and by training in skills to manage stressful life-situations.

This behavioural family therapy is in addition to an intensive rehabilitation programme that includes careful monitoring of medication, and also careers advice. Results are reported of a two-year treatment study, with a control group receiving individual instead of family therapy. It is concluded that the family therapy sessions had a major impact in areas such as compliance with treatment, lower rates of readmission, and improved quality of life for patients and other family members.

The authors note that psychotherapy as they practice it may lack charisma and be "a little tedious". Unfortunately the book seemed to be tarred with the same brush: its text of well over 400 pages has few illustrations to break up the otherwise dense prose, making for difficulty in maintaining good concentration; and repetitions in the text are of a sort more suited to training workshops and family sessions.

It is not clear for whom this book was written: at times it is simple enough for a lay readership, but at others clearly aimed at experienced professionals. The first part of the book provided an account of schizophrenia that was satisfyingly balanced and level-headed, suitable for most students and trainees, while the sections describing their methods of working with families have much of value for those working not just with schizophrenics but also most other child and adult psychiatric patients. Above all, this is a book about good clinical practice and realisation of a broad scientific understanding in a caring partnership with patients and their families.

HUGH BARNES, *Consultant Child and Adolescent Psychiatrist, Bristol Royal Hospital for Sick Children*

Proceedings of the 15th European Conference on Psychosomatic Research. Edited by HUBERT LACEY and D. A. STURGEON. London: John Libbey. 1986. Pp 372. £35.00, $62.00.

The proceedings of the 15th European Conference on Psychosomatic Research contain sections on topics such as psychotherapy, liaison psychiatry, illness behaviour, eating disorders, cardiovascular disorders, hypertension, respiratory and malignancy, psychodermatology, and sleep. There are 95 contributors with a high overall standard both from Europe and North America. Practically every conceivable problem is covered, and thus the volume provides a miniature work of reference on the subject.

It is difficult to single out contributors, but there is a particularly strong section on cardiovascular disorders and hypertension. Price and Friedman describe their work in attempting to modify Type A behaviour to reduce coronary recurrence rates. They conclude that Type A behaviour can be reduced and such reduction is associated with a diminished risk of myocardial infarction.

Hawkins catalogues the decline of psychoanalysis in liaison psychiatry, and makes an eloquent plea for its resuscitation as "the most useful and comprehensive human psychology at present."

Lipsitt describes the changing role of the general hospital as a teaching centre for psychosomatic medicine. He describes the conflict generated in physicians attempting to practice the "new medicine" – i.e. the more regulated, cost-conscious, technologically driven and pressurised form. The physician is beset with contradictions and ambivalent demands to be both caring and "cost-effective". The difficult ethical and emotional problems for the doctor have resulted in an apparently increased demand in the US for liaison psychiatrists, who find themselves sought after for advice on bioethics, instrumentation and patient care, and support for colleagues frightened by the technological imperative in medicine.

The section on eating disorders is strong, particularly with contributions from the St George's Hospital Department. Arthur Crisp's 'Some psychopathological aspects of obesity' is a useful and comprehensive contribution.

This volume is recommended for all libraries interested in the area of psychosomatic research.

R. J. DALY, *Professor of Psychiatry, University College, Cork, Ireland*

Supervision and Training: Models, Dilemmas, and Challenges. Edited by FLORENCE W. KASLOW. New York: The Haworth Press. 1986. Pp 259. $32.95.

Good supervision is essential in psychotherapy practice. However, the literature on supervision is sparse in comparison with that on theory and technique. Rather than being specifically educated into their role, supervisors generally assume the position. The arguments in favour of better preparation and improved clarity in conceptual models are partially made by the volume under review. The editor published two books on the subject in the 1970s, and has now assembled a broad range of approaches which were first published as two issues of the journal *The Clinical Supervisor* in 1986. Most of the contributors are psychologists working in America, although one chapter comes from Cardiff and Sydney and another from Tel Aviv. Part I addresses the ecological context of place, the mechanics of obtaining supervision, and common fears of therapists. Part II has two good chapters on the stages that supervisor and supervisee mature through, one on private practice stressing the legal liability that a supervisor may, unwittingly, have for the therapist's work, and five presenting a cornucopia of techniques from rotating peer supervision to *in vivo* sequential therapy. In similar vein, Part III considers family and behaviour therapy and altering institutional structures.

The content of this book falls awkwardly between two stools. Despite attempting to be panoramic, it fails to include chapters on group and marital therapy or on supervision for supervisors, and while giving most space to supervision of individual therapy, it does not provide a detailed, integrated model that will guide and support supervisors in their role. Too many alternatives are presented. Experienced supervisors wanting the stimulus of new ideas will find it interesting.

MARK AVELINE, *Consultant Psychotherapist, Mapperley Hospital, Nottingham*

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